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Prevalence of Hypertension by Duration and Age at Exposure to the Stroke Belt

Virginia J Howard[1], **Robert F. Woolson**[2], **Brent M. Egan**[3], **Joyce S. Nicholas**[2,4], **Robert J. Adams**[4], **George Howard**[5], and **Daniel T. Lackland**[2]

[1]Department of Epidemiology, School of Public Health, University of Alabama at Birmingham, Birmingham, AL

[2]Department of Medicine, Medical University of South Carolina, Charleston, SC

[3]Department of General Internal Medicine/Hypertension, Medical University of South Carolina, Charleston, SC

[4]Department of Neuroscience, Medical University of South Carolina, Charleston, SC

[5]Department of Biostatistics, School of Public Health, University of Alabama at Birmingham, Birmingham, AL

Abstract

Background—Geographic variation in hypertension is hypothesized as contributing to the stroke belt, an area in southeastern United States with high stroke mortality. No study has examined hypertension by lifetime exposure to the stroke belt.

Methods—This association was studied in 19 385 participants in the REasons for Geographic And Racial Differences in Stroke (REGARDS) study, a national population-based cohort. Prevalent hypertension was defined as SBP ≥ 140, DBP ≥ 90, or use of antihypertensive medications. Stroke belt exposure was assessed by residence at birth, currently, early childhood, adolescence, early adulthood, mid-adulthood, and recently.

Results—After adjustment for age, race, sex, physical activity level, body mass index, smoking, alcohol, education, and income, the prevalence of hypertension was significantly more strongly related ($p < 0.0001$) with lifetime exposure, adolescence or early adulthood exposure than exposures at other times. Birthplace and current residence were independently associated with hypertension; however, lifetime, adolescence or early adulthood exposures were more predictive than joint model with both birthplace and current residence.

Conclusions—That adolescence and early adulthood periods are more predictive than residence in the stroke belt for most recent 20-year period suggests community and environmental strategies to prevent hypertension need to start earlier in life.

Keywords

southeastern United States; stroke; migration

Corresponding author: Virginia J. Howard, PhD, Department of Epidemiology, School of Public Health, University of Alabama at Birmingham, 1665 University Boulevard, Birmingham, AL 35294-0022, USA, Telephone: (205) 934-7197; FAX: (205) 934-8665, vjhoward@uab.edu.

Introduction

The United States stroke belt, usually defined as including the eight southern states of North Carolina, South Carolina, Georgia, Tennessee Mississippi, Alabama, Louisiana, and Arkansas, was first identified in 1965 with approximately a 50% higher stroke mortality then the rest of the country and it still persists today. (1-6) Within the stroke belt, a “buckle” region along the coastal plain of North Carolina, South Carolina, and Georgia has been identified with even a higher stroke mortality rate than the remainder of the stroke belt. (7) Higher prevalence of hypertension has been hypothesized as one of the causes of the higher stroke mortality rates in the southeastern US stroke belt. (6,8-9) In studies of regional comparisons of hypertension prevalence, a slightly higher prevalence of hypertension in the southeast was found for some race-sex groups (white men, white women, and black men) with a more substantial difference for black women. (10-11) In further analysis assessing age groups, in seven of eight age-race-sex strata, hypertension was more prevalent in the southeast but the regional differences were only statistically significant among black men aged 40-59 and white men aged 40-59. (9) Cross-sectional analysis of the Framingham Stroke Risk Score from the REasons for Geographic And Racial Differences in Stroke (REGARDS) national cohort study found that use of antihypertensive medications was more common in the stroke belt than the rest of the nation for both blacks and whites, but the pattern of systolic blood pressures (SBP) was not consistent: whites in the stroke belt (but not the buckle) had higher SBP than the rest of the nation and blacks in the stroke buckle (but not the belt) had lower SBP than rest of nation. (12)

Previous studies have shown that nativity or birthplace is associated with cardiovascular mortality with individuals born in the Southeast having higher rates than those born in other regions of the U.S. (13-14) These studies, however, did not assess the relationship of birthplace or extent of lifetime exposure in the stroke belt to blood pressure or hypertension. Studies in other countries have examined blood pressure measurements and other cardiovascular risk factors by birthplace, region of residence in childhood and adulthood and conclude that current residence is more influential than region of birth. (15,16) These studies, however, have focused on only a few residency periods. We use lifetime residential history data from a US adult cohort to examine the prevalence of hypertension by exposure to the stroke belt at birth, current residence, and six other measures of life time exposure to the stroke belt.

