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THE GREAT MIGRATION AND STROKE RISKS: DID LEAVING THE STROKE BELT PROTECT AFRICAN-AMERICAN MIGRANTS FROM STROKE?

BY

EMERY ALDEN MATHIESON
MASTER OF PUBLIC HEALTH

HUBERT DEPARTMENT OF GLOBAL HEALTH

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SOLVEIG A. CUNNINGHAM, PHD MSC
COMMITTEE CHAIR

The Great Migration and Stroke Risks: Did Leaving the Stroke Belt Protect African-American Migrants From Stroke?

By

Emery Alden Mathieson

Bachelor of Arts
Northwestern University
2011

Thesis Committee Chair: Solveig A. Cunningham, PhD MSc

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ABSTRACT

THE GREAT MIGRATION AND STROKE RISKS: DID LEAVING THE STROKE BELT PROTECT AFRICAN-AMERICAN MIGRANTS FROM STROKE?
BY EMERY MATHIESON

OBJECTIVE

The Great Migration, the massive exodus of African-Americans out of the American South to urban centers in the north and west that following the American Civil War until the 1970s, offers an opportunity to test whether leaving the South protected African-Americans from stroke compared with those who stayed behind. This study aims to contribute to knowledge about associations between location and stroke health amongst African-Americans.

METHODS

The association of migration out of the Stroke Belt (SB) region with history of stroke was studied in a sample of 6,983 African-Americans born in the SB region. Data were from the REGARDS Stroke study. Stroke history was judged by self-report, past stroke symptoms, or both. Location variables were created measuring exposure to SB: stayed in SB or left age ≤ 30, portion of life in SB, and age one left the SB. Descriptive statistics and T-tests measured changed across both location and stroke outcome strata. Regression models were used to evaluate associations between one of three location variables (reported above) and history of stroke. Models were adjusted for motivations for migration seen in the Great Migration. Controlling for these factors evaluated if migration itself or rather the motivators and outcomes of migration have an association with history of stroke.

RESULTS

For each bivariate model, time spent in the SB had a statistically significant association with stroke history [Set A OR 1.18 (1.06, 1.32), Set B OR 1.26 (1.08, 1.48), Set C OR (Age 18-30 level) 0.75 (0.65, 0.86)]. Adjusting for variables mirroring the economic and educational motivators of migration seen in the Great Migration, each location variable representing exposure to the SB region grew statistically insignificant.

CONCLUSIONS

The association between migration out of the SB and changes in indications of stroke come not from the migration itself; rather the opportunities afforded only to an African-American living outside the SB modify stroke risk. It is predicted that racism and discrimination in the SB region limit the opportunities that African-Americans can achieve, forcing many to migrate elsewhere to be afforded such opportunities and to experience the health benefits associated.

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INTRODUCTION

Background

Stroke incidence is a global issue. Currently, stroke is the third most common cause of death worldwide (Markus, 2004, p. 57). The World Health Organization estimated that, in 2008, 15 million people worldwide suffered a stroke, equating to a person every five seconds. The WHO went on to say that a third of the 15 million persons who suffer a stroke each year will go on to die from his or her stroke (Grysiewicz, Thomas, & Pandey, 2008, p. 871).

Many factors contribute to stroke risk. These factors are commonly categorized as modifiable, controllable through medicine and behavior change, and non-modifiable, innate characteristics that one is unable to change such as age, gender, race, and genetics. Despite extensive research into the causes of stroke and how these factors modify stroke risk, there still exists a proverbial "missing piece." Known factors only contribute to a portion of society's understanding of stroke risk leading researchers to explore other factors that may contribute to this unexplained portion of stroke risk.

Beginning in the early 1940s, researchers began to notice the emergence of geographic differences in stroke mortality within the United States. The term ''Stroke Belt'' became an informal description of the band of southern states where elevated stroke risk and mortality were observed (Grysiewicz et al., 2008, p. 882). By 1965, further research had solidified the concept of the "Stroke Belt," identifying it as a region located in the southeastern United States with approximately 50% higher stroke mortality rates than those living elsewhere in the United States. The Stroke Belt frequently includes eight southern states: North Carolina, South Carolina, Georgia, Tennessee,

Mississippi, Alabama, Louisiana, and Arkansas. The Stroke Belt Buckle, a region within the Stroke Belt of yet higher stroke incidence, includes Georgia, North Carolina, and South Carolina (Howard, Labarthe, Hu, Yoon, & Howard, 2007, p. 1). Beyond simply the higher stroke risk overall, we see that the impact of living in the Stroke Belt is more strongly felt in some populations than it is in others. Individuals living in the Stroke Belt find themselves at higher stroke risk, no matter their race. However, African-Americans, who already face far greater stroke risk, find themselves faced with yet an additional stroke risk burden when living in the Stroke Belt, far more so than white southerners. Howard et al. estimates that this additional stroke burden on southern African-Americans accounts for an annual increase in public health burden of $520 million (when compared to the expected higher stroke rate for African Americans living in the non-south) (Howard et al., 2007, p. 5).

Objective And Research Question

This study draws upon the historical "Great Migration," a massive migration of African-Americans out of the American south following the end of the American Civil War though the late 1970s, as a natural experiment. The study uses a sample population of African-Americans born in and migrated out of the Stroke Belt during the Great Migration era from the REGARDS study described later. The study assesses whether African-Americans who left the Stroke Belt during this Great Migration period fared better than those who stayed in regards to history of stroke. Using motivations for migration from Great Migration as shown in studies of the migration, the association between migration and stroke outcome is tested when controlling for the motivations and outcomes of migration. In essence, is the act of moving out of the Stroke Belt protective against stroke or rather is it changes to one's life brought about by migration that truly

modifies stroke risk? Additionally, how does this association change when behavioral and health indicators enter the model? While the exact motivations for each study participant may not be known, using literature on African-American migration during the Great Migration, this study was able to hypothesize about motivations and impacts of migration of study participants.

Significance

Clinically, the causes of stroke are well understood. The links between clinical indicators such as hypertension, diabetes, diet, and smoking and stroke outcome are all very well understood. New discoveries in genetics hope to paint even more of the stroke risk picture, with some scientists estimating that genetics could be responsible for upwards of 40% of stroke risk (Baird, 2010, p. 248). However, the identification of clinical causes of stroke fails to identify the factors earlier in life that eventually compound and lead to negative health outcome including, but not limited to, stroke. While many studies continue to evaluate clinical risk factors for stroke, these medical conditions are often just as much an outcome of socio-economic and behavioral factors as the stroke itself. Further, these clinical characteristics represent very late stages in the progress towards a stroke outcome, often only emerging later in life when little can be done to arrest them or their impact on stroke risk. No doubt, to fully understand the dynamics of stroke risk, one must understand those clinical characteristics that serve as harbingers for stroke. One must also realize, though, that these clinical characteristics explain only part of the equation. In identifying whether migration itself or the results of said migration modify stroke risk, this study hopes to identify opportunity for health intervention years in advance of the first stroke symptoms.

