Birth and adult residence in the Stroke Belt independently predict stroke mortality

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ABSTRACT

Background: Understanding how the timing of exposure to the US Stroke Belt (SB) influences stroke risk may illuminate mechanisms underlying the SB phenomenon and factors influencing population stroke rates.

Methods: Stroke mortality rates for United States–born black and white people aged 30–80 years were calculated for 1980, 1990, and 2000 for strata defined by birth state, state of adult residence, race, sex, and birth year. Four SB exposure categories were defined: born in a SB state (North Carolina, South Carolina, Georgia, Tennessee, Arkansas, Mississippi, or Alabama) and lived in the SB at adulthood; non-SB born but SB adult residence; SB-born but adult residence outside the SB; and did not live in the SB at birth or in adulthood (reference group). We estimated age-, sex-, and race-adjusted odds ratios for stroke mortality associated with timing of SB exposure.

Results: Elevated stroke mortality was associated with both SB birth and, independently, SB adult residence, with the highest risk among those who lived in the SB at birth and adulthood. Compared to those living outside the SB at birth and adulthood, odds ratios for SB residence at birth and adulthood for black subjects were 1.55 (95% confidence interval 1.28, 1.88) in 1980, 1.47 (1.31, 1.65) in 1990, and 1.34 (1.22, 1.48) in 2000. Comparable odds ratios for white subjects were 1.45 (95% confidence interval 1.33, 1.58), 1.29 (1.21, 1.37), and 1.34 (1.25, 1.44). Patterns were similar for every race, sex, and age subgroup examined.

Conclusion: Stroke Belt birth and adult residence appear to make independent contributions to stroke mortality risk. ***Neurology***® **2009;73:1858–1865**

GLOSSARY

CI = confidence interval; **SB** = Stroke Belt.

The geographic patterns of stroke in the United States are an enduring puzzle.[1] Residents of the southeastern states are 20%–50% more likely to die of strokes than residents of the rest of the country, but the Stroke Belt (SB) has not been adequately explained by conventional stroke risk factors, social resources, or medical care access.[1,2]

We know little about how the timing of SB exposure influences stroke risk. Most prior studies define SB exposure based on place of residence at stroke onset. Biologic and epidemiologic evidence suggests risk begins to accumulate in early life, however,[3-6] when many cardiovascular risk factors are first established.[7-14] Studies comparing in-migrants to natives of South Carolina[4] and New York[15] suggest the importance of place of birth or childhood residence. One national cohort study, based on self-reported stroke, also implicated place of childhood residence in establishing stroke risk.[16] Finally, rates of circulatory disease and other causes of mortality differ by census division of birth and census division of death.[17,18] If early life envi-

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ronments have lasting effects on stroke risk, this may help illuminate the persistent SB puzzle and prioritize promising avenues for stroke reduction.[19]

We use national data on stroke mortality from 1980, 1990, and 2000, cross-classified by state of birth and state of adult residence, to assess the stroke risk associated with SB exposure at birth, in adulthood, both, or neither.

METHODS

Data sources. The 1980, 1990, and 2000 US Census Public Use Microsample data, upweighted to represent the actual US population, were used to define the at-risk population.[20] Samples were restricted to individuals born in any of the 49 US states (excluding Hawaii) or the District of Columbia (DC); residing in a US state or DC and ages 30–80 on the census date; and self-reporting race as black or white.

Mortality records for 1980, 1990, and 2000 were obtained from the National Center for Health Statistics underlying cause of death files.[21] Stroke deaths were identified as those mortality records with underlying causes of death ICD-9 codes 430–438 (1980 and 1990) or underlying cause of death ICD-10 codes I60–I69, G45.8, or G45.9 (2000).[22] Records were included only for deaths that would have been included in the census sample when the individual was alive (i.e., born in a US state, resided in a US state at death, age 30–80 on census date, and reported race black or white). Mortality records and census data were linked for each stratum defined by the covariates and exposures in each analysis (described below), including state of birth, state of residence at death (linked to census state of residence), race, sex, and birth year.

Data collection and measurements. We defined the SB as including 7 states (North Carolina, South Carolina, Georgia, Tennessee, Arkansas, Mississippi, or Alabama) corresponding to the US Department of Health & Human Services Stroke Belt Elimination Initiative.[23] We considered 4 primary exposure categories: born in the SB and residing in the SB during adulthood (the doubly exposed); born in the SB but did not live there in adulthood (i.e., at census date; the SB out-migrants); born outside the SB but resided in the SB in adulthood (the SB in-migrants); or neither born in the SB nor lived there in adulthood. We emphasize that we only have information on 2 time points (birth and the census/death date) and those described as nonexposed may in fact have lived in the SB for long periods in between those 2 times.