Methods

This report uses cross-sectional data from the REasons for Geographic And Racial Differences in Stroke (REGARDS), a longitudinal population-based cohort study designed to investigate factors associated with the excess stroke mortality observed among African Americans and residents of the Southeastern United States. (17) REGARDS was designed to be a national cohort of community-dwelling individuals age 45 years and older, randomly selected with approximately equal representation of whites and blacks, men and women, with oversampling from the stroke belt. Begun in January 2003, the study aimed to recruit 30 000 participants aged 45 and older, with approximately 15 000 currently living in the Stroke Belt region and 15 000 currently living in other regions of the country. Within each region, the goal was to recruit approximately half African Americans, half whites, half men and half women. Using mail and telephone contact methods, participants were recruited from commercially available lists of U.S. residents purchased from Genesys, Inc. The lists were stratified with respect to age, race, sex, and geographic region of current residence to accommodate the REGARDS sampling strategy. (17) Race was defined by self-report, and by design, Hispanics and Latinos were excluded. When enrollment was completed in October 2007, the final cohort of 30 239 was 26% black women, 16% black men, 29% white women, and 29% white men, 35% from the stroke belt, 21% from the stroke buckle, and 44% from the rest of the 40 contiguous states.

Defined according to standards recommended by Morton et al. (18) the telephone response rate was 33% and the cooperation rate was 49% (similar to other epidemiological studies, the Multi-Ethnic Study of Atherosclerosis, MESA, for example with a 39.8% participation rate among those contacted and to whom the study was explained.)

Using a computer-assisted telephone interview, trained interviewers obtained demographic information, medical history and an array of other potential risk factors. Consent was obtained initially on the telephone and subsequently in writing during an in-person evaluation. A brief physical exam including blood pressure measurements was conducted 3-4 weeks after the telephone interview. Self-administered questionnaires were left with the participant to gather information. All involved ethical review boards approved the study methods. Additional methodological details are provided elsewhere. (17)

Age, race, sex, use of antihypertensive therapy (are you now taking any medicine for high blood pressure?), smoking, alcohol use, physical activity level, annual family income, and education level were defined from self-report. Height, weight and blood pressure measurements were obtained from the in-person component. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were defined as the average of two measurements taken by a trained technician using a standard protocol and regularly tested aneroid sphygmomanometer (American Diagnostic Corporation), measured after the participant was seated for 5 minutes. The majority of blood pressure measurements were performed between 7 am and noon (91%); 1% were performed earlier than 7 AM, and 8% were performed in the afternoon. Where possible, blood pressures were taken in the left arm and a large size cuff was used if the arm circumference was greater than 13 inches. Both the cuff bladder width and pulse obliteration level were also recorded. The cuff was inflated to 20 mmHg above the pulse obliteration level, and slowly deflated (approximately 2 mmHg/second) to obtain the blood pressures. This process was repeated to obtain the second blood pressure on the same (left) arm. Blood pressure quality control was monitored by central examination of digit preference and retraining of technicians took place as necessary. Prevalent hypertension was defined as SBP $\geq$ 140 mmHg or DBP $\geq$ 90 mmHg or self-reported use of antihypertensive medications. Smoking was categorized into strata of current, former, and never. Alcohol consumption was categorized as number of drinks per day (rare defined as < 1 drink/week, moderate between 1 drink/week and no more than 2 drinks/day, heavy > 2 drinks/day/, former alcohol use, never use). Physical activity level was defined by response to the question "*How many times per week do you engage in intense physical activity, enough to work up a sweat?*" categorized as a healthy level if 4 or more times per week, light if 1-3 times a week or none. Body mass index (BMI) was determined as a function of measured height and weight.