LITERATURE REVIEW

Clinical Information on Strokes

Pathology and Pathogenesis of Strokes

Clinically, a stroke is characterized as the rapid onset of neurological symptoms, which last over 24 hours or leads to death and has a believed vascular cause (Markus, 2004, p. 57). While a stroke can be the result of several different diseases, all strokes share a common outcome: the sudden-onset of a disruption of the blood supply to a particular part of the brain (Markus, 2004, p. 57). Grysiewicz et al. notes "strokes can either be ischemic (an occlusion of a blood vessel) or hemorrhagic (a rupture of a blood vessel)" (2008, p. 872). Approximately 80% of strokes are ischemic, while the other 20% are considered hemorrhagic (Markus, 2004, p. 57).

Ischemic strokes occur when a blockage occurs in a blood vessel, cutting off blood supply to an area of the brain. Within ischemic strokes, there are three subtypes which demonstrate the mechanism by which the ischemic stroke occurred: thrombotic, embolic, or lacunar. First, thrombus ischemic strokes occur when plaque has built up over time causing blood platelets to adhere to each other until the blood vessel is blocked, at which time the ischemic event occurs. Embolic strokes occur when a clot of blood cells forms in another location within the body and travels to a blood vessel in the brain, where it then blocks blood flow. Lacunar ischemic strokes occur when blood small penetrating arteries in the brain coagulates due to a condition such as hypertension (Bergman, Kindler, & Pfau, 2012, p. 39).

A less common condition known as transient ischemic attack (TIA) is identical to that of an ischemic stroke, except that the symptoms last less than 24 hours (often lasting minutes or hours) (Markus, 2004, p. 57).

Hemorrhagic strokes, while less common, have a mortality rate about three to five times higher than that of ischemic strokes. In Hemorrhagic strokes, a blood vessel bursts, draining blood into a cavity in the brain. Not only is an area of the brain no longer supplied with blood but also the pooling blood puts pressure on the brain possibly causing further brain damage. Hemorrhagic strokes are generally a result of long-term uncontrolled hypertension, aneurysms, or abnormal connection of veins and arteries (Bergman et al., 2012, p. 40). Like ischemic strokes, hemorrhagic strokes have multiple subtypes: intra-cerebral and subarachnoid. Intracerebral hemorrhagic strokes occur when bleeding occurs within the brain, while subarachnoid hemorrhagic strokes occur when bleeding occurs between the inner and outer layers of tissue that covers the brain, within the subarachnoid space (Grysiewicz et al., 2008, p. 872).

Following a stroke, roughly ten percent of persons will die within 30 days. Half of these deaths are as a result of immobility caused by the stroke, while a quarter of patients perish due to the direct neurological impact of the stroke (Pendlebury & Rothwell, 2004, p. 68). Amongst those patients who survive the 30-day window, half will remain disabled six months after the stroke incident and one-third will still be functionally dependent at one year. Most patients exhibit some degree of recovery after the stroke incident. Improvements generally occur rapidly over the days and weeks following a stroke, but can continue more slowly for a couple years (Pendlebury & Rothwell, 2004, p. 68).

While less common than a first stroke, recurrent strokes are still a major cause for concern. Within a week of a stroke or TIA, there is an 8-12% risk of stroke reoccurrence.

Extending the timeframe to a month, the risk of stroke reoccurrence jumps to nearly 20% (Pendlebury & Rothwell, 2004, p. 68).

Health Impacts of Stroke

Globally, stroke has a profound impact on mortality and morbidity. Currently, Stroke is the third most common cause of death (Markus, 2004, p. 57). In 2008, 15 million people worldwide suffered a stroke, a person every five seconds according to WHO estimates (Grysiewicz et al., 2008, p. 871). A third of this group go on to die from stroke (Grysiewicz et al., 2008, p. 871). The impacts of stroke often extend far beyond the immediate medical toll, as another third (five million persons) are left permanently disabled, placing an immense burden on families and communities around the world (Grysiewicz et al., 2008, p. 871). DALYs, a sum of life-years lost due to premature death and years lived with disability (adjusted based on severity), is an indicator of societal impact of medical conditions in terms of both mortality and morbidity. By 2030, it is estimated that stroke-related disability will be the fourth most important cause of disability in western societies in terms of years of life affected (Grysiewicz et al., 2008, p. 871).

In the United States, stroke is the third leading cause of death, killing more persons each year than heart disease and cancer. An estimated 780,000 new or recurrent strokes occurred in the United States in 2008: equaling 2160 strokes per day or one stroke every 40 seconds. (Grysiewicz et al., 2008, p. 871). Four years later in 2012, this number has grown to approximately 795,000 persons per year (Bergman et al., 2012, p. 36). While many of these are first-time strokes, approximately 185,000 of them are recurrent strokes (Bergman et al., 2012, p. 36). Of those who suffer a stroke each year in the United States, approximately 150,000 will die as a result. Nearly 1 out of every 16

Americans will die as a result of a stroke (Grysiewicz et al., 2008, p. 871). Beyond stroke mortality, "stroke is the primary cause of profound long-term disability in the United States with an estimated 5.8 million stroke survivors in 2008" (Grysiewicz et al., 2008, p. 871).

The burden on society of stroke mortality and morbidity is immense. In the United States, "the total cost of stroke has been estimated at $65.5 billion in 2008" (Grysiewicz et al., 2008, p. 871). In 2009, another study estimated the total cost of strokes in the United States was $68.9 billion (Bergman et al., 2012, p. 36). On an individual scale, the Stroke PORT study estimated the lifetime cost (both direct and indirect) of having a stroke as $104,000, far beyond the means of many Americans (Howard et al., 2007, p. 5).

Treatments for Strokes

When a person develops symptoms of a stroke, the race is on to accurately categorize and treat the stroke. The adage "time is brain" refers to the time sensitive nature of acute stroke care, "as delays in diagnosis and treatment may render the patient neurologically impaired and disabled" (Gorelick, Gorelick, & Sloan, 2008, p. 924). "The importance of the accurate classification of stroke type and subtype needs to be appreciated, because ischemic and hemorrhagic strokes differ in their causes, pathophysiology, treatments, and outcomes" (Baird, 2010, p. 246). Thus, a health care worker's "ability to recognize signs and symptoms of a stroke is a crucial factor in preparing patients for stroke intervention" (Bergman et al., 2012, p. 36). Implementing the "Stroke Chain of Survival," as described by Gorelick et al., ensures health care workers are able to rapidly assess and treat acute stroke victims, whether his or her stroke is ischemic or hemorrhagic in nature (2008, p. 924). The "Stroke Chain of Survival" uses

the acronym the "seven Ds" to guide acute stroke care: "1. Detection of onset of stroke signs and symptoms; 2. Dispatch through activation of the EMS and prompt response; 3. Delivery of the patient to a pre-notified hospital, with appropriate pre-hospital care; 4. Door (ED triage); 5. Data compilation including such ED evaluation as head CT scan; 6. Decision regarding potential therapies; 7. Drug therapy" (Gorelick et al., 2008, p. 925). The following paragraphs describe this process in further detail.