The role of education in geographic disparities is of special interest, but individual education is frequently missing from 1980 and 1990 death certificates, with a strong geographic patterning. In 2000, missing rates were lower (39 states had education information on over 95% of death certificates) and we imputed educational attainment (<12 years, 12 years, and >12 years) when missing. Imputations were based on other stroke deaths, matched on race, sex, year of birth, state of birth, and SB residence. In 3 states with a high fraction of missing data, we used education data from an adjacent state with a similar population education distribution (for South Dakota, we imputed education values based on the distribution in North Dakota; for Kentucky, we used Tennessee; and for Georgia, we used North Carolina). We excluded people who were born or died in Rhode Island from the education-adjusted analyses because missingness was extensive and no adjacent state had a similar educational distribution. This approach increases uncertainty in the effect estimates for the education adjusted models; we used multiple imputations to incorporate this uncertainty into the confidence intervals (CIs).

Analyses. Mortality rates were calculated by linking mortality and population data within each stratum defined by the exposure and covariates. We first compared crude stroke mortality rates for white and black subjects in 4 cross-classified SB exposure categories. We then used logistic regression (weighted to represent the eligible US population) to estimate race-stratified odds ratios for stroke mortality for the following comparisons:

1. SB born vs all others, adjusting for age and sex.
2. SB born vs all others, adjusting for age, sex, and state of adult residence.
3. SB residence in adulthood vs all others, adjusting for age and sex.
4. SB residence in adulthood vs all others, adjusting for age, sex, and state of birth.

Place of birth influences educational attainment and independently affects health, so models with place of adult residence alone (e.g., model 3) are potentially confounded by place of birth. We present these results for comparison with other studies. However, neither educational attainment nor place of adult residence confounds place of birth, although they may mediate its effects. In consideration of the temporal sequence of exposures, for the year 2000, we repeated model 1 above adding adjustment for education. CIs for models including education were calculated using multiple imputation methods (5 imputed data sets) to reflect uncertainty arising from the missing data.[24]

Finally, we used logistic regression to compare the 3 SB exposure categories (SB doubly exposed; SB out-migrants; SB in-migrants) vs those who did not live in the SB at birth or in adulthood, allowing assessment of the independent or interactive effects of SB birth and adult residence. We present models stratified by race, sex, and age (<60, ≥60 years).

We provide conservative CIs, calculated adjusting for clustering within the state of residence, although the clustering adjustment made little difference to our findings. In all models, age is modeled using both linear and quadratic terms. Analyses were conducted using SAS 9.2.

RESULTS

In 1980, eligible adults who were born in the SB and lived in the SB in adulthood had a higher stroke mortality rate (122/100,000) than any other exposure group, including SB out-migrants (115), SB in-migrants (63), or those who were neither born in the SB nor lived in the SB in adulthood (78) (table 1). This pattern of excess mortality associated with SB exposure at birth and adulthood was evident in every year among both white and black subjects. Mortality rates declined from 1980 to 2000 in every SB exposure category. Black subjects who were born in the SB and lived in the SB in adulthood had the greatest absolute mortality decline, from 211 in 1980 to 96 in 2000. Over 95% of black subjects who resided in the SB in 1980 were born there, compared to 82% of white subjects who resided in the SB in adulthood.

Adjusting for age and sex, black subjects born in the SB (table 2, model 1) had a 28% (95% CI 1.13,

Table 1 Crude stroke mortality rates by race, Stroke Belt birth, and Stroke Belt adult residence, 1980, 1990, 2000