Birthplace, Age and Years of Exposure to the Stroke Belt

Current state of residence was obtained from the sampling frame and confirmed by telephone at time of enrollment. City and state of birth and residence from birth to time of enrollment was obtained from the self-administered "Places You Have Lived Questionnaire." Participants were asked to include places where they had lived at least one year and age at each move. Each participant's questionnaire was processed into individual records, each containing city/state name associated with birthplace and if applicable, any move from birthplace. Records with a valid state name were compared to a file obtained from the US Census Bureau to determine match with city and state. The Federal Information Processing Standards (FIPS) codes for states, counties, and named populated places were used to identify the city/state of birth and each residence/move. (20) For records without a perfect match on city/state, a computer program was written to identify up to 10 city/state combination from the census files that were mnemonically closest to the spelling provided by the participant. These were reviewed by study staff and further separated into those where a match could be found, subjectively rated as a

strong match or a weak match, and those where no match could be found. For those with a strong or weak match, the state and country FIPS codes were available for comparison, i.e., Talladelga, example for Talladega, Alabama. The weak matches were reviewed by senior staff/faculty to ensure validity. For those where no match could be found, the records were reviewed manually by senior staff/faculty, using searches including Google map to attempt to identify the correct spelling (e.g., Bham is recognized manually as Birmingham, AL.) This resulted in three subdivisions: 1) city/state match could be found; 2) state was correct but no city was identified in the state close enough to suggest a match; and 3) spellings that were clearly in error and neither the city nor state could be recognized. For purposes of this paper, participants with one or more residences falling into this third category were excluded from analyses. *The residency data used in this paper are only coded at the state level to categorize exposure periods to the stroke belt states. To categorize exposure to the 'stroke buckle"(comprised of approximately 160 counties along the coastal plains of North Carolina, South Carolina and Georgia), requires the resolution of spelling and recognition of over 100,000 cites/towns, work that is ongoing*.

The study had an active tracking process to contact participants to return languishing questionnaires. Analyses were therefore restricted to participants with in-home evaluations conducted by September 1st 2006 to allow time for retrieval, data processing, and coding. By that date, 24 271 participants had been enrolled. By January 2008, residential history questionnaires had been retrieved on 21 052 (86.7%). Of these, 19 498 (92.6%) had records that allowed assessment of lifetime state of residence. Further removing 113 individuals with missing hypertension (the primary outcome variable) reduced the analysis dataset to 19 385 individuals.

Statistical Analysis

The following measures of exposure to the stroke belt were calculated for each participant:

1. Place of birth (stroke belt versus non-stroke belt)
2. Current place of residence (stroke belt versus non-stroke belt)
3. Percent of childhood years (age 0 to 12 years) in stroke belt,
4. Percent of adolescent years (age 13 to 18 years) in stroke belt,
5. Percent of young adulthood (age 19-30 years) in stroke belt,
6. Percent of middle adulthood (age 31-45 years) in stroke belt,
7. Percent of recent years (past 20 years) in stroke belt,
8. Percent of entire life in the stroke belt (birth to current age encompassing the above measures)

The prevalence of hypertension within each of these measures was calculated. Logistic regression was used to establish the association of prevalent hypertension with each measure of exposure, first in a model adjusting for demographic factors (age, sex, race), secondly adding the socioeconomic factors of income and education and lastly, after adjusting for potential confounders of smoking, alcohol use, physical activity level and BMI. While the residential measures of exposure to the stroke belt are correlated, the goal of the analysis is to assess if the differences between measures introduced by moving between regions contain differential information on the likelihood of prevalent hypertension. Specifically, the results of the logistic models were examined to determine which, if any, exposure measures were predictive of prevalent hypertension. Because with this sample size most all differences will be statistically significant for even the smallest differences, where more than one exposure index was found

to be predictive, they were ranked according to the Wald Chi-square statistic. For all possible pairs of exposure variables, within each race-sex group and overall, whether one was "more significant" than the other was assessed by bootstrapping the contrast. In each of 2 000 replications, the Wald Chi-square was calculated for both measures, with the exposure measure with the largest associated Wald Chi-square considered as the "more significant" for the specific replication of the bootstrap. The proportion of times that each index was "more significant" than all others was calculated and compared to the null hypothesis 0.5 (i.e., a 50-50 distribution) using a z-score calculated under the asymptotic normal assumption.

