

Is the 'Stroke Belt' Worn From Childhood?

Risk of First Stroke and State of Residence in Childhood and Adulthood

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Background and Purpose—Most Stroke Belt studies define exposure based on residence at stroke onset. We assessed whether residence in the Stroke Belt during childhood confers extra stroke risk in adulthood, even among people who left the region.

Methods—Stroke-free Health and Retirement Study participants (n=18 070) followed up (average, 8.4 years) for first stroke (1452 events) were classified as living in 1 of 7 Stroke Belt states in childhood or at study enrollment (average age, 63 years). We used Cox proportional-hazards models to compare stroke risk for people who had never lived in the Stroke Belt with those who had lived there at both ages, in childhood only, or in adulthood only.

Results—Compared with never having lived in the Stroke Belt, the hazard ratio for Stroke Belt residence in both childhood and adulthood was 1.23 (95% CI, 1.06, 1.43) and for Stroke Belt residence in childhood only was 1.25 (95% CI, 1.02, 1.55). Stroke Belt residence at enrollment but not during childhood was not significantly related to stroke risk (hazard ratio=1.01; 95% CI, 0.70, 1.46), but the small sample in this group resulted in wide CIs. Results changed little after risk factor adjustment, including comprehensive adult socioeconomic measures. Subgroup analyses found similar patterns by sex and birth cohort. In contrast, blacks who had lived in the Stroke Belt in childhood only did not appear to have significantly elevated stroke risk compared with blacks who had never lived in the Stroke Belt.

Conclusions—The excess stroke risk for people who had lived in Stroke Belt states during childhood implicates early life exposures in the etiology of the Stroke Belt. (*Stroke*. 2007;38:2415-2421.)

Key Words: cerebrovascular disorders ■ early-life environment ■ epidemiology ■ geography ■ life span
■ Stroke Belt

Efforts to explain the geographic patterns of stroke incidence and mortality in the United States have met with little success.¹ Excess stroke mortality in the southeastern states' Stroke Belt has long been a focus of concern, but since 1968, new areas of excess risk have emerged in the Pacific Northwest and elsewhere.² The evolving geographic pattern not only partially reflects the differential rates of change in recognition³ and treatment⁴ of stroke but also suggests that social determinants play an important role in stroke mortality rates. Recent research suggests that exposure to risk factors operate throughout the life span to influence stroke risk.⁵⁻⁸ Of the many explanations for the Stroke Belt examined to date, none provide adequate explanations for geographic differences.¹ Part of the puzzle arises because most studies define Stroke Belt exposure on the basis of state of residence at the time of stroke onset and have not incorporated information on place of residence earlier in life. If biologic vulnerabilities evolve throughout the life span, exposure to the Stroke Belt during early life might cause an increased risk of stroke later in life. This question needs to be addressed in the context of a longitudinal study. The only 2 prior studies assessing the role of early-life exposure in the Stroke Belt phenomenon were based

on samples drawn from a single state rather than national data.^{5,9} No previous national studies have assessed how exposure to the US Stroke Belt at different points in the life span are related to subsequent stroke risk, which has implications for the development of promising intervention strategies.¹⁰

On the basis of follow-up data from the Health and Retirement Study (HRS), we assessed whether childhood residence in 1 of 7 Stroke Belt states conferred extra stroke risk in middle to old age, even among individuals who no longer lived in the Stroke Belt. We compared the excess risk associated with Stroke Belt residence in childhood only with that for individuals who remained in the Stroke Belt throughout life or immigrated to the Stroke Belt after childhood. HRS has several strengths for addressing this research topic: it is a large, national, longitudinal study of older Americans and includes information on place of residence at study enrollment, place of residence in childhood, and unusually comprehensive socioeconomic status assessments.