	Born in the Stroke Belt						Not born in the Stroke Belt					
	Resided in the Stroke Belt at adulthood			Did not reside in the Stroke Belt at adulthood			Resided in the Stroke Belt at adulthood			Did not reside in the Stroke Belt at adulthood		
	Stroke fatalities	Population (1,000)	Rate per 100,000 population	Stroke fatalities	Population (1,000)	Rate per 100,000 population	Stroke fatalities	Population (1,000)	Rate per 100,000 population	Stroke fatalities	Population (1,000)	Rate per 100,000 population
Full sample												
1980	13,238	10,815	122	6,499	5,676	115	1,245	1,965	63	61,704	79,562	78
1990	10,102	12,206	83	5,013	5,658	89	1,376	3,227	43	48,797	93,438	52
2000	9,956	13,495	74	4,582	5,206	88	1,944	4,930	39	48,956	103,223	47
White												
1980	8,140	8,393	97	2,558	2,979	86	1,120	1,854	60	56,241	74,701	75
1990	6,311	9,246	68	1,966	3,005	65	1,222	2,995	41	44,178	86,667	51
2000	6,540	9,926	66	1,903	2,904	66	1,748	4,397	40	43,848	94,371	46
Black												
1980	5,098	2,422	211	3,941	2,696	146	125	111	112	5,463	4,861	112
1990	3,791	2,961	128	3,047	2,653	115	154	232	66	4,619	6,771	68
2000	3,416	3,569	96	2,679	2,302	116	196	533	37	5,108	8,853	58

Restricted to US residents age 30–80 born in the United States (excluding Hawaii). Population rates calculated using Integrated Public Use Microdata Series 1980 census 5% samples and 1990 and 2000 census weighted sample.

1.44) increased odds of stroke mortality in 1980, a 28% (95% CI 1.16, 1.40) increased odds of stroke mortality in 1990, and a 22% elevation in odds of stroke mortality in 2000 (1.13, 1.32) compared to black subjects born outside the SB. The excess was similar for white subjects born in the SB. Adjustment for state of adult residence attenuated the effect estimates, but SB birth remained a significant predictor of stroke mortality in all years for both black and white subjects (table 2, model 2).

Table 2 Odds ratios (OR) for stroke mortality associated with Stroke Belt birth and Stroke Belt adult residence for black and white subjects in 1980, 1990, and 2000

	1980 mortality				1990 mortality				2000 mortality			
	Black		White		Black		White		Black		White	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Model 1												
Stroke Belt birth	1.28	(1.13, 1.45)	1.23	(1.17, 1.29)	1.28	(1.16, 1.41)	1.27	(1.20, 1.34)	1.22	(1.13, 1.32)	1.30	(1.22, 1.39)
Non Stroke Belt birth	1.00		1.00		1.00		1.00		1.00		1.00	
Model 2*												
Stroke Belt birth	1.25	(1.17, 1.34)	1.14	(1.09, 1.20)	1.21	(1.14, 1.28)	1.19	(1.12, 1.28)	1.17	(1.10, 1.23)	1.19	(1.12, 1.26)
Non Stroke Belt birth	1.00		1.00		1.00		1.00		1.00		1.00	
Model 3												
Stroke Belt adult residence	1.39	(1.25, 1.54)	1.23	(1.16, 1.30)	1.42	(1.30, 1.55)	1.24	(1.18, 1.31)	1.29	(1.20, 1.40)	1.28	(1.21, 1.36)
Non Stroke Belt adult residence	1.00		1.00		1.00		1.00		1.00		1.00	
Model 4†												
Stroke Belt adult residence	1.30	(1.21, 1.40)	1.08	(1.03, 1.13)	1.35	(1.23, 1.48)	1.06	(1.01, 1.12)	1.23	(1.15, 1.32)	1.11	(1.07, 1.16)
Non Stroke Belt adult residence	1.00		1.00		1.00		1.00		1.00		1.00	

All models adjusted for age, age-squared, and sex. Confidence intervals (CI) calculated accounting for clustering on state of residence or state of birth.
*Additionally adjusted for state of adult residence (49 indicator variables).
†Additionally adjusted for state of birth (49 indicator variables).

Adjusting for educational attainment attenuated the association between SB birth and stroke mortality rates in 2000 among black subjects from 1.22 (table 2, model 1) to 1.18 (1.10, 1.26; not shown in tables). For white subjects, education adjustment attenuated the SB birth effect estimate from 1.30 to 1.22 (1.14, 1.31).

SB residence in adulthood was also associated with elevated stroke mortality for both black and white subjects (table 2, model 3, adjusted only for age and sex). The risk associated with SB adult residence appeared to be somewhat larger for black than for white subjects in 1980 and 1990. Adjusting for birth state (table 2, model 4) substantially attenuated the excess mortality for white subjects, but effect estimates for black subjects remained large and statistically significant.