Results

Table 1 shows the baseline characteristics by region and by race-sex group within region. Thirty-eight percent (7 224) of the participants were African American, 54.3% (10 239) were women, and 53% (10 006) resided in the stroke belt at the tine of the home examination. The mean (SD) age was 66.1 (9.0) with a range of 45-96 years. There were 11 247 participants (58.0%) with prevalent hypertension. Overall, blacks had higher prevalence of hypertension than whites did: 73.0 % of black females, 67.6% of black males compared to 47.9 % of white females and 51.9% of white males.

The prevalence of hypertension by proportion of time in each of the stroke belt exposure measures is provided in Table 2 (reading down the columns.) Among persons who have ever lived in the stroke belt (19 385-6 600=12785), the highest prevalence of hypertension (61.9%) was among individuals who had lived their entire life in the stroke belt, followed by those who had lived in the stroke belt 1-33% of their life (59.5.0%), followed by 57.8% for those with 67-99% lifetime exposure, and 56.35% for 34-66% lifetime exposure. This is in contrast to the prevalence of 54.9% for the 6 600 participants with no exposure to the stroke belt. A similar pattern of increasing prevalence of hypertension with increasing amount of time lived in the stroke belt was also present for the first 12 years of life.

Results of the multivariable logistic regression models predicting prevalent hypertension for all of the stroke belt exposures (any exposure vs. no exposure) indices are provided in Table 3. Our data shows higher prevalence of hypertension among current residents of the stroke belt (age-race-sex adjusted OR = 1.20; 95% CI 1.13 – 1.28), and an interaction test between region and race-gender failed to support a differential impact between the race-gender groups ($p = 0.34$) (not shown in Table 3). Because the results of the logistic regression were all significant by the same small p-value ($p< 0.0001$), the Wald chi-square statistic was used to rank the strength of association between each of the indices and prevalent hypertension. For all of the indices, across the basic demographic model as well as the risk factor adjusted models, the odds of hypertension was significantly greater than 1, ranging from an OR=1.28 (95% CI: 1.20, 1.37) for living in the stroke belt between ages of 19-30 in the simplest demographic model to an OR=1.15 (95% CI: 1.08, 1.23) for being born in the stroke belt or living in the stroke belt first 12 years of life in the SES model (reference group is those with no exposure to the stroke belt during that time period.)

Focusing on the final multivariable model, the lifetime exposure to the stroke belt appeared to be the best predictor of prevalent hypertension (Wald chi square 30.9), followed by exposure during ages 19-30 (Wald chi-square 28.4), then the last 20 years (Wald chi-square 26.1) followed closely by ages 13-18 (Wald chi-square 25.9). This ranking from high to low was generally consistent across the models.

Results of the bootstrapping are shown in Table 4, providing the relative strength of association (rank) of measures of exposure to the stroke belt with the prevalence of hypertension. Specifically, across all participants (column 1), total lifetime exposure was significantly more

associated with the prevalence of hypertension than any of the other 7 indices, followed by a tie with exposure between the ages of 13-18 and 19-30 which were significantly more related to prevalent hypertension than the remaining 5 exposures, followed by exposures in the last 20 years which was more associated than the remaining 4 exposures, followed by current residence in the stroke belt (more significant than the final three), followed by exposure during ages 31-45 (more significant than the final two), and finally exposures during the first 12 years of life and being born in the stroke belt whichh tied for having the weakest association with hypertension.

For both white men and white women, the most significant stroke belt exposure associated with prevalent hypertension was lifetime exposure. Currently living in the stroke belt was the least (or next to last) exposure category associated with prevalent hypertension. For black males, early life exposures (living in stroke belt during ages 13-18, for the first 12 years of life, and being born in the stroke belt) were the most significant stroke belt exposures associated with prevalent hypertension. This is in sharp contrast to that for black women where the later periods of exposure to the stroke belt (currently living in the stroke belt and living there during the last 20 years) were the most significantly associated with prevalent hypertension, and the early periods (birth and first 12 years of life) were the least associated with prevalent hypertension.