As noted above, the rapid detection of the signs and symptoms of stroke plays a vital role in the eventual acute stroke treatment. The Cincinnati Pre-hospital Stroke scale is a popular test used to determine if a patient is likely experiencing a stroke. Using the test, patients are evaluated on three conditions: "(1) facial asymmetry; (2) speech disturbance; and (3) upper extremity weakness", all of which a positive presence could indicate a stroke (Gorelick et al., 2008, p. 925). Medical professionals are generally taught this methodology using the acronym FAST referring to "facial droop, arm drift, and speech (dysarthria and aphasia) with T for onset time" (Gorelick et al., 2008, p. 925). Onset time, or "last time well" as it is sometimes known, plays an essential role in treatment decisions later on in the stroke care process and will be discussed further in following sections. It should be noted that the Cincinnati Pre-hospital Stroke Scale is by no means the only evaluation methodology used to evaluate potential strokes; however, it is commonly used over other methods such as the LAPSS test given its simplicity in administration (Gorelick et al., 2008, p. 925).

Once a patient is believed to be suffering a stroke and requisite history and initial physical examinations have been completed, the stroke patient should immediately be sent for a neuroimaging study (Gorelick et al., 2008, p. 928). Clinically, it is impossible

to distinguish between an intracerebral ischemic stroke and an intracerebral hemorrhagic stroke from a physical evaluation alone; however, intracerebral ischemic stroke is far more common regardless of location or race, occurring in about 60% of stroke cases. "Because management of cerebral [hemorrhagic stroke] differs markedly from that of [ischemic stroke], urgent brain imaging is required in all stroke patients" (Markus, 2004, p. 61). Given the decision to conduct brain imaging, health care workers have two options for imaging: computed tomography (CT scan) or magnetic resonance imaging (MRI) (Bergman et al., 2012, p. 4). CT scans are most widely used in the diagnosis of acute stroke. CT scans can be obtained within minutes, making it feasible for critically ill patients needing immediate care. Assuming imaging is conducted within three hours of the onset of stroke symptoms, CT scans can distinguish between true strokes and other conditions which may mimic stroke symptoms such as a tumor or infection (Gorelick et al., 2008, p. 928). Brain MRI, on the other hand, is an increasingly used imaging technology. Previously, MRI scans failed to show any significant advantage over traditional CT scans, especially in the detection of hemorrhagic strokes. However, with advances in MRI technology, brain MRI has grown more accurate than CT scans "for detection of acute ischemia; it can now easily detect acute and chronic hemorrhage. (Gorelick et al., 2008, p. 932). Imaging is also needed to identify the specific sub-type of ischemic and hemorrhagic strokes, as this information also factors into eventual treatment (Bejot et al., 2007, p. 85).

Using a combination of patient history, symptoms displayed, and information gathered from brain imaging, health care workers are tasked to evaluate a host of different treatment options. With advances in acute stroke research, the number of

different treatment options at the disposal of health care workers has grown further. Often a combination of two or more of these options is used to ensure the best patient outcomes possible. Guidelines for the use of these treatments is continually changing and being further refined; however, in general, the following treatments represent the basis of much of the acute stroke treatment conducted today.

A number of medical drug options exist for treating acute stroke. Starting in 2005 following FDA approval, health care providers have been using the intravenous (IV) drug rt-PA. The NINDS rt-PA Stroke Study, which played a vital role in the eventual FDA approval, demonstrated that when used within the first three hours of a stroke event, rt-PA lead to an improvement in clinical outcomes at three months when compared with a placebo (Gorelick et al., 2008, pp. 934–5). According to Gorelick et al., "An estimated 1.8%–2.1% of acute ischemic stroke patients receive intravenous (IV) rt-PA" (2008, p. 923). This low frequency may be due in part to the lack of well-developed local care systems for rt-PA treatment, limiting treatment to mostly larger trauma hospitals. Also, FDA approval extends only to the first three hours of a stroke event, further limiting use to patients who are able to receive immediate care when stroke symptoms present themselves (Gorelick et al., 2008, pp. 923–4). Further, treatment with rt-PA is not without possible negative consequences. The same study that aided in FDA approval of rt-PA found "for every 18 stroke patients treated with IV rt-PA, two more will achieve as good an outcome as they would have without IV rt-PA and one will have significant intracerebral hemorrhage related to treatment (Gorelick et al., 2008, pp. 934–5).

Other drug therapies rely on the use of antiplatelet and anticoagulant drugs. Antiplatelet drugs prevent blood cells from further sticking together and break up existing

clusters of blood cells. Pendlebury & Rothwell note "aspirin started within 48 hours of acute [ischemic] stroke onset reduces mortality and recurrent stroke, so early brain imaging to exclude [hemorrhagic stroke] is important" (2004, p. 65). Excluding hemorrhagic stroke diagnosis is vital, as antiplatelet drugs would actually impair the body's ability stop hemorrhaging blood, worsening the condition. Oral anticoagulant drugs prevent blood from coagulating which could create new embolus, clots of blood cells that could get stuck in a blood vessel in the brain, causing yet further strokes. Use of anticoagulant drugs is especially relevant in patients with atrial fibrillation, a heart condition that increases the chance of embolus forming within the body. Anticoagulant drugs are generally given later in stroke treatment after a course of antiplatelet drugs have been given to the patient, sometimes occurring a few weeks after a stroke event (Pendlebury & Rothwell, 2004, p. 65).

Beyond drug therapies, there exist a few mechanical options for stroke treatment. A clot retrieval tool or device, such as the MERCI endovascular embolectomy device, travels through a patient's blood vessel until it arrives at the location of the clot. Doctors are then able to physically pull the clot out of the blood vessel, alleviating the ongoing stroke. The FDA approved the MERCI device for use in 2005 for patients in the first eight hours of a stroke event. The Penumbral System, another clot retrieval device, was approved by the FDA in 2007 for use in clinical practice (Gorelick et al., 2008, p. 937).

Conceptual Links to Stroke Risk

In assessing the impact of location and migration exposure, this study draws upon existing research capturing the motivations, behavioral changes, and health outcomes associated with migration. Described below are a host of different factors that fit into these categories, referencing existing literature on known stroke risk modifiers.

Bio-Demographic Factors

ETHNICITY/ RACE

Ethnicity and race both play a significant role in the modification of stroke risk. Using data from a 2005 Centers for Disease Control and Prevention surveillance study, the prevalence of stroke overall was "6.0% among American Indian/Alaska Natives, 4.0% among blacks, 2.6% among Hispanics, 2.3% among whites, and 1.6% among Asians" (Grysiewicz et al., 2008, p. 873). Focusing specifically on first-time ischemic strokes, the Northern Manhattan Stroke Study (NOMASS) found the incidence of age adjusted ischemic strokes per 100,000 people (age > 20) to be "88 in whites, 191 in blacks, and 149 in Hispanics" (Grysiewicz et al., 2008, p. 874). Limiting ages to ages 45 to 84 saw the incidence per 100,000 persons jump to 360 in white males, 230 in white females, 660 in black males, and 490 in black females (Grysiewicz et al., 2008, p. 874). Increases in stroke risk for African-Americans are believed to be associated with the greater prevalence and severity of hypertension amongst African-Americans (Markus, 2004, p. 58). Not only does ethnicity and race modify one's stroke risk, but they also modify one's risk of stroke mortality. In 2004, a study found that the death rate due to stroke per 100,000 persons was 48.1 for white males, 74.9 for black males, 47.2 for white females, and 65.5 for black females (Grysiewicz et al., 2008, p. 874).

