PERIPHERAL ARTERIAL DISEASE IN A MULTIETHNIC NATIONAL SAMPLE: THE ROLE OF CONVENTIONAL RISK FACTORS AND ALLOSTATIC LOAD

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Background: Limited data exist on the prevalence of peripheral arterial disease (PAD) among ethnically diverse populations. Our objectives were to assess the prevalence of PAD in a multiethnic national sample and examine risk factor control and allostatic load (a marker of dysregulation of the inflammatory, metabolic, and cardiovascular systems) by race/ethnicity among individuals with PAD.

Methods: We analyzed data from the 1999–2002 National Health and Nutrition Examination Survey for individuals aged ≥40 with a measured ankle brachial index (N=5,083). PAD was defined as an ankle brachial index <0.9. We performed bivariate and multivariate analyses to describe the association of race/ethnicity with PAD, controlling for sociodemographic factors, clinical risk factors and allostatic load.

Results: Rates of PAD were higher among African Americans (7.8%) than Whites (3.4%) or Mexican Americans (5.1%) (P<.001). African Americans with PAD were more likely to be taking antihypertensive medications, were less likely to report vigorous physical activity, and had higher allostatic load scores than Whites. Although 95% of individuals with PAD report a routine place for care, almost half had a measured blood pressure >140/90 mm Hg, 28% were smokers, and 61% had a cholesterol value ≥200 mg/dL.

Conclusions: Within this nationally representative sample, African Americans had the highest rates of PAD. Although conventional risk factor control, including control of hypertension and hyperlipidemia, were similar between racial groups, African Americans with PAD had higher allostatic load scores. Among all individuals with PAD, evidence showed suboptimal cardiovascular risk factor control. (*Ethn Dis*. 2007;17:669–675)

Key Words: Peripheral Vascular Disease, Race/Ethnicity

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INTRODUCTION

Peripheral arterial disease (PAD) is a highly prevalent manifestation of atherosclerosis that affects 8–12 million US adults and is associated with significant morbidity and mortality.[1,2] PAD can be diagnosed by the ankle brachial index (ABI), a noninvasive measurement of limb blood pressures. Prevalence of PAD varies by population and by type of measurement used.[1–4] Although previous studies report higher rates of PAD among African Americans,[5–7] few reports have focused on potential explanations for this difference.[4,8] Several studies suggest that the greater prevalence of PAD among African Americans is not explained by conventional risk factors,[9] leading authors to hypothesize that genetic or environmental factors may play a role.[9–11] Differential exposures to stressors have been postulated as a potential explanation for a portion of health disparities.[12] The term allostatic load is a recent conceptualization of the biologic burden of adaptation to physiologic and environmental stress,[13,14] predicts mortality and decline in physical functioning,[15] and is higher among African Americans than among other racial/ethnic groups.[16] Allostatic load is a cumulative measure of dysregulation in multiple physiologic systems (metabolic, cardiac, inflammatory) and is assessed through summary measures of biologic and physiologic measurements, including blood pressure, cholesterol, glucose regulation and inflammatory markers. Although inflammatory markers, including C-reactive protein and homocysteine,[17–19] have been implicated in the development of atherosclerosis, the relationship between allostatic load and PAD has not been studied.

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The purpose of the current study is to provide estimates of PAD among an ethnically diverse national sample and to assess the magnitude of association between known risk factors and allostatic load with PAD by race and ethnicity, in addition to assessing the level of risk factor control among individuals with PAD. To accomplish these aims, we analyzed data from the National Health and Nutrition Examination Survey (NHANES) 1999–2002, the most recent data from the ongoing NHANES. Previous analysis from NHANES did not include data from 2001–2002 and did not address potential explanations for dif-

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ferences in rates of PAD by race and ethnicity.[4,8]

METHODS

Study Population

The National Health and Nutrition Examination Surveys (NHANES) 1999–2000 and 2001–2002 are conducted by the National Center for Health Statistics and are designed to give a nationally representative sample of the US population.[20] The current analysis was granted an exemption from review by the institutional review board of the University of Washington.