The contribution of each of the measures of exposure to the ability to predict hypertension can be indexed by comparing the information they provide to that provided by the “traditional” risk factors for hypertension. Specifically, the change in Somer's D (an index of the likelihood of predictive probabilities from the regression model being concordant with prevalent versus absent hypertension) was calculated by removing each of the risk factors one at a time. (21) Large changes were found in the Somer's D associated with removing race/sex (-0.040), age (-0.048) and BMI (-0.072), but smaller contributions to the predictive power for exercise (-0.003), education (-0.002), income (-0.002), alcohol (-0.002), and smoking (-0.002). In contrast, the increase of Somer's D for adding exposure at ages 19-30 or exposure at ages 13-18 was 0.002, suggesting that each of these had a similar impact on hypertension as education, income, alcohol use, and smoking (and only marginally smaller than exercise). The other indices of exposure were all associated with an increase in the Somer's D of 0.001, suggesting a smaller (but still significant) addition of information.

Discussion

Our study shows that exposure to the stroke belt is associated with higher prevalence of hypertension among a national sample of adults 45 and older. After adjustment for age, race, sex, physical activity level, body mass index, smoking, alcohol use, education, and income, the prevalence of hypertension was significantly most strongly related ($p < 0.0001$) to lifetime as well as adolescence or early adulthood exposure to the stroke belt than exposures at other times. Birthplace and current residence in the stroke belt were each independently associated with hypertension, however, lifetime, adolescence or early adulthood exposures were more predictive than the joint model with both birthplace and current residence. There was consistency across the race-sex groups in the adolescence/early adulthood/youngest ages with the exception of black women, where the later age periods of exposure to the stroke belt were most predictive of hypertension.

Previous studies suggest a relationship between birthplace in the Southeast and stroke mortality. Our findings extend those previous reports by showing that early life exposure to the stroke belt is associated with hypertension, which is the major population attributable risk factor for stroke. (22) Individuals are more likely to spend a greater period of their early life

in the region of their birthplace. Moreover, health attitudes and behaviors established in adolescence and early adulthood are likely to persist in later years.

The findings of our study further suggest that regional differences in lifestyle during adolescence and early adulthood have the most substantial contributions to regional differences in hypertension. One could speculate that these are periods where life style choices, such as dietary patterns and exercise habits, are established. The southern region of the US consisting of the eight traditional stroke belt states plus eight other states in the mid-Atlantic area plus the District of Columba has the highest sodium and lowest potassium intakes, dietary factors that interfere with achieving an optimal blood pressure. (23) In addition, data from multiple sources show that there are generally lower levels of healthy behaviors in the southern region of the country. (24-26) The 2005 US Behavioral Risk Factor Surveillance Study (BRFSS) shows that 7 of the 16 states with the highest percentage of persons who reported no leisure time physical activity (> 26%) were stroke belt states. (25) In 1991 BRFSS data, the geographical areas with the highest prevalence of 2 or more cardiovascular risk factors were the Midwestern and southern states, and from 1991 to 1999, the prevalence increased by 10% or more in 36 states, significantly so for 21 states, including 7 of the 8 stroke belt states. (24,26) The 2001 BRFSS showed that four of the top 10 states in obesity prevalence were within the stroke belt. (24) Our study does not have data on lifetime salt consumption but additional analyses could be performed using the single measurement obtained through baseline dietary assessment, a method Strazzullo et al used in their meta-analysis of salt intake and CVD outcomes. (27)