African-Americans, who already face a greater stroke mortality risk in general, saw an even greater mortality risk living in the South when compared to non-southern counterparts. Howard et al. found "across age and sex strata, the black-to-white stroke mortality ratio was consistently higher for southern states, with an average black-to-white stroke mortality ratio that ranged from 6% to 21% higher among southern states than in non-southern states" (2007, p. 1). Thus, African-Americans living in the American South

are at a higher risk of death from stroke not only because they are African-American but also because they live in the south (Howard et al., 2007, p. 6). The profound change in stroke risk amongst African-Americans compared to other races made African-Americans a clear choice for inclusion in the study. Working with a population that represents the lion's share of stroke mortality enables analyses to be as wide reaching as possible.

GENDER

Chief amongst non-modifiable characteristics, gender plays a large role in one's stroke risk. Males are more likely to suffer a stroke, but, overall, more females suffer strokes each year than do males. This seemingly contradictory statement notes the impact of age on stroke risk in addition to gender. At each age, males are more likely to suffer a stroke than are women; however, females have longer longevity than men meaning they are more susceptible to increased stroke risk due to age (Markus, 2004, p. 57). In 2004, women accounted for 61% of stroke deaths, again due to longer longevity than men (Grysiewicz et al., 2008, p. 875).

AGE

As mentioned above, age plays a significant role in one's stroke risk. Data indicate that each consecutive decade after age 55, one's stroke risk approximately doubles (Grysiewicz et al., 2008, p. 874). Age, as expected, is the strongest risk factor for both ischemic stroke and hemorrhagic stroke (Markus, 2004, p. 57). Markus notes "the risk of stroke in 75–84- year-olds is 25 times that in 45–54-year-olds" (2004, p. 57). The prevalence of atherosclerosis, a disease that causes fatty deposits to form in blood vessels potentially creating blockages, increases with age. Thus, as one's age increases

they are more likely to develop atherosclerosis, which in turn is a risk factor itself for ischemic and hemorrhagic stroke.

Birth Weight and Fetal Nutrition

Birth weight, an indicator of fetal nutrition, has an unexpectedly strong link to stroke and a variety of other diseases. The concept, known as Fetal Origins of Adult Diseases (FOAD), is based on "the premise of "developmental plasticity"—a single genotype, influenced by specific intrauterine events, has the capability to produce different phenotypes" (Barker, Eriksson, Forsén, & Osmond, 2002, p. 158). Essentially, during developmental periods, the fetus is malleable and able to adapt to be best suited to the environment to which it believes it will be born into. For example, faced with the adversity of malnutrition in Utero, a fetus will alter the structure and function of various organs so as to preserve brain development and promote survival. These adaptations then allow the child to thrive in the extra-uterine environment where a similar level of malnutrition is present. The changes that occur during one's time in Utero allow him or her to survive in whatever the eventual, predicted environment may be. Some evidence suggests that this programming occurs both in the Utero environment as well as into childhood (Barker et al., 2002, p. 158).

Socio-Economic Factors

The negative health implications of this plasticity result when there exists a disconnect between the predicted and the actual. Researchers found that persons born during a long-term famine in the Soviet Union adapted to the lack to nutrition and were able to thrive without the expected diseases that would come about as a result of chronic malnutrition. On the other hand, persons born during a famine in the Netherlands from 1944 to 1945 experienced far worse health outcomes. The Dutch famine was fairly short-

lived resolving by the year 1945. Persons in Utero during the famine adapted to the lack of nutrition, expecting to find a similar environment outside to womb (Barker et al., 2002). No longer faced with a lack of nutrition, Dutch infants at the time "exhibited 'catch-up' or compensatory growth during infancy. Their intrauterine environment, which supported short-term survival, was ill matched with their subsequent extra uterine environment. Thus, the Dutch fetuses paid the price for their intrauterine adaptation" (Barker et al., 2002, p. 160).

Today, a number of studies both on human and animal subjects have demonstrated the association between low birth weight followed by exponential growth and an increased risk for a host of diseases such as obesity, insulin resistance, dyslipidemia, and hypertension. These conditions, in turn, modify stroke risk directly and indirectly. While there is some evidence that developmental plasticity can be reprogrammed over time, the effect of developmental nutrition still remains profound. Further, attempting to reprogram adaptations created in Utero is akin to placing a Band-Aid on a cut; only in addressing the underlying issue rather than treating it can health outcomes be improved (Barker et al., 2002, p. 161).

While birth weight and fetal nutrition, themselves, were unknown for the study participants, location analyses later attempted to capture the impact of early life environments and evaluate its association with stroke incidence later in life.

INCOME, EDUCATION, AND OPPORTUNITY

Income and Education, and the opportunities that arise thanks to income and education, play a substantial role in health. It should be noted that income and education form a cyclical relationship whereby income improves access to education, which in turn improves one's income. Due to this multiplicative effect, the disparity between someone

who has economic and educational opportunities, and one who does not, is vast. This discrepancy transfers into large differences in health outcomes. Principally, economic and educational success improves health by increasing health knowledge and understanding as well as increasing access to healthcare. Addo et al. found that persons in lower socio-economic groups demonstrated a 30% higher incidence of stroke compared to those in other socio-economic groups (Addo et al., 2012, p. 1189). As health issues compound over time, the earlier in one's life that they are able to achieve better socio-economic status and thereby receive better healthcare, the better prognosis they will have in regards to stroke incidence and general health (Addo et al., 2012).

Behavioral Factors

ALCOHOL CONSUMPTION

Alcohol consumption, interestingly, has both an aggravating and mitigating impact on stroke risk. Evidence suggests that moderate alcohol consumption may protect against both ischemic heart disease and stroke; however, heavy alcohol consumption and/or abuse is a risk factor of stroke. More so, heavy drinkers are at a higher risk of suffering a hemorrhagic stroke. While the exactly mechanism behind this higher risk is unknown, it is believed that heavy drinking increases blood pressure and predisposition to atrial fibrillation. (Markus, 2004, p. 58)

PHYSICAL ACTIVITY

Cohort and case-control studies demonstrate that a lack of physical activity or exercise is associated with an higher risk of stroke (Markus, 2004, p. 58). A study of females aged 65 to 74 found that low physical activity led to a relative risk for stroke of 1.82. On the other spectrum, moderate to high levels of physical active were actually protective against stroke in the middle-aged men studied. These findings were later

extrapolated in the Northern Manhattan Stroke Study (NOMASS), where increased physical activity was found to be protective against stroke across race, sex, and age. Further, increases in level of intensity and duration of physical activity was met with further decreased stroke risk (Grysiewicz et al., 2008, p. 879).

CIGARETTE SMOKE

Cigarette Smoking, a behavior widely considered deleterious to health, has also been identified as an independent risk factor for stroke in a variety of different studies. Cigarette smoking has been prescribed a relative risk of 1.9 for ischemic stroke overall, with variation depending on one's age: RR = 2.9 for persons < 55 years old, RR = 1.8 for persons aged 55 to 74 years, RR = 1.1 for persons over 70 years. Even after smoking cessation, ex-smokers remain at higher risk for stroke (Grysiewicz et al., 2008, pp. 876–877).

