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Brain aging in African-Americans: The Atherosclerosis Risk in Communities (ARIC) experience

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Abstract

Reported rates of dementia differ by race, although most studies have not focused on carefully measured outcomes, confounding by education or other demographic factors, nor have they studied other outcomes to dementia. In this review we will discuss the experience in the Atherosclerosis Risk in Communities (ARIC) study evaluating racial disparities relating to stroke, subclinical brain infarction, leukoaraiosis, as well as cognitive change and dementia. ARIC is a biracial cohort of 15,792 participants from four U.S. communities, initially recruited in 1987–1989, and seen at a total of 5 in-person visits (most recently seen in 2011–2013 with annual follow-up phone calls. We will provide evidence from ARIC studies that disproportionate rates of vascular risk factors explain at least some of these observed disparities by race, but particular risk factors, including diabetes, may differentially affect the brain in African-American versus white participants. In addition, we will review some of the disparities by race in studies focusing on the genetics of stroke, small vessel disease, and dementia.

INTRODUCTION

Disparities in stroke rates and stroke-related mortality are commonly reported in the so-called “stroke belt”,^{1,2} with higher rates among African-American individuals than among whites. Beyond clinical stroke, African-Americans appear to be at increased risk for adverse subclinical brain changes and related sequelae, including cognitive impairment and dementia.³ Possible explanations for the excess burden of negative neurologic outcomes in African-Americans include: 1) higher prevalence of vascular risk factors, including hypertension,⁴ diabetes,⁵ and smoking; 2) earlier onset of risk and greater severity, or more poorly controlled⁶ risk factors; 3) greater sensitivity to risk factors (i.e., greater target organ damage at comparable levels of risk factor severity), or 4) differences in the social and environmental context. However, the precise cause of excess risk in African-Americans remains to be determined.

In this report, we review findings from the Atherosclerosis Risk in Communities (ARIC) study, with over 25 years of long-term follow-up on clinical and subclinical brain outcomes

in a large cohort of African-American and white men and women. We will summarize findings related to stroke, subclinical cerebrovascular disease (silent infarcts, leukoaraiosis), brain atrophy, cognitive decline and dementia, with particular emphasis on disparities by race. We will provide evidence that observed disparities by race may be due to differences in risk factor presence, severity, and control. In addition, we will review genetic differences in, or other explanations for, some of these observed associations by race, and will discuss plans to evaluate unanswered questions regarding race-specific disparities in brain health.

The ARIC study

ARIC study participants were randomly recruited from four U.S. communities (Forsyth County, NC (both African-American and white participants); Jackson, MS (all African-American participants); Washington County, MD (majority white participants); and suburbs of Minneapolis, MN (majority white participants)), with an initial visit in 1987–1989, when participants were 45–64 years old.⁷ Participants have had four additional in-person visits, in the field centers associated with each community: in 1990–1992 (visit 2), 1993–1995 (visit 3), 1996–1999 (visit 4), and most recently in 2011–2013 (visit 5; Table 1). All participants are contacted via annual follow-up (AFU) calls, and records for hospitalized events are obtained, abstracted, and reviewed via expert adjudication as well as an algorithmic diagnosis; stroke cases are therefore all identified by expert review. Dementia was not reviewed previously in ARIC, and for the studies summarized in this paper, when used as an outcome relied upon hospitalization codes.⁸ Whites and African-Americans all had the same standardized visit protocols (table 1) with the exception of the MRI visits at Visit 3 and the ARIC Brain MRI visit, when recruitment was only at the Forsyth County, NC and Jackson, MS sites, thus leading to a larger proportion of African-Americans than otherwise represented in the cohort.

The cohort underwent cognitive testing with the Delayed Word Recall, Digit Symbol Substitution, and the Word Fluency tests at visits 2, 4, 5, and the Brain MRI ancillary visit, with a more detailed neurocognitive battery also administered at the Brain MRI visit (to a subset; Table 1) and at visit 5.