Southern states have been reported to have among the poorest childhood health circumstances including high rates of low birth weight, high infant mortality, child mortality, teen mortality, and teen birth rates. (28) This suggests that we consider the fetal origins theory that prenatal and early life conditions are associated with predisposition to diseases and risk factors later in life, sometimes referred to as the Barker hypothesis. (29) A recent meta-analysis of almost 2000 adults from 20 Nordic cohorts supports the inverse relationship between birth weight and systolic blood pressure, with or without adjustment for BMI, showing heterogeneity in shape and strength of association by sex and age. (30) One of the few studies to be conducted in the US, actually of a cohort within a stroke belt state (Louisiana), the Bogalusa Heart Study, found that birth weight was significantly inversely associated with progression of systolic and diastolic blood pressure as well as pulse pressure through early adulthood, and this was after adjustment for later health indicators such as BMI. (31) Lower birth weights in the Southeast could explain a portion of the higher hypertension prevalence in this portion of the United States. (32-33) This is an area that merits further study. Unfortunately REGARDS did not obtain data on birth weight.

The findings in our study are subject to some important limitations. The cross-sectional design provides only a one-time assessment of blood pressure so the estimate of prevalent hypertension for any individual may be incorrect. Error in either direction is possible, however, misclassification was minimized because our definition included individuals who reported taking antihypertensive medications but who were normotensive at time of exam. We do not have data on lifetime socioeconomic conditions or environmental exposures that are known to contribute to the development of hypertension. (34) Inherently, self-reported measures of smoking status, physical activity, alcohol, and income are prone to bias, however we have actual measurement of BMI. Additionally, while the validity of the assessment of physical activity is well established, it is easier to work up a sweat in a hot climate than a cooler northern climate.

While strengths of this study include a national, general population sample, a large African-American population and the availability of lifetime residential history, it is possible that those agreeing to participate are non-representative of the general population. We do not have

information on cooperation rates by strata. There is also a potential bias in that approximately 25% of the participants who initially consented by telephone could not or did not continue to the in-person exam. While this would affect the estimate of prevalence of hypertension, it has only a minor role in the association of hypertension with the stroke belt exposure measures. In comparison with other cardiovascular cohort studies however, our cooperation rate compares favorably, in particular given that participation involved allowing a stranger into the home and disrobing for an electrocardiogram.

The addition of adolescent (ages 13 to 18) or early adulthood (ages 19 to 30) stroke belt exposure had a similar impact on the ability to predict hypertension as education, income, alcohol use, and smoking, and had only marginally smaller impact than exercise. However, while the difference between exposures at these ages compared to other ages was statistically significant, there was only a marginal increase in the predictive ability. While this difference is small, that exposures to the stroke belt during adolescent or early adulthood are more strongly associated with later prevalent hypertension may offer clues to guide interventions to reduce the burden of the disease.

Our data also indicate risk for hypertension associated with multiple stages of life including the latter stages, especially for black women. Thus, strategies for hypertension prevention should be designed to reach across age, sex and race. It is not clear if the association between early life exposure to the stroke belt causes risk of future cardiovascular disease from vascular damage incurred in early adulthood or whether it is the exposure to poor lifestyle that influences learned habits that extend into later life. More research is needed on what components related to early life in the stroke belt could be contributing to the excess stroke risk later in life.

Our data suggest that after control for risk factors for hypertension, the adolescence period and the young adulthood period of exposure to the stroke belt are most predictive of hypertension later in life. This suggests a window of opportunity to be targeted for intervention to prevent the development of hypertension. It is during these age periods that individuals begin to have more choices and more control over behavioral and lifestyle factors in such areas as diet, physical activity, and smoking that impact the development of hypertension. There are many proven nonpharmacologic approaches to preventing hypertension. (35) Because hypertension is the major contributor to diseases such as stroke, coronary artery disease, and end-stage renal disease that are among the leading causes of mortality, community and environmental strategies to prevent hypertension need to start earlier in life, specifically in adolescence and young adulthood.

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Table 1

Description of the study population by domain of variables used in analyses. Data are presented as number (percentage) of participants unless otherwise indicated; continuous variables are expressed as mean (SD).