Table 3 compares stroke mortality risk in individuals cross-classified by SB exposure at birth or in adulthood. The greatest excess is experienced by the doubly exposed (born in the SB and lived there in adulthood) with a 34%–55% elevation in odds of stroke death for black subjects and a 29%–45% elevation for white subjects, compared to similar individuals who did not live in the SB at either time point. The excess risk of stroke mortality for SB inmigrants was inconsistent but generally small, with the exception of black subjects in 1990. White subjects born in the SB continued to experience excess mortality risk even if they did not reside in the SB in adulthood, with odds ratios of 1.31 in 1980 and 1.20 in 2000. For black subjects, the excess stroke risk among SB out-migrants was generally smaller: 1.20 in 1980 and only 1.09 in 2000.

Stratifying on race and sex suggests that although SB exposure is harmful for both men and women, the relative excess may be greater for men in both black and white subjects (table 3). SB exposure predicted excess risk in both those under age 60 and individuals 60+.

DISCUSSION Cross-classifying by state of birth and state of residence, we find that excess risk of stroke mortality associated with living in the Southern SB is apparent even among people who were born in the SB but no longer lived there in adulthood. Individuals who were born outside of the SB but lived there in adulthood appear to have modestly elevated odds of stroke mortality compared to individuals who did not reside in the SB at birth or in adulthood. The greatest elevation in odds of stroke mortality was evident in those exposed to the SB at birth and in adulthood. This pattern was evident in all 3 years examined—1980, 1990, and 2000—despite substantial secular declines in absolute stroke mortality rates. SB exposure was associated with excess mortality among men and women; black and white subjects; and those younger or older than 60. In the year 2000, education appeared to contribute but not fully explain the effect of SB birth. These results suggest that SB birth and adulthood both contribute to excess stroke risk for black and white subjects, but for white subjects the contribution of SB birth predominates.

The current analyses are limited by the geographic and temporal detail available in the census and death certificates. There is substantial heterogeneity in stroke risk within states.[25] SB exposure of recent migrants may be misclassified. In the 2000 census, approximately 93% of SB residents reported living in the SB 5 years previously (calculations from Integrated Public Use Microdata Series 5% sample).[20] Similarly, many individuals do not reside long in their birth state; thus state of birth may not accurately represent childhood exposure. However, data from the 1940 Census 1% microsample indicates that the majority of people born in the SB likely resided in the SB at least through childhood. For example, 88% of 15-year-olds lived in their state of birth.[20] Misclassification may also result from incorrect recall of birth state. For the education-adjusted analyses, we used only 3 levels of education to facilitate data linkage, but this rough measure fails to capture the full continuous range of education or multiple dimensions of education quality that probably historically differ in SB states.[26,27]

Cause of death data on mortality records are imperfect and classification rules change over time.[28] The declines in stroke mortality rates evident here may be misleading if the cause of death coding patterns have evolved to give greater emphasis to underlying conditions such as atherosclerosis or diabetes rather than stroke. It is difficult to rule out this possibility with the current data. However, the pattern of reporting changes would have to be highly complex in order for this potential bias to account for our primary finding that childhood and adult residence appear to independently contribute to stroke risk. Although cause of death classification norms may differ regionally, we expect this would be patterned by place of death, not place of birth. Any possible bias from regional differences in cause of death recording should have similar effects for the group who were born and died in the SB as for the group who were born outside the SB but died in the SB. We also have no information on major stroke risk factors, stroke subtypes, or nonfatal stroke, precluding consideration of a number of interesting etiologic questions.[29,30]

Analyses of ecologic data routinely face 2 important methodologic challenges: avoiding ecologic fallacies and disentangling contextual from composi-

Table 3 Odds ratios (OR) for stroke mortality associated with Stroke Belt birth and Stroke Belt adult residence in subgroups stratified by race, race and sex, or age and race in 1980, 1990, and 2000