Harm associated with cigarette smoking extends beyond the tobacco user himself or herself, also harming persons exposed to "second hand" cigarette smoke (Grysiewicz et al., 2008, pp. 876–877). Two case-control studies of interest evaluated the association between second hand smoke (SHS) and stroke. In You et al., the relative stroke risk for people exposed to a spouse smoking 1 to 20 cigarettes each day was 1.72. When the spouse smoked greater than 20 cigarettes a day, the relative risk jumped to 2.59. In another study, Bonita et al., those exposed to SHS at home or work for a year or longer had a relative risk of 1.82 for having a stroke in the decade following SHS exposure (Joubert, Cumming, & McLean, 2007, p. 74).

Health Factors

OBESITY

Obesity, as indicated by a body mass index (BMI) greater than 27 for females and 30 for men is a significant risk factor for ischemic stroke. For women, when controlling for other cardiovascular risk factors, a BMI of 27 to 28.9 resulted in a 1.75 times higher risk for ischemic stroke. A female with a BMI greater than 32 has a relative risk for ischemic stroke of 2.37. Males with a BMI over 30 saw a relative risk for ischemic stroke of 1.95. Each incremental BMI unit increase for a male resulted in a 6% higher relative risk for stroke. Additionally, weight gained after the age of 18 increases one's risk of ischemic stroke further. Abdominal adipose fat, fat located around the waist and hips, is an additional risk factor for men, indicating that not only weight gain but the location of said gain plays a role in stroke risk (Grysiewicz et al., 2008, p. 879).

Understanding Links Between Location and Stroke Risk

Stroke Belt

Beginning in the early 1940s, researchers began to notice the emergence of geographic differences in stroke mortality within the United States. The term ''Stroke Belt'' became an informal description of the band of southern states where elevated stroke risk and mortalities was observed (Grysiewicz et al., 2008, p. 882). By 1965, further research had solidified the concept of the "Stroke Belt," identifying it as a region located in the southeastern United States with approximately 50% higher stroke mortality rates. The Stroke Belt frequently includes eight southern states: North Carolina, South Carolina, Georgia, Tennessee, Mississippi, Alabama, Louisiana, and Arkansas. The Stroke Buckle, a subset of the Stroke Belt including Georgia, North Carolina, and South

Carolina, are marked by yet higher stroke morality risk in comparison to other Stroke Belt states (Howard et al., 2007, p. 1).

Given the startling information on the change in risk of stroke mortality faced by southern African-Americans, researchers have sought to explain how residency in the South contributes to increased stroke mortality for all races, but especially African-Americans. The following sections review existing, preliminary research on the possible causes of increased stroke mortality risk in certain locations as opposed to others.

Temperature and Stroke Risk

COLD WEATHER

Anecdotally, stroke occurrence increases during cold weather, especially in the winter. There are, however, few studies on the true impact of cold weather on stroke risk (Hong et al., 2003, p. 473). Of what data are available, the onset of ischemic stroke is associated with a drop in temperature, even when controlling for air pollution (Hong et al., 2003, p. 476). The impact of cold weather on stroke risk was greatest in the winter, suggesting that low temperatures as well as fluctuations in temperature are associated with ischemic strokes (Hong et al., 2003, pp. 476–7). Kyobutungi, Grau, Stieglbauer, and Becher confirm this hypothesis stating, "We found no consistent or relevant relationship between temperature change and stroke risk for different characterizations of this variable except for large day-to-day variations where the risk seemed to increase" (2005, p. 696).

Another important consideration is that a lag exists between temperature changes and modification of stroke risk. Hong et al. found the strongest association to be between temperature and the stroke risk the day after exposure to cold weather. According to the study, the odds of having a stroke were 2.9 times higher 24-48 hours from when a person

experiences an interquartile range decrease in temperature (Hong et al., 2003, p. 473). Hong et al. generalized these findings, stating "the peak risk is plausibly 6 hours to 5 days after exposure", with the risk decreasing at the 54 hour mark (Hong et al., 2003, p. 473).

Researchers believe that decreasing temperatures affect blood viscosity or coagulation. As temperatures grow colder, blood begins to thicken, increasing the chance of a blockage forming (Hong et al., 2003, p. 476). Yet, ambient temperature is a poor indicator of actual individual temperature and cold exposure. Persons are certainly more likely to stay indoors during extreme weather period. Further, clothing and other factors vary individual exposure. In conclusion, the role of temperature on stroke is inconclusive. Until true individual exposure to cold can be monitored, associations between temperature and stroke risk will remain weak (Hong et al., 2003, p. 477).

The group particularly at risk of the impact of cold weather is women older than 65 years who are not obese. Also previous hypertension or high cholesterol adds further to the impact of cold weather on ischemic stroke risk (Hong et al., 2003, p. 473). By comparison, obese persons are less affected by the decrease in temperature, due to an apparent insulating effect caused by the fat. Obesity itself, however, is indeed a risk factor for stroke despite reduced modification caused by cold weather (Hong et al., 2003, p. 477).

WARM WEATHER

The impact of warm weather, unlike that of cold weather, is highly disputed, and different studies offer contradictory results. Villeneuve et al. argue that a 5°C increase in average temperature over a 3 day period leads to a 10% reduction (OR=0.90, 95% CI: 0.82, 0.97) in the risk of ischemic stroke This association was only significant when

stratifying by season, and grew insignificant if evaluating the year as a whole (Villeneuve et al., 2012, p. 195).

Basu et al., contrarily, argues that both ischemic and hemorrhagic stroke risk increase during periods of greater ambient temperature in Scotland and Australia (Basu, Pearson, Malig, Broadwin, & Green, 2012, p. 818). Basu et al. only found this association significant in persons under the age of 65. In addition, the impact of higher temperatures on stroke risk was 7.2% larger for Hispanics as it was for Caucasians (Basu et al., 2012, p. 817)

Green et al did report an increase in ischemic stroke risk following an increase in apparent temperature, but the association did not carry over to ho hospital admissions for all cerebrovascular diseases overall (Basu et al., 2012, p. 818). Lim et al concludes saying that high temperature is harmful for ischemic stroke alone and therefor does not impact hemorrhagic stroke (Lim, Kim, & Hong, 2013, p. 152).

Temperature and Harvesting

Researchers believe that decreasing temperatures affect blood viscosity or coagulation. As temperatures grow colder, blood begins to thicken, increasing the chance of a blockage forming (Hong et al., 2003, p. 476). Yet, ambient temperature is a poor indicator of actual individual temperature and cold exposure. Persons are certainly more likely to stay indoors during extreme weather period. Further, clothing and other factors further vary individual exposure. In conclusion, the role of temperature on stroke is inconclusive. Until true individual exposure to cold can be monitored, associations between temperature and stroke risk will remain weak (Hong et al., 2003, p. 477).

Further yet, one must consider the chance that associations between stroke risk and temperature may exist due to harvesting. Harvesting in this instance refers to that the

increase in stroke mortality around cold or warm weather may only represent an earlier death of those about to die soon already. Cold or warm weather does not increase one's risk for stroke mortality, it simply causes the stroke event and mortality to occur sooner (Lim et al., 2013, p. 152).