Subjects and Methods

Study Population

HRS is a longitudinal survey of a national sample of US adults age 50+ years and their spouses. Study details are provided else-

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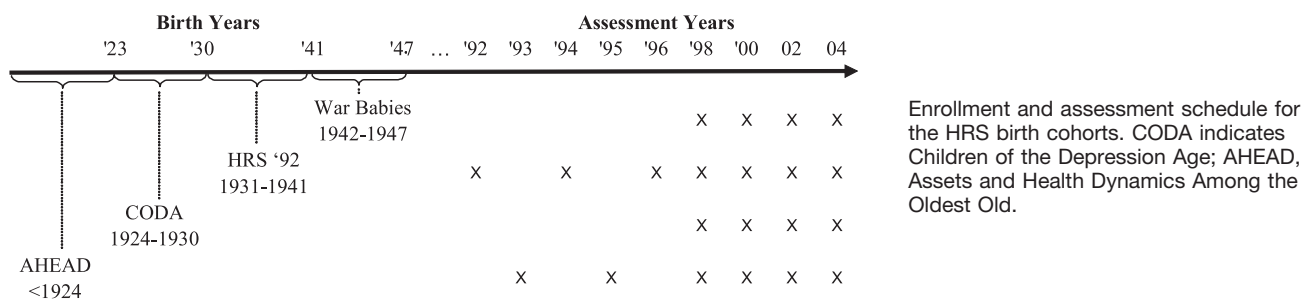
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Enrollment and assessment schedule for the HRS birth cohorts. CODA indicates Children of the Depression Age; AHEAD, Assets and Health Dynamics Among the Oldest Old.

where.¹¹⁻¹³ Enrollment waves occurred in 1992, 1993, and 1998, staggered by birth cohort (the Figure). Biennial interviews (or proxy interviews for decedents) were conducted through 2004. We included HRS participants born in 1900 to 1947 who were age 50+ years and stroke-free at their baseline interview.

From 22 582 age-eligible respondents interviewed in person at baseline, we excluded 323 (1.4%) because of unknown stroke status at enrollment, 1068 (4.7%) who reported prevalent stroke at enrollment, 254 (1.1%) with an unknown last interview date, 1760 (7.8%) with unknown state of childhood residence, and 1107 (4.9%) cases with missing adult risk factor information. The final sample comprised 18 070 respondents reporting 1452 events.

Data Collection and Measurement

At enrollment (average age, 63 years), respondents identified their state of current residence and the state of residence "most of the time when you were in high school." Participants who did not attend high school were asked about residence during grade school or "about age 10." Respondents also reported states of birth. For respondents who reported birth state but not state of childhood residence ($n=1205$), we classified Stroke Belt residence in childhood based on birth state. Participants were classified as living in 1 of 7 Stroke Belt states (North Carolina, South Carolina, Georgia, Tennessee, Arkansas, Mississippi, or Alabama) in childhood and at study enrollment, creating 4 exposure categories: not living in the Stroke Belt at either time, Stroke Belt residence in childhood only, Stroke Belt residence at enrollment only, or living in the Stroke Belt both in childhood and at enrollment. These states were selected as defining the Stroke Belt for these analyses because the recent US Department of Health and Human Services' Stroke Belt Elimination Initiative targets these states.¹⁴

We considered 3 sets of covariates: demographic (age, race, Hispanic ethnicity, and sex); socioeconomic (mother's education [<8 years, ≥ 8 years, missing]; father's education [<8 years, ≥ 8 years, missing]; father's occupation [0 to 3, military, farming, missing]; retrospective self-reported childhood health [a 5-point scale from poor to excellent, missing]; years of education completed; household income at study enrollment [in 1992 dollars, adjusted for household size and logged]; and household wealth at study enrollment [in 1992 dollars, adjusted for household size and logged]); and cardiovascular risk factors (first available report of current smoking status, body mass index, vigorous physical activity [dichotomized at 3+ times per week], and self-reported baseline diagnoses of hypertension, diabetes, and heart disease).

Stroke Outcomes

Incident events were defined as first nonfatal or fatal strokes, based on self-report or proxy report of a doctor's diagnosis. Reports of temporary ischemic attacks were not coded as strokes. For deceased participants, interviews were conducted with proxy informants, predominantly spouses. At each assessment, respondents were asked the month and year of stroke, except for 1994 interviews, when only year was recorded. For these events, December was assigned for strokes in 1992, April for 1993 strokes, and January for 1994 strokes. Stroke events for which the exact timing between 2 interview waves was uncertain ($n=278$) were assigned the median stroke date for events reported by other participants in the same interview wave.

Eliminating these cases from the analyses did not alter the main results presented here.