Physical Exam and Laboratory Data

PAD was assessed by determining the ankle brachial index (ABI), calculated as the ratio of systolic blood pressure in the posterior tibial arteries to that in the right brachial vessel.[21] Measurements were taken by a trained technician using a standardized protocol with the subject supine using an 8 MHz- Doppler probe to determine the systolic pressure at each of the sites.[21] Systolic pressure was measured in the right arm (brachial artery) and both ankles (posterior tibial arteries). Brachial systolic blood pressure was measured twice for participants age 40–59 and once for those age ≥60 years. PAD was defined as ABI ≤.90 in either leg. ABIs ≥1.5 or greater were excluded from analyses because these values indicate incompressible calcified vessels and are not interpretable (n=26).[22] Physical examination data also included height and weight and were used to calculate body mass index (BMI). Laboratory data included hemoglobin A1C, lipids (total cholesterol, low density lipoprotein [LDL] cholesterol, and triglycerides), serum creatinine, serum albumin, homocysteine, and C-reactive protein. Glomerular filtration rate (GFR) was estimated using the Modification of Diet in Renal Disease formula, based on serum creatinine, age, sex, and race.[23]

Sociodemographics, Medical History, Health Status, and Physical Activity

Self-reported sociodemographic information included race/ethnicity, age, education, and income. Individuals were classified as African American, Mexican-American, White, and other race. Due to the heterogeneous nature of "other race," these individuals were excluded from the analyses (n=346). Self-reported medical history included personal history of coronary artery disease, stroke, diabetes, hypertension, dyslipidemia, smoking status, and medication use. Access to care was assessed with two questions: having a routine place for care and having health insurance. Self-reported health status was ascertained as excellent, very good, good, fair, or poor.

Respondents were asked about symptoms of claudication and their ability to perform moderate and vigorous physical activity. All respondents were asked if they experienced "pain in either leg while walking." If they answered affirmatively, they were then asked, "Does this include pain in your calf or calves?" Respondents were asked to report if they had performed any vigorous activity, such as running or swimming, for at least 10 minutes during the previous 30 days. In addition, they were asked if they had performed any moderate physical activity, such as brisk walking or bicycling, for at least 10 minutes during the previous 30 days.

Measure of Allostatic Load

On the basis of previous research using NHANES data,[16] 10 biomarkers made up the allostatic load score and include systolic and diastolic blood pressure and BMI from the physical exam, glycosylated hemoglobin, albumin, creatinine clearance, triglycerides, C-reactive protein, homocysteine, total cholesterol and GFR. For each biomarker, we empirically determined the high-risk threshold on the basis of the distribution of that biomarker.[15,16] Each participant was assigned a point for each biomarker that was beyond the threshold (defined as below the 25th percentile for GFR and albumin and above the 75th percentile for all other markers). The points were summed to generate an allostatic load score, with a range from 0 to 10. The high-risk thresholds were systolic blood pressure 138 mm Hg, diastolic blood pressure 81 mm Hg, BMI 31.2 kg/m^2, hemoglobin A1C 5.6%, albumin 4.47 g/dL, creatinine clearance 78.5 mL/min/1.73 m^2, triglycerides 189.5 mg/dL, C-reactive protein 0.49 mg/dL, homocysteine 10.1 μmol/L, and total cholesterol 233.9 mg/dL. Based on previous analysis,[16] an allostatic load of four or greater was used to define a high allostatic load score.

Statistical Analysis

We performed bivariate analysis to assess the association of PAD with demographic and clinical characteristics. To compare PAD groups with regard to these characteristics, we used the chi-square test for categorical variables and t-tests for continuous variables. We used a stepped approach to multivariate modeling of PAD, adjusting for other characteristics associated with PAD in previous studies.[24–26] First, we created a model that included demographic variables associated with PAD (race/ethnicity, age, and sex). Our second model included these sociodemographic variables and allostatic load. Our third model included sociodemographic variables and a clinical history of insulin-treated diabetes, hypertension, smoking status, level of physical activity, and obesity (BMI >30 kg/m^2).

RESULTS

Rates of PAD were highest among African Americans (7.8%) compared to Whites (3.4%) and Mexican Americans

Table 1. Prevalence of PAD by population characteristics among US adults aged ≥40 in NHANES 1999–2002, *N*=5083