Environment and Stroke Risk

Neighborhood greenness, the presence of trees, parks, and other natural amenities, has long been a selling point for property. Often considered an indication of a good living environment, neighborhood greenness, amongst other factors such as "good" schools, is often an important consideration of homebuyers. Up until recently, though, neighborhood greenness held only an intangible benefit for persons, certainly in regards to stroke health. A study by Pereira et al., however, found that the "odds of hospitalization for heart disease or stroke was 37% (95% CI: 8%, 57%) lower among adults in neighborhoods with highly variable greenness […] compared to those in predominantly green, or predominantly non-green neighborhoods" (2012, p. 1). Interestingly, the mix of green and non-green environments was found to have a larger impact on the reduction of stroke risk than predominantly green neighborhoods, suggesting that perhaps the combination of greenness within an urban environment provides the most benefit to stroke risk. This would imply, then, that living in an urban environment has some modification on stroke risk as well. Pereira et al. believed the link between greenness of environment and stroke risk could be attributed to higher levels of physical activity, which was made easier or even possible thanks to the mix of natural and built environments. Unfortunately, analyses were unable to evaluate if greenness was in fact associated with increases in exercise, leaving the question unanswered (Pereira et al., 2012, p. 5).

Pollution and Stroke Risk

While much of the focus of environmental protection today surrounds greenhouse gases and their impact on global warming, air pollutants still take a large toll on the health of the United States and the world overall. Over a period of seven years, a Korean study investigated the impact of a set of air pollutants, PM_{10}, NO_2, SO_2, CO, O_3, on stroke mortality risk. The study determined that exposure to these pollutants resulted in a slightly higher risk of stroke incidence: relative risk 1.05. On top of that, exposure increased the risk of death due to ischemic stroke about 3-6%, an association that did not carry over to hemorrhagic stroke (Joubert et al., 2007, p. 74). In another study, Hong et al. found exposure to PM_{10} and other gaseous pollutants to be a significant risk factors for ischemic stroke mortality, even after controlling for confounding factors such as "long-term trends, seasonal changes, day-of-the-week effects, and meteorologic influences" (Hong et al., 2002, p. 187). Researchers Maheswaran and Elliott found that living within 200 meters of a main road (an indication of proximity to air pollutants) resulted in a 5% increase in stroke mortality when compared to people living over one kilometer away (Joubert et al., 2007, p. 74). Then, in 2012, Villeneuve et al. published findings demonstrating that short-term elevations in NO_2 and $PM_{2.5}$ concentrations during summer months led to an increased risk of ischemic stroke amongst Edmonton residents during the years 2003 to 2009 (Villeneuve et al., 2012, p. 197).

While the exact mechanism by which air pollution impacts stroke risk is unknown, "it has been hypothesized that alveolar inflammation, induced by exposure to particulate air pollution, causes acute cardiovascular events in susceptible individuals" (Hong et al., 2002, p. 190). Likewise, it is hypothesized that air pollution causes inflammation of blood vessels restricting blood flow and potentially creating ideal stroke

conditions. This, however, does not explain why the impact of air pollution is only seen on ischemic stroke and not hemorrhagic stroke as well. Further studies are needed to explain this lack of association (Hong et al., 2002).

Interestingly, researchers have observed a delay between pollutant exposure and the impact on stroke risk. Further still, the delay varies depending on the specific pollutant. Using single-pollutant analyses and interaction models, Hong et al. found that PM_{10} exposure had the largest impact on stroke mortality the same day as exposure. However, for NO_2, SO_2, and CO, the greatest impact on stroke mortality came two days after exposure (Hong et al., 2002, p. 189). In 2007, Joubert et al. found lag times of up to three days before significant impact on ischemic stroke risk was observed (Joubert et al., 2007, p. 74).

Like studies evaluating the association between temperature change and stroke risk, air pollution studies are limited by the fact that true individual exposure remains unknown. Research relies on ambient conditions and measurements to determine study variables, and, therefore, is unable to take into account an individual's exposure (Hong et al., 2002, p. 190).

Migration and the Impact on Health

Dynamics of Migration

Judging migration as simply a change in physical location is a gross underestimation of the impact migration has not only on the person migrating but also the populations affected. It also fails to identify the motivations that lead a person to migrate, be them economic betterment, political upheaval, education or other purposes. Bhugra describes migration as a "process of social change" when a person or group of persons leaves one place for another, be it temporarily or permanently (Bhugra, 2004, p.

129). "It must be emphasized that migration is not only a trans-national process but can also be rural–urban" (Bhugra, 2004, p. 129). It is important to understand migration as a process, as its motivations and impacts are rarely felt only over a short period of time. Rather, a migration can influence a person for years or even the rest of his or her life.

Before considering the motivations for migration, one would be unwise to overlook the social toll of the migration itself. According to Bhugra, migrants experience "at first a sense of loss, dislocation, alienation and isolation, which will lead to processes of acculturation" (Bhugra, 2004, p. 129). This initial "culture shock" places the migrant at odds with society. The migrant, in a new location, away from a social support structure, faces great levels of stress following migration. The ability to deal with this stress and establish oneself in the new location is vital in minimizing the psychosocial impact of migration. Failure to do so undoubtedly result in feelings of isolation and alienation (Bhugra, 2004, p. 129). "In a classic study, Ödegaard reported that the rates of schizophrenia among Norwegians who had migrated to the USA were higher when compared with Norwegians who had stayed back in Norway," noting that inability to assimilate in one's new culture places great stress on one's psyche and can result in mental illness if not addressed (Bhugra, 2004, p. 130).

To overcome the negative health impacts of the migration event, the opportunities afforded by migration must be, or at least be believed to be, equal to or greater than the known costs. While some motivations to migrate are personal, there are a number of fairly universal motivators for migration. According to Bhugra, "rural–urban migration is more likely to be for economic or educational reasons, whereas migration across nations may be for social, educational, economic or political reasons" (Bhugra, 2004, p.

129). In the case of disadvantaged populations, we see opportunities to escape poverty, deprivation, and inequality as main drivers of migration (Bhugra, 2004, p. 129).

One must also consider the discrepancy between those who may wish to migrate and those who actually migrate. This process, described as selection, limits the population of migrations to those who are best in a position to migrate. While hire wages could doubtlessly motivate someone to migrate out of a lower wage area, only those persons who could afford to migrate would be able to take advantage of this opportunity. If one is unable to afford the economic or social costs of migration, those initial costs discussed earlier, they will be unable to migrate, no matter the potential pay off resulting from migration. As such, one sees that persons migrating for higher wages are likely already the most productive individuals, likely possessing the most education as well. So while migration can present great opportunities for all of a population, only those who are comparatively better off are able take advantage of said opportunities. In doing so, it may open local opportunities for a new set of persons to become the better off persons, or migration could leave a vacuum of knowledge and skills in a population actually leaving it worse off than before (Black, Sanders, Taylor, & Taylor, 2011, p. 21). This concept is commonly described as brain drain, in which the smartest members of society leave to pursue economic opportunities elsewhere leaving the population uneducated and without potential for improvement.