Methods of Analysis

Cox proportional-hazard models were fitted for comparison of the risk of incident stroke for 4 groups: those who had lived in Stroke Belt states in both childhood and at enrollment; in childhood only; at enrollment only; or at neither time point. Individuals who had not lived in the Stroke Belt in either childhood or at enrollment were the reference group. We present hazard ratios (HRs) and 95% CIs for comparing stroke risk in each exposure category with stroke risk in the reference group. Survival was defined as time from the baseline interview to first stroke, proxy-reported death due to other causes, or last interview date. Primary analyses were adjusted for demographic variables only. To explore whether measured risk factors could account for geographic disparities in stroke in this sample, we present models adjusted for socioeconomic characteristics and cardiovascular risk factors. We also present models stratified by sex, race, and birth year (1900 to 1930 versus 1930 to 1947). Analyses were conducted with SAS 9.1.

Results

Participant characteristics are summarized in Table 1. In 151 569 person-years of follow-up, 1452 first strokes occurred, with the highest rate affecting participants who had lived in the Stroke Belt in childhood but not at enrollment.

In models adjusted for demographics, participants who had lived in the Stroke Belt in childhood but not at enrollment had a 25% (95% CI, 1.02, 1.55) excess risk of stroke compared with participants who did not live in the Stroke Belt at either time point (Table 2). Stroke Belt residence in both childhood and at study enrollment was associated with a similar HR of 1.23 (95% CI, 1.06, 1.43). In contrast, individuals who lived in the Stroke Belt at study enrollment but not in childhood did not have a significantly elevated stroke risk (HR=1.01; 95% CI, 0.70, 1.46), although the 95% CI includes both the null and the parameter estimates for the other Stroke Belt categories. These estimates changed little after adjustment for childhood and adult socioeconomic status or for measured cardiovascular risk factors. In a fully adjusted model, respondents who had lived in the Stroke Belt in childhood only had 21% excess risk, and respondents who lived in the Stroke Belt at both time points had 15% excess risk compared with those who did not live in the Stroke Belt at either time point. Stroke risk for those who lived in the Stroke Belt at enrollment only did not differ significantly from either those who never lived in the Stroke Belt or those who lived in the Stroke Belt in childhood only or at both time points.

To determine whether the pattern of excess risk could be attributed to people who only recently moved in or out of the Stroke Belt, we repeated the analyses after restricting to

TABLE 1. Characteristics of HRS Participants Born Between 1900 and 1947, by Timing of Stroke Belt residence

	Neither in Childhood nor at Enrollment		Childhood Only		Enrollment Only		Both in Childhood and at Enrollment		P Value†
	n	%	n	%	n	%	n	%	
n	14 238	100	1064	100	426	100	2342	100	
Mean years of follow-up (SD)	8.4	(3.5)	8.4	(3.5)	8.4	(3.6)	8.4	(3.5)	
Total person-years of follow-up	119 306		8952		3600		19 711		
Incident strokes	1097		114		29		212		
Crude stroke rate/1000 person-years	9.2		12.7		8.1		10.8		
Mean years of education (SD)	12.3	(2.9)	10.9	(3.6)	12.7	(2.9)	11.0	(3.6)	<0.01
Median wealth (IQR)*	79 196	(146 499)	34 093	(87 584)	56 725	(109 743)	41 802	(91 337)	<0.01
Median income (IQR)*	20 647	(24 613)	15 466	(23 291)	23 731	(23 000)	15 519	(21 787)	<0.01
Mean age at enrollment, y (SD)	63.8	(10.2)	63.5	(10.1)	61.4	(9.9)	62.2	(9.9)	<0.01
Male	6351	45	429	40	206	48	983	42	<0.01
Black	1229	9	632	59	48	11	747	32	<0.01
Mother's education									<0.01
<8 years	3361	24	360	34	89	21	781	33	
8+ years	9523	67	564	53	295	69	1237	53	
Unknown	1354	10	140	13	42	10	324	14	
Father's education									<0.01
<8 years	3957	28	407	38	125	29	876	37	
8+ years	8491	60	468	44	237	56	1003	43	
Unknown	1790	13	189	18	64	15	463	20	
Father's occupation									<0.01
Professional/managerial	1559	11	77	7	55	13	124	5	
Sales/clerical	1302	9	27	3	32	8	135	6	
Skilled labor	2761	19	93	9	77	18	280	12	
Unskilled labor	2893	20	163	15	80	19	342	15	
Farming	2440	17	374	35	71	17	834	36	
Unknown	3200	22	321	30	103	24	620	26	
Mean self-reported childhood health (SD)	1.8	(1.0)	1.9	(1.0)	1.8	(1.0)	2.0	(1.1)	<0.01
Hypertension	5879	41	540	51	186	44	1138	49	<0.01
Diabetes	1543	11	155	15	49	12	299	13	<0.01
Heart disease	2567	18	222	21	73	17	427	18	0.13
Current smoker	2902	20	266	25	107	25	506	22	<0.01
Vigorous activity	3891	27	218	20	126	30	554	24	<0.01
Mean BMI (SD)	26.8	(5.0)	27.5	(5.1)	26.6	(4.9)	27.1	(5.3)	<0.01