Domain	Variable	Strata	Belt	NonBelt
Demographic	Age (mean ±SD)		65.6 ± 9.0	66.6 ± 9.2
	Race - Sex	Black females	2216 (21.5)	2462 (27.2)
		Black males	1187 (11.5)	1559 (17.2)
		White females	3544 (34.4)	2356 (26.0)
		White males	3368 (32.7)	2686 (29.6)
Socioeconomic status	Educational Level	< High school	1392(13.5)	934(10.3)
		HS graduate	2784(27.0)	2324(25.7)
		Some college	2676(25.9)	2513(27.7)
		College graduate	3462(33.6)	3289(36.3)
	Household Income	< $20,000	2010(22.1)	1571(19.6)
		$20 to $35K	2551(28.1)	2292(28.6)
		$35 to $75K	3068(33.8)	2741(34.2)
		>$75,000	1452(16.0)	1415(17.6)
Risk factor	Smoking Status	Never	1443(14.0)	1286(14.2)
		Past	4621(44.9)	3840(42.5)
		Current	4220(41.0)	3902(43.2)
	Alcohol Use	Never	3522(34.7)	2169(24.5)
		Past	1861(18.3)	1653(18.6)
		Current light	4086(40.2)	4373(49.3)
		Current heavy	688(6.8)	672(7.6)
	Physical Activity Level (times per week)	None	3367(33.0)	3072(34.3)
		Light (1-3)	3674(36.0)	3266(36.5)
		Healthy (4+)	3152(30.9)	2622(29.3)
	BMI (mean ± SD)		28.8 ± 5.8	29.0 ± 5.8
Outcome	Hypertension	No	4283(41.5)	3855(42.5)
		Yes	6036(58.5)	5211(57.5)

Table 2

Prevalence of Hypertension by Length of Time in the Stroke Belt within Selected Age Periods of the Participant's Life. (read down columns of exposure) (Note that for "Born" and "Current" stroke belt exposure, the percentage of time in the stroke belt is either "None" or "100%." For the exposures at other age periods, partial percentages of time in the stroke belt are calculated.) (Participants born outside the US are not included in the "born" column. For participants born or living outside the US at any age, their time outside the US was not counted in the denominator or numerator.)

		Measure of Exposure to the Stroke Belt							
		Yes/No		Percent of the Life Period in the Stroke Belt					
Length of Time in the Stroke Belt	**Number of Participants / (% Hypertensive)**	**Born***	**Current†**	**Life time**	**First 12 years of life**	**13 to 18 years old**	**19 to 30 years old**	**31 to 45 years old**	**Last 20 years of life**
No or none of the time period (0%)	**N**	9609	9066	6600	9174	9608	9468	9721	8892
	(% Hypertensive)	(54.1)	(57.5)	(54.9)	(54.1)	(54.6)	(56.3)	(57.4)	(57.6)
1% - 33%	**N**			2943	465	273	1143	545	471
	(% Hypertensive)			(59.5)	(56.6)	(59.0)	(57.4)	(55.2)	(49.5)
34% - 66%	**N**			1903	429	741	1420	754	711
	(% Hypertensive)			(56.3)	(59.9)	(61.0)	(56.8)	(56.9)	(53.9)
67% - 99%	**N**			2779	600	649	1110	695	663
	(% Hypertensive)			(57.8)	(61.2)	(65.9)	(55.9)	(52.1)	(51.4)
Yes or all of the time period (100%)	**N**	9419	10319	5160	8458	7864	6137	7633	8648
	(% Hypertensive)	(62.4)	(58.5)	(61.9)	(62.2)	(61.3)	(61.4)	(59.8)	(59.7)

Notes:

*For the exposure of "Born," participants are either not born in the region (9609 participants of whom 54.1% are hypertensive) and hence do not have this exposure, or are born in the stroke belt (9419 participants of whom 62.4% are hypertensive) and hence do have the exposure. Unlike periods of life where part of the time can be in the stroke belt and part elsewhere, for the "born" exposure, a person is either "completely" born in the stroke belt or "completely" not born in the stroke belt; hence, the exposure is "none" or "all." Data not shown: 357 were born outside the US.

†Similar to the "born" exposure, individuals must be either currently living in the stroke belt or not living in the stroke belt – again a 0% exposure or a 100% exposure. Specifically, 9066 participants lived outside the stroke belt at time of enrollment (of whom 57.5% are hypertensive) and 10,319 lived in the stroke belt at time of enrollment (of whom 58.5% are hypertensive). Again, it is not possible to be partially a current resident of stroke belt or not.