Stroke

The ARIC study design allows evaluation not only of stroke prevalence but incidence, which allows not only comparison of rates by race but also comparison of risk factors in association with incident cases. At the onset of the ARIC study, prevalent stroke or transient ischemic attack (TIA) was estimated in 5.5% of African-Americans, but in 6.3% of whites.⁹ At the Forsyth County, NC site, (the only site with both African-Americans and whites), rates for prevalent stroke or TIA were consistently higher for African-Americans than for whites.⁹ In addition, over the course of the 25 years since study onset, more *incident* strokes have been identified in African-American than in white participants: compared to Minneapolis, MN whites, the incidence rate ratio per 1000 person-years for Jackson, MS African-Americans was 2.37 (95% CI 1.78–3.16) and 1.88 (95% CI 1.12–3.13) for Forsyth County, NC African-Americans.¹⁰ Differential by race appears highest for lacunar-type strokes, (OR 2.98, 95% CI 1.87–4.76), independent of other vascular risk factors or

demographics.¹¹ In a recent analysis in ARIC, the standard American Heart Association/American Stroke Association-recommended ICD-9 codes for diagnosis of stroke were also found to have slightly higher positive predictive value in blacks than in whites (compared to expert review as the gold standard), possibly reflective of the higher stroke rates.¹²

As a possible explanation for observed disparities in stroke rates, it has been hypothesized that risk factors for stroke are either more prevalent in African-Americans than in whites (table 2), or have differential effects in African-Americans than in whites, perhaps partially explaining these disparities in stroke rates. Our data support both hypotheses, at least partially: in ARIC, hypertension and diabetes are more common in African-Americans than in whites, although smoking rates appear lower in African-Americans.¹³ Specific risk factors have been evaluated to determine if their effect on stroke differs by race: stroke incidence rates were consistently higher in African-Americans, across different obesity groups, without evidence of stronger risk conferred by obesity among different race groups,¹⁴ and hypertension and smoking each had similar risk for stroke in African-Americans and whites. In contrast, African-Americans with diabetes had a 2.5-fold increased risk of stroke, in adjusted models, compared to African-Americans without diabetes, but whites with diabetes only had a 1.7-fold increased odds compared to whites without diabetes (p-interaction=0.02).¹⁵ Socioeconomic status is also another likely important difference between African-Americans and whites in ARIC, which is likely an important surrogate for access to medical care. However, even among groups with annual income <\$35,000/year, a likely surrogate for poor access to medical care, incidence rates are consistently higher in African-Americans than in whites. This is true both in younger participants with low income, with a much higher incidence rate per 1000 person-years in African-Americans (IR 4.26; 95% CI 3.40–5.27) than in whites (IR 1.19, 95% CI 0.82–1.69), and in older age groups with low income.¹⁵

In addition to possible differences in risk factors and their control, unique risk factors are noted in African-Americans that may increase stroke risk beyond what has previously been recognized; sickle cell trait, previously thought to be a benign carrier state, was associated with an increased hazard of ischemic stroke (HR 1.4, 95% CI 1.0–2.0) in ARIC.¹⁶ Genetic differences might also account for stroke disparities by race, as will be discussed later in this review.

In summary, incident strokes, especially lacunar strokes, are more common in African-Americans than in whites. Our data support that these disparities are due to differences in prevalence of risk factors for stroke, with a suggestion that some risk factors (e.g. diabetes) might have a larger incremental increase in risk of stroke in African-Americans than in whites. In addition, unique risk factors, including genetic factors such as sickle cell disease or trait, may exist in African-Americans that might explain some of the differential in stroke rates.

Subclinical brain disease

Although prior studies in ARIC have focused on a clinical diagnosis of stroke, the shift to consideration of subclinical brain changes within the recommended definition of stroke¹⁷

emphasizes the importance of brain imaging to evaluate disease processes that may be related to stroke pathophysiologically, but do not present clinically as stroke. Because risk factors for stroke are similar to those for subclinical cerebrovascular disease, it would be expected that disparities by race seen at one end of the disease spectrum would be seen in other brain pathologies along that same spectrum. Brain imaging done on a subset of the ARIC cohort has yielded informative data not only about differences in subclinical brain changes by race, but also disparities in the strengths of the associations between risk factors and these subclinical imaging changes. In 1993–1995, over 1800 ARIC participants (55 years and older) from the Forsyth County, NC and Jackson, MS sites were invited for a Brain MRI, with a repeat brain MRI study conducted in a subset (N=1130) in 2004–2006. Methods describing the rating of both MRI scans for infarcts,¹⁸ leukoaraiosis,¹⁹ and brain volumes²⁰ are reported elsewhere.²¹

Subclinical infarcts

Infarcts were common on the initial brain MRIs of ARIC participants, found in 15.3% of the cohort (ages 55 to 72). The prevalence of these lesions increased with age (nearly doubling from ages 55–59 to 60–64, and nearly tripling for persons aged 65–72). The majority were clinically silent, occurring in participants without a history of stroke or TIA (only 1.5% had a clinical stroke or TIA). Most were also of a typical size and location of lacunar infarcts, and were found twice as often in African-Americans (21%) as in white participants (10%),¹⁸ primarily due to differences in risk factors.