	Population Characteristic	PAD, *n*=369 % (SE)	No PAD, *n*=4714 % (SE)
Race/ethnicity *	African American	7.8 (.8)	92.2 (.8) ††
	White	3.4 (.7)	96.6 (.7)
	Mexican American	5.1 (.4)	94.9 (.4)
Age	40–49 years	1.3 (.3)	98.7 (.3) ††
	50–59 years	2.7 (.6)	97.3 (.6)
	60–69 years	6.0 (.8)	94.0 (.8)
	70–79 years	15.3 (1.1)	84.7 (1.1)
	≥80 years	21.9 (2.3)	78.1 (2.3)
Male		4.2 (.5)	95.8 (.5)
Female		5.2 (.5)	94.8 (.5)
Education	Less than High School	8.4 (.8)	91.6 (.8) ††
	High School	5.5 (.7)	94.5 (.7)
	More than High School	3.0 (.3)	97.0 (.3)
Annual income†	$0–$14,999	9.7 (.8)	90.3 (.8) ††
	$15,000–$24,999	6.9 (1.2)	93.1 (1.2)
	$25,000–$54,999	4.6 (.6)	95.4 (.6)
	≥$55,000	2.1 (.3)	97.9 (.3)

PAD=peripheral arterial disease; NHANES=National Health and Nutrition Examination Survey; SE=standard error.
* Data available for *n*=4941;
† Data available for *n*=4550
†† $P<.05$ for χ^2

(5.1%) ($P<.001$) (Table 1). The elderly had very high rates of PAD: 15.3% among those age 70–79 and 21.9% among those age ≥80 compared to ≤6% in younger persons.

Individuals with PAD were more likely to report a history of coronary artery disease, stroke, diabetes, hypertension, and dyslipidemia and were significantly more likely to be current or previous smokers (Table 2). Individuals with PAD were much less likely to report either moderate or vigorous activity than those without PAD. Although 95% of individuals with PAD reported having a routine place for health care, 49% had a measured blood pressure >140/90 mm Hg, 61% had a total cholesterol level ≥200 mg/dL, and 73% had an LDL cholesterol level ≥100 mg/dL. Individuals with PAD had higher allostatic load scores than those without PAD, with higher levels of systolic blood pressures, lower GFR, and higher levels of homocysteine and C-reactive protein.

The risk factor profile among individuals with PAD differed by race and ethnicity in several respects (Table 3). African Americans were more likely to be taking an antihypertensive medication, have lower triglyceride levels and GFR, and have higher BMI, hemoglobin A1C and C-reactive protein levels than Whites. African Americans were less likely to report vigorous physical activity (16%) than Mexican Americans (24%) or Whites (35%) ($P<.001$). African Americans with PAD had higher allostatic load scores than Whites ($P<.05$).

In multivariate analysis that controlled for age and sex (Table 4), African American race was associated with higher odds of PAD. A high allostatic load, as defined by a score of four or greater,[16] was significantly associated with PAD, controlling for age, sex, and race/ethnicity. Significant predictors of higher PAD odds included age, hypertension, diabetes treated with insulin, and current or previous smoking history (model 3). Reports of vigorous physical activity were associated with lower odds of PAD.

DISCUSSION

In this multiethnic nationally representative sample, rates of PAD were higher among African Americans than Whites and Mexican Americans. Several risk factors differed by race and ethnicity among those with PAD; African Americans were more likely to be treated for hypertension, have higher BMIs and were less likely to report moderate physical activity over the past 30 days. African Americans were also noted to have the highest allostatic load scores, suggesting a greater dysregulation of metabolic, inflammatory, and cardiovascular systems. Previous authors have postulated that allostatic load represents a marker of the cumulative wear and tear from repeated adaptation to stress.[15] Multiple mechanisms for the higher rates of atherosclerosis among African Americans have been studied, including clustering of risk factors, less access to care, poorer quality of care, lower levels of physical activity, and genetic and environmental factors.[6,9,11] A previous study of national NHANES data reported higher allostatic load scores for African Americans,[16] but this is the first study to link higher allostatic load scores to PAD.

The prevalence of PAD by race and ethnicity reported in this national sample is lower than several prior studies[5,7] but similar to a study using

Table 2. Medical history, PAD risk factors, and allostatic load among 5083 US adults aged ≥40 in NHANES 1999–2002