	Stroke Belt birth; Stroke Belt adulthood		Non-Stroke Belt birth; Stroke Belt adulthood		Stroke Belt birth; non-Stroke Belt adulthood	
	OR	95% CI	OR	95% CI	OR	95% CI
White*						
1980	1.45	(1.33, 1.58)	1.08	(0.97, 1.21)	1.31	(1.23, 1.39)
1990	1.29	(1.21, 1.37)	1.07	(0.97, 1.19)	1.20	(1.12, 1.27)
2000	1.34	(1.25, 1.44)	1.11	(1.03, 1.21)	1.20	(1.13, 1.28)
Black*						
1980	1.55	(1.28, 1.88)	1.13	(0.83, 1.54)	1.20	(1.02, 1.41)
1990	1.47	(1.31, 1.65)	1.49	(1.11, 2.01)	1.11	(1.04, 1.19)
2000	1.34	(1.22, 1.48)	1.01	(0.91, 1.14)	1.09	(1.01, 1.17)
White females						
1980	1.36	(1.25, 1.48)	1.15	(1.03, 1.29)	1.28	(1.19, 1.38)
1990	1.24	(1.16, 1.33)	1.17	(1.05, 1.29)	1.22	(1.14, 1.31)
2000	1.31	(1.22, 1.42)	1.15	(1.04, 1.28)	1.25	(1.16, 1.36)
White males						
1980	1.57	(1.42, 1.73)	1.00	(0.87, 1.14)	1.33	(1.21, 1.46)
1990	1.36	(1.27, 1.45)	0.97	(0.86, 1.10)	1.17	(1.09, 1.26)
2000	1.39	(1.29, 1.50)	1.07	(0.98, 1.16)	1.15	(1.07, 1.23)
Black females						
1980	1.50	(1.23, 1.83)	1.14	(0.79, 1.64)	1.20	(1.03, 1.41)
1990	1.44	(1.28, 1.61)	1.70	(1.30, 2.21)	1.07	(0.99, 1.16)
2000	1.30	(1.18, 1.43)	0.96	(0.85, 1.08)	1.11	(1.01, 1.21)
Black males						
1980	1.63	(1.34, 1.97)	1.09	(0.79, 1.52)	1.19	(1.01, 1.42)
1990	1.52	(1.33, 1.74)	1.28	(0.83, 1.97)	1.16	(1.06, 1.26)
2000	1.41	(1.27, 1.57)	1.06	(0.89, 1.25)	1.07	(0.98, 1.16)
White age <60 y*						
1980	1.34	(1.22, 1.46)	0.93	(0.77, 1.11)	1.19	(1.06, 1.35)
1990	1.25	(1.14, 1.37)	0.98	(0.84, 1.13)	1.23	(1.05, 1.42)
2000	1.36	(1.23, 1.51)	1.07	(0.91, 1.26)	1.17	(0.98, 1.40)
White age ≥60 y*						
1980	1.48	(1.35, 1.62)	1.11	(0.98, 1.24)	1.32	(1.24, 1.42)
1990	1.30	(1.22, 1.39)	1.08	(0.98, 1.20)	1.19	(1.13, 1.27)
2000	1.35	(1.25, 1.46)	1.11	(1.03, 1.21)	1.21	(1.14, 1.28)
Black age <60 y*						
1980	1.68	(1.40, 2.02)	0.99	(0.60, 1.63)	1.28	(1.05, 1.55)
1990	1.53	(1.36, 1.73)	1.27	(0.86, 1.88)	1.04	(0.95, 1.14)
2000	1.28	(1.15, 1.43)	0.92	(0.80, 1.06)	1.03	(0.94, 1.12)
Black age ≥60 y*						
1980	1.52	(1.25, 1.85)	1.15	(0.88, 1.52)	1.17	(1.00, 1.36)
1990	1.45	(1.26, 1.68)	1.60	(1.21, 2.11)	1.13	(1.04, 1.23)
2000	1.39	(1.23, 1.56)	1.09	(0.92, 1.28)	1.12	(1.03, 1.21)

Parameter estimates are compared to the reference group of people who were neither born in the Stroke Belt nor resided in the Stroke Belt in adulthood. All models are adjusted for age and age squared. Confidence intervals (CI) adjusted for clustering on state of adult residence.
*Additionally adjusted for sex.

tional effects. The ecologic fallacy arises when one infers individual level relationships from ecologic relationships; however, in the current study the primary independent variables are ecologic exposures. This obviates the risk of ecologic fallacy because there are no conclusions regarding individual level variables. The concern regarding distinguishing contextual from compositional effects is key, and our analyses cannot address this. People born in SB states may be more vulnerable to stroke because of individual genetic or behavioral risk factors (compositional effects) or because of environmental toxins or toxic social dynamics (contextual effects). Our results cannot pinpoint a specific explanation, but they rule out explanations that would not affect early life (e.g., acute medical care). There are both contextual and compositional explanations that might operate from childhood forward, although we note that compositional explanations relating to race are not adequate. Selective migration also complicates interpretation of these results.[31] In the 2000 census, SB in-migrant black subjects had higher socioeconomic status in terms of education, income, and occupational characteristics than all other black subjects. Similarly, SB in-migrant white subjects were of higher socioeconomic status than other white subjects (details available from the authors, estimates based on reference[20]). The excess stroke risk among the in-migrants compared to the non-migrants may therefore be underestimated in our analyses. The effect estimates for the SB born (table 2, model 1) are the least vulnerable to bias arising from selective migration.