On follow-up MRI, 10 years later, incident infarcts were identified on 20.1% of participant scans, with similar rates by race (African-American women: 20.9%; African-American men: 20.3%; white women: 20.7%; white men: 17.9%) but higher rates by age.²¹ However, in an analysis evaluating different-sized silent infarct-like lesions, as well as the quantity of silent lesions, with separate evaluation for lesions 7 mm and below (smaller lacunes) as well as those 8–20 mm in diameter (larger lacunes), African-American race was a significant predictor, independent of other vascular risk factors, of a larger number of silent lacunar infarcts of both sizes (adjusted OR's 1.55 (95% CI 1.18–2.93) and 1.58 (95% CI 1.00–2.49), respectively).

Leukoaraiosis

Often studied separately from silent infarcts, leukoaraiosis (or white matter hyperintensities (WMH)) may represent part of the spectrum of small vessel disease. In ARIC, WMH was common at the baseline MRI (1993–1995) and progression was common at the follow-up MRI. On the baseline MRI, although fewer African-Americans had any WMH, those who did had more severe WMH than whites.²²

As is the case for clinical stroke, one possible explanation for disparities in extent of WMH and lacunes by race is differences in risk factor distribution, as supported by some other cohorts including minorities such as the Washington Heights-Inwood Columbia Aging Project;²³ alternatively, the same risk factors might differentially impact arteries and therefore brain health in persons in different race groups. Based on the initial ARIC MRI, when WMH was rated using a 0–9 categorical scale, a diagnosis of hypertension, one of the

most frequently implicated risk factors for small vessel disease, was associated with a higher odds of WMH in African-Americans than in whites. This was especially true for African-Americans with a history of treated but uncontrolled hypertension, in whom the odds of WMH was 4.68 (95% CI 2.65–8.27), compared to 2.38 in whites (95% CI 1.28–4.44).¹⁹ Similar results were found for actual systolic blood pressure,¹⁹ with more prevalent WMH in association with an equivalent level of blood pressure, suggesting that disparities observed are not only due to differences in risk factor severity. This is further supported in analyses of other risk factors, including smoking, which conferred a nearly 4.5-fold greater risk of severe grade WMHs in African-Americans compared to whites,²² although similar results were not seen for WMH progression.²⁴

On follow-up MRI, WMH was measured both using the 0–9 visual rating scale, as well as with a volumetric measurement of WMH volume. In the evaluation of WMH progression, defined using imputed volumes for the baseline ARIC MRI, based on the quadratic association between the visual scale and volumetric measurement on MRI follow-up, most participants had some progression of WMH, but by visual grade only 4% of whites, and 10% of African-Americans ($p < 0.001$) had substantial progression (defined as progression of two or more grades).²⁵ Despite this difference in progression, the equivalent increase in systolic blood pressure was not clearly associated with a difference in amount of WMH progression by race; cumulative blood pressure, which was a time-averaged systolic blood pressure reflecting blood pressures over approximately 15 years, was associated with, on average, 2.56 more cm^3 WMH volume progression per each 20 mm Hg in whites, compared to 2.48 cm^3 in African-Americans.²⁵ Of note, midlife blood pressure was a stronger predictor of late-life WMH progression than was later life blood pressure, but this age-of-onset preference was found in African-Americans only, perhaps reflecting a longer exposure to high blood pressure in the years prior to the initiation of the ARIC study.²⁵

Because of similarities in risk factors and likely pathophysiology, leukoaraiosis and infarcts are often considered two types of small vessel disease. Consideration has been given to combining small vessel disease phenotypes into a composite measure. In ARIC, a composite measure of progression of small vessel disease, combining WMH progression with incident lacunar infarcts, was more strongly associated with retinal markers of small vessel disease than the markers defined separately.²⁶ It is possible that a composite measure of small vessel subtypes will provide a better understanding of racial disparities with respect to prevalence, progression, and risk factors for brain aging.