	Characteristic	PAD *n*=369	No PAD *n*=4714
Medical history, % (SE)	Coronary artery disease	13 (2.2)	4 (.3) *
	Stroke	10 (1.8)	3 (.4) *
	Diabetes	21 (2.5)	9 (.6) *
	Hypertension	61 (2.9)	34 (1.2) †
	Dyslipidemia [a]	59 (.9)	41 (3.7) *
Smoking status, % (SE)	Current	28 (2.0)	20 (.9) †
	Previous	42 (3.0)	32 (1.0)
	Never	30 (3.2)	47 (1.3)
Medications, % (SE)	Insulin[b]	46 (8.2)	20 (2.4) *
	Oral hypoglycemics[b]	56 (6.9)	71 (2.8)
	Antihypertensives[c]	88 (2.3)	84 (1.4) *
	Anti-lipemics[d]	64 (5.3)	50 (1.5) *
Healthcare access, % (SE)	Routine place for care	95 (1.1)	90 (.8) *
	1+ doctor visits/past year	90 (2.6)	87 (.8) *
	Has health insurance	92 (2.2)	89 (.8)
Self-reported health status, % (SE)	Excellent	9 (1.2)	21 (1.3) †
	Very good	19 (2.5)	31 (.8)
	Good	35 (3.2)	30 (.9)
	Fair	23 (2.2)	15 (.8)
	Poor	13 (2.3)	3 (.4)
Pain in calf or calves with walking, % (SE)		32 (3.2)	11 (4.8) †
Physical activity/past 30 days, % (SE)			
Vigorous activity		15 (2.2)	30 (1.3) †
No vigorous activity		75 (2.8)	66 (1.2)
Unable to do vigorous activity		10 (2.9)	4 (0.4)
Moderate physical activity, % (SE)		32 (2.7)	50 (1.3) †
No moderate physical activity		60 (2.7)	48 (1.3)
Unable to do moderate physical activity		8 (2.4)	2 (.3)
Poor risk factor control	Hypertension >140/90, % (SE)	49 (3.0)	23 (.8) †
	Cholesterol >200 mg/dL % (SE)	61 (2.8)	59 (1.3)
	LDL cholesterol >100, % (SE)e	73 (4.9)	80 (1.4)
Allostatic load components - Examination and laboratory data			
Systolic blood pressure, mean (SE)		142 (2)	128 (2) †
Diastolic blood pressure, mean (SE)		67 (1)	74 (.3) †
Total cholesterol, mean mg/dL (SE)		213 (3)	212 (4)
Triglycerides, mean mg/dL, (SE)		176 (12)	168 (6)
BMI, mean kg/m^2, (SE)		27.8 (0.4)	28.3 (.2)
Glomerular filtration rate mL/min/1.73 m^2 (SE)		72.4 (1.9)	85.5 (.6) †
Hemoglobin A1C, mean (SE)		5.96 (.08)	5.58 (.02) †
Homocysteine, mean umol/L, (SE)		11.1 (.4)	8.8 (.09) †
C-Reactive Protein, mean mg/dL (SE)		0.71 (.06)	.43 (.02) †
Albumin, mean g/dL (SE)		4.2 (.02)	4.4 (.01) †
Allostatic load score, mean (SE)		3.3 (.08)	2.2 (.05) †
Allostatic load score ≥4, % (SE)		49 (2.6)	23 (1.0) †

* $P<.05$;
† $P<.001$
Column totals may vary due to rounding error; [a] data available for $n=4016$; [b] among individuals with diabetes ($n=640$): [c] among individuals with hypertension ($n=2006$); [d] among individuals with high cholesterol ($n=1766$); [e] among n=1944 individuals with fasting blood work.

earlier NHANES data.[4] Previous small non-population-based studies have reported higher rates of PAD and lower extremity amputation in the African American population. Among individuals aged ≥50 years from four primary care clinics in Texas, the prevalence of PAD was higher among African Americans (22.8%) and Mexican Americans (13.7%) than among Whites (13.2%).[5] Of 1775 participants from the SHEP (Systolic Hypertension in the Elderly Program) clinical trial whose average age was 71 years, the prevalence of lower extremity PAD was 25% in White men, 38% in Black men, 23% in White women, and 41% in Black women.[7] Other studies involving nursing home patients and a multicenter study conducted among US primary care practices demonstrated similar prevalence of PAD among whites and Mexican Americans.[27,28] In a retrospective study of the arteriograms of 135 men admitted for

Table 3. Risk factor profile by race and ethnicity among individuals with and without PAD, among 4941 US adults aged ≥40 in NHANES 1999–2002