Our findings confirm prior evidence on the persistence of the US SB and extend these results by demonstrating that the timing of SB exposure may influence the risk conferred.[1,32] Although most prior SB work focuses on state of residence at stroke onset to define SB exposure,[33,34] several studies indicate that place of birth and childhood residence may influence stroke risk. The importance of place of birth has been shown in the United Kingdom[35] and, more directly related to the SB, in a study of South Carolina residents[4] and one national US study.[16] Our findings differ from these studies in suggesting that although either exposure at birth or in adulthood elevates risk of fatal stroke, the greatest risk is among individuals who resided in the SB at both time points. Our results also extend prior evidence demonstrating a geographic patterning of circulatory disease mortality among black subjects stratified by migration status[17] by focusing specifically on the SB states, showing temporal stability of the patterns through 2000, and presenting models both with and without stratification by place of death. The only other national analyses incorporating place of birth demonstrated the importance of nativity but were limited in that neither focused on the SB region.[17,18]

Prior research has considered whether racial disparities in stroke arise in part because black subjects live in higher risk areas than white subjects.[36] Our results suggest that the most important geographic characteristic is place of residence in childhood. In 1980 and 1990, the OR associated with adult SB residence for black subjects was greater than the OR associated with adult SB residence among white subjects, but this appeared to be largely because nearly all black adult SB residents were born in the SB, while a substantial fraction of white adult SB residents were born outside the SB. The ORs associated with SB birth is similar for black and white subjects.

These results suggest independent risk associated with childhood or adult SB residence. 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.[37] The finding that the harm of SB residence accumulates over life implicates health-promoting resources such as preventive medical care, social conditions, or environmental exposures. Acute medical treatments provided after stroke onset, which would more likely be predicted by place of residence at stroke onset, rather than place of birth or childhood residence, are unlikely explanations. Behavioral norms regarding diet, physical activity, and smoking are profoundly influenced by childhood social conditions,[38] and many of these behaviors appear to be largely determined before adulthood. Such risk factors may affect prevalence of silent infarcts,[39] increasing vulnerability to clinical strokes among Southerners much later in life.

These results do not support genetic explanations because the phenomenon appears to affect both black and white subjects. Given the large fraction of northern African Americans whose families immigrated from the South during the 20th century,[40] a genotype shared by southern black and white subjects but not northern black subjects seems unlikely.

Our understanding and ability to eliminate the US SB may be advanced by focusing on the timing of exposure. Future research should address whether there is truly a "dose" phenomenon, in which the harm accumulates with increasing exposure, or if the timing of exposure at specific critical periods is of greater importance. Our results suggest that conceptualizing SB exposure based on place of current residence is inadequate.

AUTHOR CONTRIBUTIONS

Statistical analysis was conducted by Anna Kosheleva.

DISCLOSURE

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CDC, AAN to Health Care Professionals: Monitor Patients for GBS

The Centers for Disease Control and Prevention (CDC) and the American Academy of Neurology (AAN) collaborated to reach out to neurologists across the US to monitor and report any possible new cases of Guillain-Barré syndrome (GBS) following 2009 H1N1 flu vaccination.

Neurologists and health care professionals nationwide who diagnose patients with vaccine-associated GBS should use the CDC and FDA Vaccine Adverse Event Reporting System (VAERS) to report their observations.

In addition, neurologists and all health practitioners in the 10 Emerging Infections Program (EIP) states—California, Connecticut, Maryland, Minnesota, New Mexico, New York, Colorado, Oregon, Georgia, and Tennessee—are asked to report all new cases of GBS, regardless of vaccination status, to their state's surveillance officer.

The AAN hosted a series of webinars providing an in-depth look at H1N1 vaccination and how it may pose a risk for GBS and information about the vaccination monitoring campaign.

For additional information about the monitoring campaign, or to watch the webinars or download VAERS form and information on reporting to surveillance officers in your state, visit the AAN's GBS toolkit page, *www.aan.com/view/gbstoolkit*.

Birth and adult residence in the Stroke Belt independently predict stroke mortality

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