IQR indicates interquartile range; BMI, body mass index. Sample members were stroke-free at baseline. Some percentages do not sum to 100 due to rounding. Unless otherwise noted, for each group, the left-hand column refers to the No. of respondents and the right-hand column refers to the percentage of respondents with the specified characteristic. The Stroke Belt exposure patterns for Hispanics (n=835) and individuals whose fathers were in the army (n=107) are omitted.

*Income and wealth values were standardized by dividing by the square root of household size and are reported as median (IQR).

†P-value testing the hypothesis that Stroke Belt residence category and each covariate are statistically independent, calculated by ANOVA for continuous variables and χ^2 tests for categorical variables.

participants who reported living “in or around” their current residence for 20+ years (n=9226, 887 events). In this subset, stroke risk was elevated for participants residing in the Stroke Belt in childhood only (HR=1.43; 95% CI, 1.11, 1.86) or both in childhood and at enrollment (HR=1.26; 95% CI, 1.05, 1.52) after adjustment for demographic characteristics. Although the CIs were too wide to rule out a similar effect, there was no evidence that Stroke Belt residence at enrollment but not in childhood was associated with excess stroke risk (HR=0.94; 95% CI, 0.53, 1.67) in this subgroup.

Disentangling place of birth and place of childhood residence is difficult because only 427 participants (with 31 strokes) reported migrating into the Stroke Belt between birth and childhood, and 154 (18 strokes) reported moving out of the Stroke Belt between birth and childhood. In a demographics-adjusted model simultaneously adjusting for Stroke Belt birth and childhood residence in the Stroke Belt, place of birth was not significantly associated with excess risk (HR=0.86; 95% CI, 0.64, 1.16), but Stroke Belt residence in childhood predicted a heightened stroke risk

TABLE 2. HRs for First Incident Stroke by Timing of Stroke Belt Residence in HRS Participants Born Between 1900 and 1947

	Demographics Adjusted*		SES Adjusted†		Cardiovascular Risk Factor Adjusted‡	
	HR	(95% CI)	HR	(95% CI)	HR	(95% CI)
All strokes						
Neither childhood nor enrollment	Reference		Reference		Reference	
Childhood only	1.25	(1.02, 1.55)	1.22	(0.99, 1.52)	1.21	(0.98, 1.49)
Enrollment only	1.01	(0.70, 1.46)	0.97	(0.67, 1.40)	0.94	(0.65, 1.36)
Both childhood and enrollment	1.23	(1.06, 1.43)	1.20	(0.97, 1.33)	1.15	(0.98, 1.35)

Each model used all 18 070 eligible HRS sample members, with 1452 events during 151 559 person-years of follow-up.

*Adjusted for demographic covariates: race, Hispanic ethnicity, baseline age, age squared, and sex.

†Adjusted for demographic and socioeconomic status (SES) covariates: mother's education, father's education, father's occupation, self-reported childhood health, own education, baseline income, and baseline wealth.

‡Adjusted for demographic, SES, and cardiovascular risk factors: first available report of current smoking status, body mass index, vigorous physical activity, and self-reported baseline diagnoses of hypertension, diabetes and heart disease.

(HR=1.43; 95% CI, 1.04, 1.95). The pattern of excess risk was very similar for men and women, although many CIs included the null (Table 3). The interaction of sex and Stroke Belt residence categories was not significant.