Characteristic		With PAD n=367			Without PAD n=4,574		
		African American (n=93)	Mexican American (n=69)	White (n=205)	African American (n=823)	Mexican American (n=1276)	White (n=2475)
Diabetes, % (SE)		22 (.9)	29 (5.4)	19 (3.1)	15 (1.6)	15 (1.3)	8 (.7)†
Hypertension, % (SE)		68 (5.7)	49 (9.5)	60 (3.7)	48 (2.1)	25 (2.3)	32 (1.3)†
High cholesterol, % (SE)		46 (5.9)	53 (6.5)	61 (4.6)	40 (1.9)	46 (2.6)	41 (1.1)
Smoker, % (SE)	Current	31 (5.3)	17 (6.5)	28 (2.5)*	29 (2.4)	21 (1.8)	19 (1.2)†
	Previous	29 (4.8)	37 (6.5)	44 (3.6)	22 (2.3)	29 (1.9)	35 (1.2)
	Never	39 (4.6)	46 (11.4)	28 (3.8)	49 (3.8)	50 (2.3)	46 (1.5)
Taking medication, % (SE)	For hypertension [a]	98 (1.6)	74 (6.9)	87 (3.1)*	87 (1.6)	73 (3.4)	84 (1.6)*
	For high cholesterol	75 (9.0)	53 (10.6)	63 (5.9)	46 (2.4)	39 (.4)	52 (1.7)*
Healthcare access, % (SE)	One place for routine care	91 (3.3)	83 (3.3)	83 (1.2)	89 (1.4)	78 (2.6)	92 (.9)†
	Health care insurance	86 (3.9)	91 (4.4)	95 (2.5)	81 (1.5)	67 (2.9)	92 (.9)†
Physical activity/past 30 days, % (SE)	Vigorous	16 (3.4)	24 (6.9)	35 (2.9)*	31 (2.2)	33 (2.1)	54 (1.5)†
	Moderate	11 (3.5)	14 (2.3)	14 (2.3)	23 (1.8)	25 (2.3)	32 (1.6)*
Allostatic load components and risk factor control Mean (SE)	Systolic blood pressure	144 (2)	144 (3)	141 (2)	133 (1)†	127 (2)	127 (1)
	Diastolic blood pressure	70 (2)	65 (2)	67 (1)	77 (1)*	74 (.5)	74 (.4)
	Total cholesterol (mg/dL)	207 (4)	215 (4)	214 (7)	209 (2)	214 (4)	212 (1)
	Triglycerides (mg/dL)	140 (12)*	177 (20)	182 (15)	112 (4)†	191 (14)	169 (7)
	BMI (kg/m^2)	30.4 (1.2)*	27.0 (1.0)	27.3 (.5)	29.5 (.2)†	28.7 (.3)	28.1 (.2)
	Glomerular filtration rate [b]	86.5 (4.2)*	84.7 (2.8) †	69.6 (2.0)	95.8 (1.1) †	98.3 (1.3) †	83.4 (.6)
	HbA1C %	6.6 (.2)*	6.2 (.3)	5.8 (.1)	5.9 (.05)†	5.9 (.06)†	5.5 (.03)
	Homocysteine (umol/L)	12.1 (1.5)	9.7 (.4) *	11.0 (.4)	9.5 (.3)*	8.3 (.2)*	8.9 (.1)
	C-reactive protein(mg/dL)	.96 (.15)*	.81 (.20)	.65 (.07)	.60 (.04)†	.45 (.02)	0.41 (.02)
	Albumin (mg/dL)	4.1 (.02)*	4.2 (.02)	4.2 (.03)	4.2 (.02) †	4.3 (.02)	4.4 (.01)
Allostatic load, mean (SE)		3.9 (.2)*	3.0 (.3)	3.3 (.1)	2.7 (.1)†	2.2 (.1)	2.3 (.1)

* $P<.05$

† $P<.001$ for overall χ^2 or t-test for comparison to White individuals.

Column totals may vary due to rounding error. [a] among individuals with hypertension ($n=2006$); [b] mL/min/1.73 m^2

evaluation of lower extremity ischemia, African American patients were found to have more severe disease in every segment of the infrageniculate arteries.[29]

Despite high rates of health care access, most individuals with PAD had suboptimal risk factor control. Unfortunately, the opportunity for intervention may be missed due to underdiagnosis of PAD.[28] Patients with PAD are at increased risk for death[30–34] and limb loss[35] and underdiagnosis may lead to inadequate process of care. Previous studies have shown that atherosclerotic risk factors are less intensively treated in patients with PAD than in patients with coronary artery disease (CAD).[36] Patients with PAD received less intensive treatment for lipid disorders and hypertension and were prescribed antiplatelet therapy less frequently than were patients with CAD.[28] Despite the fact that almost all of the respondents in our study reported a routine place for medical care, almost half of this sample with PAD had a measured blood pressure during the physical examination of ≥140/90, and two thirds had total cholesterol values >200 mg/dL. Although exercise is recommended as a mainstay of treatment for PAD,[37,38] only one third of those with PAD reported moderate exercise.