Cerebral atrophy

Measures of cerebral atrophy on the initial brain MRI were also worse in African-Americans than in whites, measured by higher sulcal and ventricular grades. In addition, especially in African-American men, progression (worsening) across the two MRI's of ventricular grade (but not sulcal grade) was marginally more frequent than progression in whites.²¹

To summarize, neuroimaging data in ARIC further supports higher rates of subclinical vascular brain changes in African-Americans than in whites, paralleling the disparities in clinical stroke. Although some data support that risk factors, such as smoking, might differentially be associated with some of these changes (leukoaraiosis) and might afford a

higher risk in African-Americans than in white smokers, other risk factors, including hypertension, do not clearly have differential associations with brain microvascular disease, nor do analyses with measures of progression of brain microvascular disease.

Cognitive Change and Dementia

Cognitive performance has been evaluated in ARIC at multiple time points over the 25 year follow-up period, allowing evaluation of changes in cognition that might be fairly subtle, and also allowing a relatively unconfounded evaluation of risk factors with these changes in cognition. Cognitive measures were repeated at ARIC visits 2, 4, and 5, and changes in these cognitive measures appear to not be associated with factors such as education²⁷ and socioeconomic status, suggesting that evaluation of change allows measurement of true racial differences in decline over time, thus allowing more direct measurement of the association between a risk factor and cognitive performance. Cognitive performance at a single time point is more likely to be confounded by factors including education and socioeconomic status, which cannot always be fully accounted for in statistical models. For instance, in the Cardiovascular Health Study, crude dementia rates did appear to differ by race, but with adjustment for education, these changes were attenuated and became nonsignificant;²⁸ adjustment, however, will only account for those easily measured confounders of these associations. This is especially important to consider, given prior reports suggesting higher rates of dementia in African-Americans in other cohorts.^{29–31} Participants in ARIC of African-American race have had, on average, lower scores on cognitive tests than their white counterparts, independent of age and education, but consistent with our studies of education,²⁷ this differential applies primarily to baseline performance on tests, without evidence of a difference in rate of decline in African-Americans, compared to whites.

Studies of cognitive change in the setting of these baseline disparities also need to consider attrition of those populations at highest risk for an adverse brain outcome, and our studies have suggested that dropout is greater- whether due to disease, mortality or refusal- in African-Americans than in whites. In ARIC, hypertension was associated with more decline on a global cognitive measure (the average of three separate tests from three distinct cognitive domains), although results in African-Americans were weaker and did not reach statistical significance. Use of inverse probability of attrition weighting (IPAW) to account for attrition strengthened associations in African-Americans, further supporting that disproportionate attrition in African-Americans might explain why some associations have appeared weaker than hypothesized.³²

As with the changes with clinical stroke and subclinical brain disease, it has been hypothesized that disparities in late-life cognitive performance by race are due to differences in risk factor distribution and severity. The data described above on hypertension do not support stronger relationships with a diagnosis of hypertension and cognitive change, and the evaluation of continuous SBP measurement demonstrated significant associations with cognitive decline only among whites, not among African-Americans.³² In addition, no disparities by race were found in rate of decline associated with diabetes.³³ The apolipoprotein Eε4 allele, the strongest genetic risk factor for Alzheimer's disease, was

associated with decline in cognitive tests when ARIC participants were younger, but with more consistent and stronger effects in whites than in African-Americans, and with an interaction between diabetes and hypercholesterolemia, each, with apoE genotype, only in whites.³⁴

When baseline cognitive performances are lower, even if decline does not differ by race, this may lead to a larger population burden of dementia. Individuals starting at a lower cognitive level may reach a threshold at which point they are diagnosed with dementia earlier than individuals starting at a larger distance above this threshold. The use of race-specific norms, as developed in ARIC,³⁵ decreases the likelihood of inaccurate diagnosis of dementia, but the status of dementia research as of the date of this review still suggests disparities in dementia rates among African-Americans and whites, with higher rates in African-Americans.