For whites, Stroke Belt residence in childhood only (HR=1.64; 95% CI, 1.24, 2.17) or in both childhood and at enrollment (HR=1.19; 95% CI, 0.99, 1.43) were associated with excess stroke risk, although statistical significance was marginal for those exposed in both childhood and enrollment. Stroke Belt residence at enrollment only was not significantly associated with excess risk. Among blacks, the CIs for all 3 exposure groups included the null. In particular, blacks who had lived in the Stroke Belt in childhood only did not report substantially increased stroke risk (HR=1.07; 95% CI, 0.78, 1.46). The interaction between race and Stroke Belt residence in childhood only was statistically significant ($P<0.01$), indicating deviation from a multiplicative model such that early-life Stroke Belt exposure conferred less relative harm for blacks than whites.

To explore whether the pattern of geographic disparities declined for more recent birth cohorts, we stratified the sample into those born through 1930 and those born in 1931 or later (this cutpoint corresponds to the HRS enrollment strategy; the Figure). The pattern of excess risk was similar in the earlier and later birth cohorts: excess risk associated with Stroke Belt residence in childhood only or in childhood and enrollment but little or no excess for those residing in the Stroke Belt at enrollment only. For respondents born before 1931, the parameter estimates were not statistically significant for any of the exposure groups. Nonetheless, the parameter estimates in the stratified models, when expressed in terms of absolute excess stroke risk, suggest that the effects of birth cohort and Stroke Belt residence in childhood are very nearly additive; the excess stroke rate in the early birth cohorts exposed to the Stroke Belt is similar to the excess stroke rate in the later birth cohorts.

Discussion

Childhood Stroke Belt residence predicted excess risk of first stroke among Americans age 50+ years, regardless of place of residence in adulthood. The excess risk associated with

childhood Stroke Belt residence remained after statistical adjustment for core demographic characteristics, childhood and adult socioeconomic status, and adult cardiovascular risk factors. Although CIs were wide, similar patterns in the parameter estimates emerged in subgroup analyses for men and women, for whites, and for individuals born before or after 1930. For blacks, Stroke Belt residence at enrollment appeared more relevant than place of residence in childhood. Overall, results suggest that the association between adult residence and stroke risk partly reflects risks incurred earlier in life.

Study Limitations

The HRS is uniquely suited for the current investigation, but some limitations should be considered. Prior research suggested an imperfect correspondence between self-reported and clinically validated strokes.^{15,16} Many individuals report stroke symptoms without clinical diagnoses, although this pattern differs little between Stroke Belt residents and others.¹⁷ Similarly, a recent evaluation of hypertension awareness found no difference between Stroke Belt residents and others.¹⁸ Although the incident stroke rates that we found are consistent with those from prior US studies^{19–21} (eg, the HRS rate for ages 50 to 64 was 4.8/1000 and 9.4/1000 for ages 65 to 75), incomplete stroke ascertainment could bias our results if it differed by state of childhood or adult residence. If individuals who had lived in the Stroke Belt in childhood were more likely to report strokes than others, this could account for our results. The most plausible explanation for such differential reporting is related to medical access and norms, which seem more likely to influence estimates for adult residence than to affect estimates for childhood Stroke Belt residence.

We have no data on stroke subtypes, on place of residence between childhood and study enrollment, or on several important stroke risk factors. Because the majority of strokes are ischemic, our findings are likely to be largely driven by the pattern for ischemic stroke. Differences in the geographic pattern of stroke subtypes potentially offer insight into likely mediators of Stroke Belt risk, but 2 US studies reported no significant geographic variations in subtype frequency,^{22,23} and the relation between early-life risk factors and stroke subtype is

TABLE 3. HRs for First Incident Stroke by Timing of Stroke Belt Residence in HRS Participants Born Between 1900 and 1947, stratified by Sex, Race, and Birth Year