The Great Migration

HISTORY AND POPULATION OF INTEREST

Following the end of the American Civil War in 1865, millions of African-American suddenly found themselves free from the shackles of slavery. No longer forced to stay in the South, African-Americans would travel across the United States in what

historians would describe as a three-pronged Great Migration, one that would span nearly a century. The first wave of migration took place between 1865 and 1896. This initial migration followed shortly after the end of the civil war and continued through the end of the United States' Reconstruction Era. Entering the twentieth century, the African-American migration turned from a trickle to a flood "as nearly two million African Americans abandoned hope for a better life in the south and headed for points north, west, and overseas" (Mathieu, 2009, p. 20). In the period 1940 to 1970, the Great Migration continued as many more African-Americans left the American South in search of opportunity. In total, over six million African-Americans "shifted the weight of their numbers, culture, and politics from the ostensibly rural south to various urban northern and western regions" (Mathieu, 2009, p. 20).

Population changes based on census data are shown in Table 1 below. Data are stratified by state within the Stroke Belt–non-buckle (Tennessee, Mississippi, Alabama, Louisiana, and Arkansas), Stroke Belt–buckle (Georgia, North Carolina, and South Carolina), and Non-Stroke Belt region. Population totals, in thousands, are displayed for the year 1920. Data for each decade that followed are displayed as a change from the previous decade's population.

Historical Discrimination and Lack of Opportunity

After the end of the American Civil War, African-Americans still found themselves in an unfriendly, harsh environment in the American South. As newly freed African-Americans began to search for work, they found themselves faced with discrimination and organized disenfranchisement. "The rural population was disproportionately mired in tenancy status within a plantation system of agricultural production" (Tolnay, 1997, p. 1214). Debt peonage, a form of re-enslavement based on

debt obligation, was commonplace on these farms, placing African-Americans in a position quite similar to pre-emancipation years (Mathieu, 2009, pp. 19–20). Tolnay notes that in urban centers, "black men toiled primarily as unskilled laborers, while black women worked as domestic servants. To be sure, some of the more successful were able to achieve a reasonably comfortable existence by becoming preachers or teachers. However, they represented a relatively small percentage of the overall southern black population (1997, p. 1214). Even as years passed and some African-Americans were able to acquire educations, deep and persistent social obstacles remained commonplace for southern African-Americans (Tolnay, 1997, p. 1214). As debt peonage and other forms of institutional disenfranchisement became less commonplace, African-Americans saw the rise of Jim Crow era laws as a new threat to their livelihood and happiness. With the emergence of this thinly veiled institutional racism, African-Americans grew ever more eager to find opportunity somewhere more accepting (Mathieu, 2009, pp. 19–20)

Reasons for Migration out of the South

African-Americans migration during what is now considered the Great Migration was driven by a number of factors, which themselves evolved over the century long migration. Directly following the American Civil War, many African-Americans took to the road to discover the nation that was now open to them. Forbidden from travelling while enslaved, freedom opened the door for travel and adventure, giving African-Americans what Mathieu described as a "wanderlust strictly forbidden during slavery" (Mathieu, 2009, p. 19). While some traveled for enjoyment and intrigue, many more did so with a purpose of reuniting families torn apart by centuries of slavery or even the civil war itself. Historical accounts describe African-Americans "chasing rumors of their [family's] whereabouts as far as Canada and the Caribbean" (Mathieu, 2009, p. 19).

While some of the migration was borne out of desire, many times the migration was borne out of necessity. The historical and continuing discrimination of African-Americans in the south had two roles in driving migration. First, discrimination and disenfranchisement restricted African-Americans from accesses many jobs beyond subsistence farming. In order to find higher paying, safer, and available jobs, African-Americans were forced to migrate north. Second, discrimination, while barring African-Americans from many job opportunities, also restricted the educational opportunities that an African-American could achieve in the South. Unable to acquire the skills to garner higher wages, African-Americans were forced north in hopes of better educational and economic opportunities (Mathieu, 2009, pp. 19–20). Coupled with political disenfranchisement and racially motivated violence, African-Americans had few reasons to stay in the American South (Tolnay, 1997, p. 1214). This trend continued into the 1970s as social reformation in the South still lagged behind that of the North (Mathieu, 2009, pp. 19–20).

(Smallwood & Elliot, 1997)

DESTINATIONS AND RATIONALE

Much of the African-American migration out of the South was in pursuit of northern urban centers. Initially, African-Americans headed to cities thanks to what Mathieu describes as "an insatiable demand for labor in sectors like coal, steel, meatpacking, railroading, and war industries" (Mathieu, 2009, p. 20). In the decade that followed 1910, the black populations in Chicago, Toledo, and Detroit grew by 148 percent, 200 percent, and 611 percent respectively. Mathieu notes, "of course, African-Americans headed west as well, with the Pacific coast's black population increasing nearly six fold from 1930 to 1950" (Mathieu, 2009, p. 21). Even by the late 1950s, urban in-migration was still constant. The motivation, however, had evolved to where African-Americans desired urban life thanks to the racially affirming outlets and institutions common in urban settings but rare in rural areas (Price-Spratlen, 2008, p. 458).

Additionally, just as many African-Americans chose to seek out cultural centers in the north and west, so too did they seek unique and different locations. Some degree of migration destination decisions revolved not around where other African-Americans were, but rather where other African-Americans were not. Price-Spratlen described this process as destination diffusion, by which America saw "a broadening of the number and type of urban destinations" sought by African-Americans (Price-Spratlen, 2008, p. 461).

Conceptual Framework

Study Population Defined

African-Americans are much more likely to experience a stroke than their Caucasians. Of interest, African-Americans living in the Stroke Belt face an even greater risk of stroke compared to African-Americans living outside the Stroke Belt. While stroke primarily affects older persons, this study wanted to understand the impacts of

early life factors on later stroke outcome. Age 30 was selected as the upward limit of location analyses as it represented the age at which a person was likely settled and no longer migrating due to economic or educational interests. In doing so, it allowed this study to evaluate the longer-term impact of migration. To not get ahead of oneself, in order to understand why African-Americans living in the Stroke Belt are at greater risk for stroke than African-Americans living elsewhere, one must first reflect on the historical motivations behind African-American migration, which gives this study evidence of potential motivations in its own study sample.

Racism and Dichotomy of Opportunity in the Stroke Belt

Starting at the close of the American Civil War, African-Americans traveled out of the Stroke Belt, settling in other places across the United States in what would later be known as The Great Migration. African-Americans, newly freed by the end of the war and the passage of the 13th Amendment to the United States Constitution, wished to pursue economic and education opportunities, but found themselves unable to do so in the highly bigoted and institutionally racist Stroke Belt. This trend would continue into the mid-twentieth century as many states in the Stroke Belt introduced Jim Crow laws aimed at limiting access to economic and education opportunities for African-Americans (Mathieu, 2009, pp. 19–20). Desiring to pursue economic and education opportunity but limited by societal and institutional structures, African-Americans had little choice but to leave the Stroke Belt to achieve opportunities already available to Caucasians living in the Stroke Belt. African-Americans left in droves, settling in cities with more liberal laws and policies, where they were free to pursue economic interests and gain educations (Tolnay, 1997).