Data using an outcome of hospitalizations with a dementia diagnosis suggest that risk factor associations with hospitalized dementia might be slightly stronger in African-Americans than in whites, supporting that this misclassification may be occurring and leading to higher rates of dementia when using discharge codes (further supported by overall higher rates of dementia in African-Americans versus whites, in studies using hospitalization codes⁸). Hypertension had similar associations with hospitalized dementia in African-Americans (HR 1.7) and whites (HR 1.6), but diabetes was a much stronger predictor in African-Americans (HR 2.8), with significant but lower effect sizes in whites (HR 1.9).³⁶ Low vitamin D levels were associated with higher risk of hospitalized dementia, but slightly stronger effect sizes were suggested in African-Americans (HR 1.53, 95% CI 0.84–2.79; versus HR 1.32, 95% CI 0.69–2.55 in whites, with a non-significant term for interaction by race),³⁷ and apoE status did not differ in its association with this hospitalized dementia outcome in ARIC.³⁶

Review of our cognitive change analyses supports the utility of evaluating change as an outcome: it allows evaluation of risk factor/ cognitive change associations, likely independent of baseline differences by race. We do not find evidence in ARIC that vascular risk factors differentially affect cognitive change by race, yet dementia rates, at least with a hospitalized dementia code, still are higher in African-Americans than in whites.

Genetics

Genetic differences may account for racial disparities in brain health. While genome-wide association studies (GWAS) have successfully identified DNA variants associated with stroke,³⁸ brain subclinical disease,³⁹ cognitive function,⁴⁰ and dementia⁴¹ in whites, few such studies have been conducted in African-Americans. In contrast, GWAS of the *risk factors* in African-Americans have yielded important insights. For example, a GWAS of blood pressure in 29,378 African-Americans from 19 discovery cohorts, including ARIC, identified 3 novel loci but showed that DNA sequence variants discovered in whites have broad effects in African-Americans.⁴² Similarly, in the Population Architecture using Genomics and Epidemiology (PAGE) study, which includes ARIC, evaluation of the generalization of GWAS associations for 5 cardiovascular disease-related traits to non-European populations showed that effects were generally consistent in direction but not in magnitude across ancestral groups, and that differential findings were far more frequent in

African-Americans.⁴³ Extension of GWAS associations identified in whites for ischemic stroke, brain infarcts on MRI, and white matter lesions on MRI to ARIC African-Americans has yielded mixed results, possibly due to differences in power, genomic coverage, as well as magnitude of effect. A polymorphism near the *NINJ2* gene was associated with ischemic stroke in both whites and African-Americans.³⁸ However, association of a polymorphism in the *MACROD2* gene identified in GWAS of MRI-defined brain infarcts in whites was not replicated in ARIC African-Americans, although 4 polymorphisms located within 50–150 kb showed evidence of association.⁴⁴ Polymorphisms at a locus on chromosome 17q25 associated with white matter lesion burden in whites were not replicated in ARIC African-Americans,³⁹ but a subsequent GWAS including populations of various ancestries showed similar direction of effects of polymorphisms at this locus in whites, African-Americans, and Hispanics.⁴⁵ These findings emphasize the need for comprehensive genetic studies of traits related to brain health in African-Americans.

Conclusions

ARIC data do support a greater burden of both clinical and subclinical brain disease in African-Americans than in whites, with a suggestion that the accumulation of these changes is also greater in African-Americans. This appears to be primarily due to differences in prevalence of vascular risk factors (table 2), although some of our data do support that certain risk factors, including diabetes, might increase risk for stroke and subclinical brain disease to a greater extent in African-Americans than in whites. This may reflect: 1) differences in risk factor severity; 2) differences at the tissue level in response to risk factors; 3) differences in genetic susceptibility to certain risk factors or treatment for those risk factors; or, most likely, 4) some combination of the above.

The strengths of the ARIC study, with 4,265 African-American men and women,⁴⁶ have been the longitudinal design, now with more than 25 years of follow-up, repeated cognitive measures allowing relatively unconfounded assessment of cognitive change, and the biracial distribution of the cohort. Results should be interpreted with caution, however; a majority of the African-Americans in ARIC were recruited from one geographic location (Jackson, MS), which has a high rate of coronary heart disease, stroke and risk factors. We do not feel, however, that this means that our findings are only a result of a unique sample in Jackson, MS differing from our other sites, since many of our findings were true also of the Forsyth County, NC site, where both whites and African-Americans have been included. Although some analyses are evaluated for site-specific findings, these are neither possible nor available for all reported analyses (for instance, the MRI data only included two field centers total). Nevertheless, ARIC's findings are largely consistent with those reported from smaller samples of African-Americans from other locations, with a general emphasis on vascular risk factors as at least a partial mechanism for any measured racial disparities.^{23,47}

African-Americans in ARIC have higher rates of incident stroke, subclinical microvascular disease, and hospitalized dementia than whites. Cognitive scores are lower at baseline in ARIC but do not appear to decline at a faster rate in African-Americans. Cardiovascular risk factors make a significant contribution to the outcomes examined, and appear to explain much of the excess burden in African-Americans. However, there are some notable

exceptions: diabetes in particular seems to have a greater effect on risk of stroke in particular, suggesting perhaps differential toxicity, and data are inconsistent for hypertension, which may differentially effect WMH but not clearly its progression. Regardless of the cause, the excess clinical and subclinical CVD burden presents an ominous pattern, likely translating to an increased dementia risk in an aging population.