Table 3

Association (logistic regression OR, 95% confidence intervals) between prevalent hypertension and measures of exposure to the stroke belt. The variable category is any exposure vs. none.

	Demographic Model*		SES Model †		Risk Factor Model‡	
Measure of exposure to stroke belt	**OR (95% CI)**	**Wald χ^2**	**OR (95% CI)**	**Wald χ^2**	**OR (95% CI)**	**Wald χ^2**
Born in stroke belt	1.22 (1.14, 1.29)	39.1	1.15 (1.08, 1.23)	18.1	1.18 (1.10, 1.26)	20.5
Currently living in stroke belt	1.22 (1.15, 1.29)	41.6	1.17 (1.20, 1.25)	22.6	1.19 (1.11, 1.27)	24.3
Lifetime in stroke belt	1.30 (1.22, 1.39)	57.4	1.23 (1.14, 1.32)	29.3	1.25 (1.16, 1.35)	30.9
First 12 years of life in stroke belt	1.22 (1.15, 1.30)	39.3	1.15 (1.08, 1.23)	16.7	1.18 (1.10, 1.27)	20.6
Living in stroke belt ages 13-18	1.24 (1.16, 1.32)	46.1	1.18 (1.10, 1.26)	22.1	1.21 (1.12, 1.30)	25.9
Living in stroke belt ages 19-30	1.28 (1.20, 1.37)	55.0	1.21 (1.13, 1.30)	28.8	1.23 (1.14, 1.33)	28.4
Living in stroke belt ages 31-45	1.24 (1.17, 1.32)	45.3	1.18 (1.10, 1.26)	22.3	1.19 (1.11, 1.28)	23.0
Living in stroke belt last 20 years of life	1.25 (1.17, 1.33)	48.2	1.19 (1.11, 1.27)	25.6	1.20 (1.12, 1.29)	26.1

Models:

*Demographic: after control for age, race and sex

†SES: after further adjustment for income, education

‡Risk Factor Model: after further adjustment for smoking, alcohol use, physical activity level, and body mass index. For all models, all measures are significant at $p < 0.0001$; the Wald χ^2 is provided to allow comparison of the relative strength of association of each exposure with hypertension (after adjustment for factors in the model).

Table 4

Results of bootstrap assessment for the significance of the relative strength of association (rank) of the measures of exposure to the stroke belt with the prevalence of hypertension in the "risk factor" model of Table 3.

Rank	All Participants*	White Men	White Women	Black Men	Black women
1st	Lifetime in stroke belt	Lifetime in stroke belt	Lifetime in stroke belt	Ages 13 to 18	Currently in stroke belt
2nd	Ages 13-18 / ages 19-30	Ages 13-18	First 12 years/Ages 31-45	Born in stroke belt	Last 20 years
3rd		Last 20 years of life		First 12 years of life	Ages 19-30
4th	Last 20 years of life	Ages 19-30	Ages 13-18 / Ages 19-30	Lifetime in stroke belt	Lifetime
5th	Currently in stroke belt	Born in stroke belt		Ages 19-30	Ages 31-45
6th	Ages 31-45	First 12 years of life	Last 20 years of life	Currently in stroke belt	Ages 13-18
7th	Born/First 12 years of life	Currently in stroke belt	Born in stroke belt	Last 20 years of life	Born in stroke belt/First 12 years of life
8th		Ages 31-45	Currently in stroke belt	Ages 31-45	

*Interpretation: the odds ratio for lifetime exposure to the stroke belt was significantly larger than the odds ratios for any other exposures. There was not a significant difference between the odds ratio for exposures for ages 13-18 and ages 19-30; however, these two odds ratios were significantly larger than all exposures in 4th to 8th positions. The odds ratio for exposures in the last 20 years of life was significantly larger than current, ages 31-45, born or first 12 years. The odds ratio for currently living in the stroke belt was significantly larger than those odds ratios listed below, and so forth.