Subgroup (Events/Person-Years)	Demographics Adjusted*		SES Adjusted†		Cardiovascular Risk Factor Adjusted‡	
	HR	(95% CI)	HR	(95% CI)	HR	(95% CI)
Males (633/65 452)						
Neither childhood nor enrollment	Reference		Reference		Reference	
Childhood only	1.20	(0.86, 1.66)	1.15	(0.82, 1.60)	1.16	(0.83, 1.62)
Enrollment only	0.85	(0.48, 1.51)	0.81	(0.46, 1.44)	0.79	(0.45, 1.41)
Both childhood and enrollment	1.21	(0.95, 1.54)	1.07	(0.84, 1.37)	1.07	(0.84, 1.37)
Females (819/86 107)						
Neither childhood nor enrollment	Reference		Reference		Reference	
Childhood only	1.30	(0.99, 1.71)	1.29	(0.97, 1.70)	1.24	(0.94, 1.64)
Enrollment only	1.15	(0.71, 1.86)	1.11	(0.68, 1.79)	1.08	(0.67, 1.76)
Both childhood and enrollment	1.25	(1.02, 1.52)	1.19	(0.97, 1.46)	1.22	(0.99, 1.50)
Whites (1192/129 227)						
Neither childhood nor enrollment	Reference		Reference		Reference	
Childhood only	1.64	(1.24, 2.17)	1.61	(1.22, 2.14)	1.53	(1.15, 2.03)
Enrollment only	0.89	(0.58, 1.36)	0.85	(0.55, 1.29)	0.83	(0.54, 1.26)
Both childhood and enrollment	1.19	(0.99, 1.43)	1.09	(0.91, 1.32)	1.11	(0.92, 1.34)
Blacks (260/22 343)						
Neither childhood nor enrollment	Reference		Reference		Reference	
Childhood only	1.07	(0.78, 1.46)	1.06	(0.77, 1.46)	1.07	(0.78, 1.48)
Enrollment only	1.88	(0.87, 4.05)	1.90	(0.88, 4.11)	1.79	(0.83, 3.87)
Both childhood and enrollment	1.28	(0.96, 1.71)	1.19	(0.87, 1.62)	1.21	(0.89, 1.66)
Birth year, pre-1931 (934/58 815)						
Neither childhood nor enrollment	Reference		Reference		Reference	
Childhood only	1.17	(0.88, 1.55)	1.14	(0.86, 1.52)	1.13	(0.85, 1.50)
Enrollment only	0.93	(0.56, 1.55)	0.91	(0.55, 1.52)	0.89	(0.53, 1.49)
Both childhood and enrollment	1.13	(0.92, 1.39)	1.06	(0.86, 1.31)	1.08	(0.87, 1.34)
Birth year, 1931 or later (518/92 755)						
Neither childhood nor enrollment	Reference		Reference		Reference	
Childhood only	1.47	(1.07, 2.02)	1.44	(1.04, 1.99)	1.44	(1.04, 1.98)
Enrollment only	1.15	(0.68, 1.97)	1.09	(0.64, 1.87)	1.09	(0.64, 1.87)
Both childhood and enrollment	1.40	(1.11, 1.77)	1.28	(1.00, 1.63)	1.29	(1.01, 1.65)

*Adjusted for demographic covariates: race, Hispanic ethnicity, baseline age, age squared, and sex.

†Adjusted for demographic and socioeconomic status (SES) covariates: mother’s education, father’s education, father’s occupation, self-reported childhood health, own education, baseline income, and baseline wealth.

‡Adjusted for demographic, SES, and cardiovascular risk factors: first available report of current smoking status, body mass index, vigorous physical activity, and self-reported baseline diagnoses of hypertension, diabetes and heart disease.

also uncertain.^{24,25} Misreporting or measurement error in socioeconomic status and cardiovascular risk factor measures is a concern, but HRS includes more extensive socioeconomic status measures than most other studies. Adjusting regression models for covariates influenced by the primary exposure identifies direct effects only under strict assumptions²⁶; thus, the estimates of early-life effects adjusted for adult characteristics should be interpreted with caution.

The lowest level of geography available to us is the state. Prior research has shown substantial heterogeneity in stroke risk within states.²² We found no significant elevation in stroke risk among those who migrated to the Stroke Belt between childhood and study enrollment. This may indicate

that Stroke Belt exposure in adulthood is harmless, but selective migration may also contribute to this finding.²⁷ Stroke Belt in-migrants may be moving to relatively urban, low-stroke-risk southern communities and thereby avoid excess risk. Most research on migration suggests migrants tend to be healthier than the nonimmigrants they leave behind. Thus, we would expect this bias to lead to an underestimation of the harmful effects of Stroke Belt residence in childhood only or adulthood only and an overestimation of the harmful effects of Stroke Belt residence in both childhood and adulthood.