Reports of higher rates of dementia among African-Americans need confirmation using appropriate norms and evaluations of cognitive change, as well as study of the possible mechanisms of higher rates of dementia in African-Americans, if present. ARIC data to date suggest that risk factors may be more prevalent and more severe in African-Americans, which may contribute to higher rates of clinical and subclinical brain vascular disease, and ultimately potentially more dementia. It is likely that the observed disparities in stroke or small vessel disease rates are indirectly responsible for some of the reported disparities identified in dementia and mild cognitive impairment (MCI), but these outcomes also need to be evaluated separately. Careful evaluation of dementia and the etiology of dementia cases (whether due to Alzheimer's disease, or vascular cognitive impairment, or a combination, for instance), particularly in African-Americans, is needed to further understand potential racial disparities in brain diseases including dementia.

Future Directions

To fully understand the burden of vascular disease on dementia risk by race, data are needed to better estimate population attributable fractions for specific risk factors, by race. In addition, more emphasis is needed on the effect of risk factor timing, duration, and treatment effects. Either independently or in combination with these other areas requiring further study, further evaluation of the potential genetic contributions, and social/ environmental contributions (such as access to health care and social structure), to disparities in brain aging requires continued study of diverse populations. The imperfect generalization to African-Americans of genetic risk prediction models using genetic variants derived from white populations further underscores the need for comprehensive genetic studies in this population if clinical utility of such models is to be achieved.

The ARIC-Neurocognitive Study (ARIC-NCS; 2011–2013) recently completed detailed neurocognitive assessments in the ARIC cohort, with brain MRI in a subset (~2000) of participants. More details about social and behavioral factors that might contribute to racial disparities are also available as part of this new phase of the study. These data, combined with expert-led adjudication of all mild cognitive impairment (MCI) and dementia cases, will allow further evaluation of MCI and dementia prevalence in the ARIC cohort and will allow further evaluation of risk factors for late-life dementia.

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Table 1

Atherosclerosis Risk in Communities (ARIC) study visit timeline.

	ARIC Visit 1: 1987–1989	ARIC Visit 2: 1990–1992	ARIC Visit 3: 1993–1995	ARIC Visit 4: 1996–1999	ARIC Brain MRI: 2004–2006	ARIC Visit 5/ Neurocognitive study: 2011–2013
Age Range (y)	45–64	48–67	51–70	54–83	62–81	70–89
Sample size	15,792	14,348	12,887	11,656	1,134*	6,538
Vascular risk factors/ markers	X	X	X	X	X	X
Cognitive testing		X		X	X	X
Brain MRI			X (in 1,929*)		X	X (in 2,000)

Numbers listed above represent number of participants seen in clinic, with the exception of ARIC-NCS which also includes some home visits. Participants have also been contacted by telephone annually (with more recent switch to semi-annual telephone calls).

* The ancillary studies allowing MRI scanning at ARIC visit 3 and the ARIC Brain MRI visit were only performed at the Forsyth County, NC and Jackson, MS field centers, and therefore included a larger proportion of African-Americans than was representative of the entire cohort at that time.

Table 2

Risk factors for stroke and neurologic disease in ARIC, by race, across ARIC visits 1–4.

	Visit 1		Visit 2		Visit 3		Visit 4	
	African-Americans	Whites	African-Americans	Whites	African-Americans	Whites	African-Americans	Whites
Age (years; mean (SD))	53.6 (5.8)	54.4 (5.7)	56.3 (5.8)	57.3 (5.7)	59.0 (5.7)	60.3 (5.7)	61.8 (5.7)	63.1 (5.6)
Hypertension (%)	55.9	27.3	55.8	29.6	60.7	35.2	67.4	42.2
Diabetes (%)	17.8	7.2	20.6	8.9	21.8	10.2	24.2	11.5
Current smoking (%)	29.9	24.8	25.7	21.4	21.4	16.7	17.7	14.1

ARIC: Atherosclerosis Risk in Communities;
SD: Standard Deviation.