There are several potential limitations to this study. NHANES does not include individuals who live in institutions such as nursing homes, so the prevalence of PAD may be underestimated. Because NHANES is a cross-sectional survey, no conclusions about causal associations can be inferred. In addition, medical history was obtained by self-report and is limited by recall and other biases. There may be variability in the performance of ABI across studies. The methodology used by NHANES to measure PAD does not include ankle pressures from the anterior tibial vessel. Because recommended determination of ABI requires use of the higher of the two systolic pressures from the dorsalis pedis and posterior tibial arteries,[39,40] the prevalence of an ABI <.9 may be overestimated by the NHANES survey, although we do not expect this overestimation to differ by race/ethnicity, co-morbidity, or allostatic load. In addition, the prevalence measures are based on noninvasive measures of disease. However, the ABI has higher sensitivity and specificity than previously used clinical indicators, such as claudication or pulse deficits.[28]

Table 4. Multivariate models of association with PAD in multiethnic national sample of 5083 US Adults aged ≥40 in NHANES 1999–2002

Characteristic		Model 1 Race/ethnicity [a]	Model 2 Allostatic load [a]	Model 3 Access/ co-morbidity [b]	Model 4 Allostatic load, co-morbidity [c]
Race/ethnicity	African American	2.1 (1.6, 2.9) †	1.9 (1.4, 2.6) †	1.9 (1.4, 2.7) *	1.8 (1.2, 2.6) *
	Mexican American	1.0 (0.7,1.5)	1.0 (0.7, 1.5)	1.0 (0.6, 1.6)	0.7 (0.4, 1.1)
	White	Reference	Reference	Reference	Reference
Age (mean)		1.1 (1.07, 1.09) †	1.1 (1.07, 1.09) †	1.1 (1.07, 1.10) †	1.1 (1.06, 1.10) †
Male		0.9 (0.6, 1.3)	0.9 (0.7, 1.4)	0.9 (0.6, 1.3)	0.9 (0.6, 1.4)
Allostatic load ≥4			2.0 (1.5, 2.6) †		1.8 (1.3, 2.3) †
At least one place for routine health care				1.6 (0.8, 3.4)	2.0 (0.9, 4.2)
Co-morbid conditions	Diabetes on insulin			3.3 (1.8, 5.9) †	
	Hypertension (>140/90 mm Hg)			1.6 (1.1, 2.3) *	
BMI >30 kg/m^2				1.3 (0.8, 1.9)	
Smoker	Current			4.0 (2.7, 5.9) †	3.6 (2.3, 5.6) †
	Previous			2.1 (1.4, 3.1) †	2.0 (1.3, 3.0) *
	Never			Reference	Reference
Physical activity	Vigorous			0.7 (0.5, 0.9) *	0.7 (0.5, 0.9) *
	Moderate			0.9 (0.6, 1.4)	0.9 (0.6, 1.4)

OR >1 associated with PAD (ABI <.9)
* $P<.05$
† $P<.001$
[a] The model included a total of 4734 persons: 356 persons with PAD, 4378 without PAD
[b] The model included a total 4042 persons: 313 persons with PAD, 3729 without PAD
[c] The model included a total 4403 persons: 330 persons with PAD, 4073 without PAD

Using angiography as the gold standard, the sensitivity of the ankle brachial index is 90%, and the specificity is 98% for stenosis of ≥50% or more in a major leg artery.[28]

In conclusion, we found that African Americans have higher rates of PAD than Whites or Mexican Americans. Risk factors differed by racial and ethnic groups for treatment of hypertension, BMI, and exercise and for allostatic load, suggesting several potential mechanisms for the noted higher odds of disease among African Americans. Despite having access to care, up to 75% had suboptimal risk factor control, suggesting that PAD is either underdiagnosed and unrecognized or not treated as aggressively as other vascular diseases such as coronary heart disease.

…we found that African Americans have higher rates of PAD than Whites or Mexican Americans.

Although the current US Preventive Services Task Force recommendations do not include screening for PAD in asymptomatic individuals,[41] screening targeted at populations with multiple risk factors including African Americans, smokers, persons with diabetes treated with insulin, and the elderly may have the greatest yield for PAD detection.