Finally, although the HRS is among the largest panel studies of US elderly, the number of events in many strata is small, resulting in wide CIs for many parameter estimates. The

nonsignificant relations between Stroke Belt residence in adulthood only and stroke risk may be attributable to small samples.

Comparisons With Previous Studies

Our results are consistent with prior research on the Stroke Belt but extend these findings and highlight the importance of exposure timing. Excess strokes in the southeastern United States have been shown for both adult and pediatric strokes.²⁸ To our knowledge, this is the first study of a national US sample to directly compare adult versus childhood Stroke Belt residence as risk factors for adult stroke. Most previous research on the Stroke Belt used state of residence at stroke onset to define Stroke Belt exposure,^{29,30} although research in the United Kingdom^{6,31–33} and US studies in South Carolina⁵ and New York^{5,9} have focused on place of birth as a risk factor. Substantial evidence links social deprivation in the prenatal period, infancy, or childhood to enduring physiologic or behavioral changes that increase stroke risk decades later,^{25,34–38} although some studies do not support this.³⁹

These findings may also help explain previous reports of evolving geographic patterns of risk.² Places that received many immigrants from Stroke Belt states during 20th-century migrations may now report excess stroke risk. Future research could examine this by testing whether excess stroke in newly emerging areas of risk (eg, Oregon and Washington) is largely experienced by immigrants from southern states or whether Pacific Northwest natives are also at risk.

Interpretation of Findings

These findings suggest that Stroke Belt residence predicts excess stroke risk because of exposures experienced in early life. Place of residence may affect stroke through access to medical care, physical risks associated with environmental conditions, social norms affecting behaviors, socioeconomic conditions created by local macroeconomic factors, or psychosocial pathways stemming from features of social organization in communities.⁴⁰ If quality of medical care were the driving factor in the Stroke Belt phenomenon, the risk might be more strongly associated with place of current residence, rather than place of residence in childhood. Behavioral norms regarding diet, physical activity, and smoking are profoundly influenced by childhood social conditions, so these factors are prime candidates to explain the Stroke Belt risk.⁴¹ Although prior evidence is mixed,^{1,42–45} our analyses suggest that Stroke Belt risk was independent of all of these variables. Although incomplete measurement of cardiovascular risk factors could lead to some underestimation, the small changes in estimated HRs after adjustment suggest that these variables are inadequate to explain the excess risk. Childhood socioeconomic status shows persistent relations with adult cardiovascular disease,⁴⁶ possibly owing to education attainment and quality, employment options, and income and wealth,⁴⁷ which influence conventional risk factors such as smoking, obesity,⁴⁸ diabetes, and alcohol consumption.⁴⁹ Adjustment for an unusually comprehensive set of socioeconomic status indicators hardly budged the effect estimates for Stroke Belt exposure; however, measures of birth weight³⁴ and related exposures that might be important for stroke^{6,50} were unavailable in the HRS data set.

Future Research

The process of elimination suggests future research should focus on environmental risks associated with geography, contextual risks arising from features of the community organization, and psychosocial risk factors not considered here, such as depression and social isolation,^{51–53} to account for the excess risk of stroke in southerners. Common southern ethnic heritage and shared genetic risk are sometimes posited as possible contributors to the Stroke Belt.⁵⁴ Genetic explanations are not supported by our findings that residing in the Stroke Belt during childhood was a more important risk than being born in the Stroke Belt. However, given the small number of subjects who moved into or out of the Stroke Belt between birth and childhood, our evidence on the relatively greater importance of place of childhood residence should be interpreted cautiously. Furthermore, genetic risks shared by black and white southerners but not African-American northerners are surprising because a large proportion of northern black families immigrated from the south from 1910 to 1970.⁵⁵

Conclusions

The effect of the Stroke Belt on the burden of stroke in the population is large, but our ability to understand and thus mitigate the phenomenon is limited by the available data. Studies currently under way may illuminate the puzzle by providing more detailed information about risks accumulated during the life span. Microlevel physical and social environmental features, rather than individual-level social conditions and behaviors, must be considered. We found that individuals who had resided in the Stroke Belt in childhood experienced heightened stroke risk at ages 50 and older, even if they had migrated out of the Stroke Belt, implicating early-life exposures in the genesis of the Stroke Belt.

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Disclosures

None.

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