ACKNOWLEDGMENTS

We would like to thank Jeff Rodenbaugh for his help with data analysis. The views expressed in this article are those of the authors and do not necessarily represent the views of the Department of Veterans Affairs.

REFERENCES

1. Melton LJ 3rd, Macken KM, Palumbo PJ, Elveback LR. Incidence and prevalence of clinical peripheral vascular disease in a population-based cohort of diabetic patients. *Diabetes Care*. 1980;3(6):650–654.
2. Criqui MH, Fronek A, Barrett-Connor E, Klauber MR, Gabriel S, Goodman D. The prevalence of peripheral arterial disease in a defined population. *Circulation*. 1985;71(3): 510–515.
3. Meijer WT, Hoes AW, Rutgers D, Bots ML, Hofman A, Grobbee DE. Peripheral arterial disease in the elderly: The Rotterdam Study. *Arterioscler Thromb Vasc Biol*. 1998;18(2): 185–192.
4. Selvin E, Erlinger TP. Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999–2000. *Circulation*. 2004;110:738–743.
5. Collins TC, Petersen NJ, Suarez-Almazor M, Ashton CM. The prevalence of peripheral arterial disease in a racially diverse population. *Arch Intern Med*. 2003;163(12):1469–1474.
6. Rucker-Whitaker C, Greenland P, Liu K, et al. Peripheral arterial disease in African Americans: clinical characteristics, leg symptoms, and lower extremity functioning. *J Am Geriatr Soc*. 2004;52(6):922–930.
7. Newman AB, Sutton-Tyrrell K, Kuller LH. Lower-extremity arterial disease in older hypertensive adults. *Arterioscler Thromb*. 1993;13(4):555–562.
8. Gregg EW, Sorlie P, Paulose-Ram R, et al. Prevalence of lower-extremity disease in the US adult population >=40 years of age with and without diabetes: 1999–2000 national health and nutrition examination survey. *Diabetes Care*. 2004;27(7):1591–1597.
9. Kullo IJ, Bailey KR, Kardia SL, Mosley TH, Jr., Boerwinkle E, Turner ST. Ethnic differences in peripheral arterial disease in the NHLBI Genetic Epidemiology Network of

Arteriopathy (GENOA) study. *Vasc Med*. 2003;8(4):237–242.
10. Rucker-Whitaker C, Feinglass J, Pearce WH. Explaining racial variation in lower extremity amputation: a 5-year retrospective claims data and medical record review at an urban teaching hospital. *Arch Surg*. 2003;138(12):1347–1351.
11. Ferdinand KC. Coronary artery disease in minority racial and ethnic groups in the United States. *Am J Cardiol*. 2006;97(2A): 12A–19A.
12. Szanton SL, Gill JM, Allen JK. Allostatic load: a mechanism of socioeconomic health disparities? *Biol Res Nurs*. 2005;7(1):7–15.
13. Seeman TE, McEwen BS, Rowe JW, Singer BH. Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proc Natl Acad Sci U S A*. 2001;98(8): 4770–4775.
14. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med*. 1998; 338(3):171–179.
15. Seeman TE, Singer BH, Rowe JW, Horwitz RI, McEwen BS. Price of adaptation—allostatic load and its health consequences. MacArthur studies of successful aging. *Arch Intern Med*. 1997;157(19):2259–2268.
16. Geronimus AT, Hicken M, Keene D, Bound J. ''Weathering'' and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health*. 2006;96(5): 826–833.
17. Fichtlscherer S, Breuer S, Schachinger V, Dimmeler S, Zeiher AM. C-reactive protein levels determine systemic nitric oxide bioavailability in patients with coronary artery disease. *Eur Heart J*. 2004;25(16):1412–1418.
18. Musicant SE, Taylor LM, Jr., Peters D, et al. Prospective evaluation of the relationship between C-reactive protein, D-dimer and progression of peripheral arterial disease. *J Vasc Surg*. 2006;43(4):772–780.
19. Ridker PM, Stampfer MJ, Rifai N. Novel risk factors for systemic atherosclerosis: a comparison of C-reactive protein, fibrinogen, homocysteine, lipoprotein(a), and standard cholesterol screening as predictors of peripheral arterial disease. *JAMA*. 2001;285(19): 2481–2485.
20. SUDAAN Software. Version 8.0.1. Research Triangle Park, NC: Research Triangle Institute; 2002.
21. Lower Extremity Diseases Procedures Manual, January 2001. National Health and Nutrition Examination Survey (NHANES). Available at: http://www.cdc.gov/nchs/data/nhanes/le.pdf. Last accessed 4/5/2006.
22. Resnick HE, Lindsay RS, McDermott MM, et al. Relationship of high and low ankle brachial index to all-cause and cardiovascular disease mortality: the Strong Heart Study. *Circulation*. 2004;109(6):733–739.
23. Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med*. 1999;130(6):461–470.
24. Criqui MH. Peripheral arterial disease–epidemiological aspects. *Vasc Med*. 2001; 6(3 Suppl):3–7.
25. Newman AB, Siscovick DS, Manolio TA, et al. Ankle-arm index as a marker of atherosclerosis in the Cardiovascular Health Study. Cardiovascular Heart Study (CHS) Collaborative Research Group. *Circulation*. 1993; 88(3):837–845.
26. Hiatt WR. Medical treatment of peripheral arterial disease and claudication. *N Engl J Med*. 2001;344(21):1608–1621.
27. Aronow WS. Prevalence of atherothrombotic brain infarction, coronary artery disease and peripheral arterial disease in elderly blacks, Hispanics and whites. *Am J Cardiol*. 1992;70(13):1212–1213.
28. Hirsch AT, Criqui MH, Treat-Jacobson D, et al. Peripheral arterial disease detection, awareness, and treatment in primary care. *JAMA*. 2001;286(11):1317–1324.
29. Sidawy AN, Schweitzer EJ, Neville RF, Alexander EP, Temeck BK, Curry KM. Race as a risk factor in the severity of infragenicular occlusive disease: study of an urban hospital patient population. *J Vasc Surg*. 1990;11(4): 536–543.
30. McKenna M, Wolfson S, Kuller L. The ratio of ankle and arm arterial pressure as an independent predictor of mortality. *Atherosclerosis*. 1991;87(2–3):119–128.
31. Criqui MH, Langer RD, Fronek A, et al. Mortality over a period of 10 years in patients with peripheral arterial disease. *N Engl J Med*. 1992;326(6):381–386.
32. Newman AB, Shemanski L, Manolio TA, et al. Ankle-arm index as a predictor of cardiovascular disease and mortality in the Cardiovascular Health Study. The Cardiovascular Health Study Group. *Arterioscler Thromb Vasc Biol*. 1999;19(3):538–545.
33. Vogt MT, Cauley JA, Newman AB, Kuller LH, Hulley SB. Decreased ankle/arm blood pressure index and mortality in elderly women. *JAMA*. 1993;270(4):465–469.
34. Zheng ZJ, Sharrett AR, Chambless LE, et al. Associations of ankle-brachial index with clinical coronary heart disease, stroke and preclinical carotid and popliteal atherosclerosis: the Atherosclerosis Risk in Communities (ARIC) Study. *Atherosclerosis*. 1997 May; 131(1):115–125.
35. Ouriel K. Peripheral arterial disease. *Lancet*. 2001;358(9289):1257–1264.
36. McDermott MM, Mehta S, Ahn H, Greenland P. Atherosclerotic risk factors are less intensively treated in patients with peripheral arterial disease than in patients with coronary artery disease. *J Gen Intern Med*. 1997;12(4): 209–215.
37. Stewart AH, Lamont PM. Exercise for intermittent claudication. Supervised programmes should be universally available. *BMJ*. 2001;323(7315):703–704.
38. McDermott MM, Liu K, Ferrucci L, et al. Physical performance in peripheral arterial disease: a slower rate of decline in patients who walk more. *Ann Intern Med*. 2006; 144(1):10–20.
39. Sacks D, Bakal CW, Beatty PT, et al. Position statement on the use of the ankle-brachial index in the evaluation of patients with peripheral vascular disease: a consensus statement developed by the standards division of the Society of Cardiovascular & Interventional Radiology. *J Vasc Interv Radiol*. 2002;13(4): 353.
40. Atsma F, Bartelink M-LEL, Grobbee DE, van der Schouw YT. Best reproducibility of the ankle–arm index was calculated using Doppler and dividing highest ankle pressure by highest arm pressure. *J Clin Epidemiol*. 2005;58(12): 1282–1288.
41. Screening for Peripheral Arterial Disease: Recommendation Statement. Available at: http://www.ahcpr.gov/clinic/uspstf05/pad/padrs.pdf. Last accessed 5/24/2006.

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