Big Picture

African-Americans, who were born in the Stroke Belt and moved elsewhere, did so to pursue economic and education opportunities. Entrenched racism limited the success that African-Americans could achieve in the Stroke Belt to only a fraction of their Caucasian counterparts. In leaving and moving to more progressive cities, African-Americans were able to achieve some semblance of the opportunity afforded to Caucasians living in the Stroke Belt. With these economic and education opportunities, comes an improvement in one's life. These improvements are further compounded by the fact that economic opportunity increases education opportunity, which in turn increases economic opportunity. With increased education and economic success, a person has greater access to healthcare as well as the ability to lead a healthier lifestyle. Over years and years, the availability of such opportunities leads to a profound impact on one's health and specifically one's potential stroke risk.

Caucasians remain at lower stroke risk, both in and out of the Stroke Belt, when compared to African-Americans because Caucasians likely face fewer hurdles in terms of economic and educational opportunities. African-Americans who live out of the Stroke Belt, while experiencing a reduced stroke risk compared to their Stroke Belt counterparts, are still worse off in terms of stroke risk compared to Caucasians. This is explained by the fact that while an African-American living outside of the Stroke Belt may experience less racism and social limitations, racism, whether de facto or de jure, likely exists everywhere.

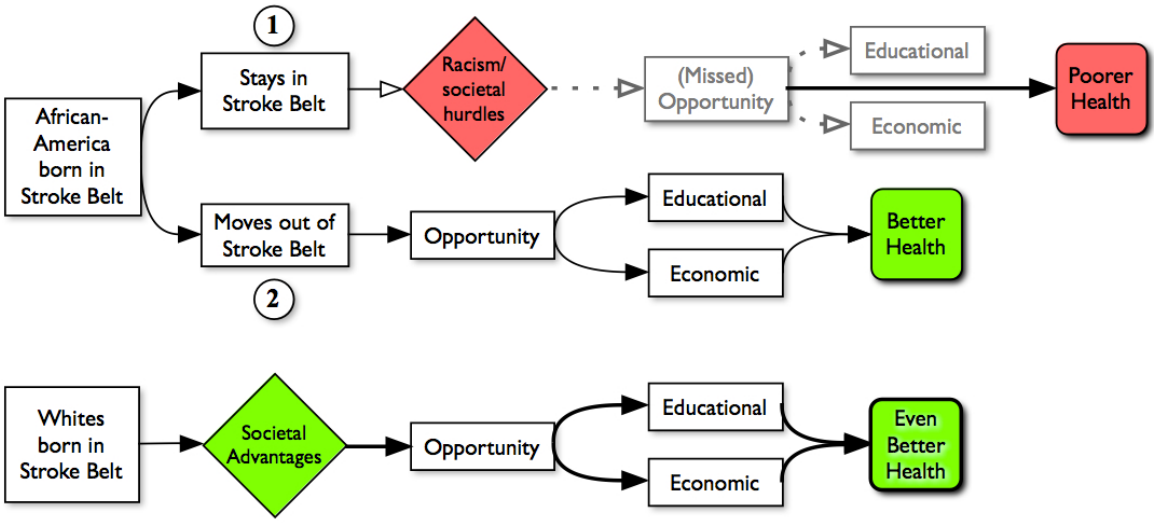
In conclusion, African-Americans who choose to leave the Stroke Belt likely find themselves in a more accepting and open minded culture, giving them the opportunity to pursue both economic and educational opportunities. These opportunities, in turn,

improve one's general health by increasing both healthcare knowledge and access to healthcare. The earlier that these opportunities can be achieved, the sooner one can improve their general health. As such, African-Americans who chose to leave the Stroke Belt earlier in their life will experience a greater duration in a status of improved health, which will result in a larger reduction in stroke risk than that of someone who chooses to leave the Stroke Belt later.

Concept of Location Exposure Study

Tasked with interpreting a wealth of location exposure data, this study constructed a number of variables, different perspectives by which to evaluate Stroke Belt exposure. The first variable developed analyzed the number of years a person lived in the Stroke Belt up to age 30. If a person was born in the Stroke Belt and lived there the full 30 years, they were considered as someone who "stayed" in the Stroke Belt. All others who were also born in the Stroke Belt but who later left were considered as someone who "left" the Stroke Belt. Another perspective took all location data into account to create a variable representing the portion of life spent in the Stroke Belt as a percentage of total life. Yet another perspective used data on the age when a person left the Stroke Belt, provided he or she was born there. Responses were recorded in a categorical variable with the following responses: left before age 5, left between ages 5 and 18, left between ages 18 and 30, and stayed in the Stroke Belt. Under age 5 represented persons who left the Stroke Belt during childhood. Ages 5 to 18 represented migration likely with one's family, perhaps for educational reasons. Ages 18 to 30 then likely represent migration based on economic and educational opportunity.

Representation of Conceptual Framework



Study Question: Do African-Americans at (2) fare better than those at (1) in terms of history of stroke?

MATERIALS AND METHODS

Study Background

Data Source

The REasons for Geographic And Racial Differences in Stroke (REGARDS) study is a national, longitudinal study sponsored by the National Institutes of Health is a national cohort study of stroke incidence in over 27,000 individuals over the age of 45. Enrollment in the study took place between 2003 and 2007 and was based on commercially available lists of residents. Study subjects were contacted initially by mail and were followed up via telephone. Telephone interviews, which were aided with the use of computer software, were used to obtain demographic, medical, and lifestyle data. Data on a variety of known stroke risk factors was also gathered from study participants. Following the initial phone interview, study subjects underwent a brief physical exam. Follow-up occurred at an interval of 6 months by telephone for updates regarding self-reported, suspected stroke ("About Regards," n.d.).

Variables

LOCATION AND AGE DATA

Upon completion of the in-person assessment, self-administered questionnaires were given to each study participant. Questionnaires were packaged in self-addressed prepaid envelopes to ease return and ensure high completion rates. On each questionnaire, study participants were asked to record the location of birth and the combination of city, state, and age at which he or she moved for each location lived across his or her life. To exclude temporary or short term living locations, participants were asked to only record locations at least one year in duration. Upon return, data were

entered into a central location repository, matching data to standard US Census Bureau Federal Information Processing Standards (FIPS) codes when possible.

OUTCOME VARIABLE

History of stroke, the outcome variable in analyses, was defined by self-report, past stroke symptoms, or both. Self-reported stroke was indicated on study questionnaire materials sent to study participants at intake and at intervals of six months after participant entered study. During physical exam, medical personnel noted reported past presence of stoke symptoms as well as evaluated medical history for such information. In the event that a participant passed away in between follow-ups, a final survey was completed by a family member or person connected to the deceased. Cause of death was recorded so as to identify deaths due to stroke.

COVARIATES

Additional study variables were used as covariates in analyses. Included variables age, sex, education category, income category, possess health insurance?, relationship status, alcohol use, tobacco use, television watching, exercise category, body mass index, and general health were defined by self-report.

Data Preparation

Software and Assumptions

All analyses were conducted using SAS Enterprise Guide 4.3 and Microsoft Excel 2011/2010. Missing values for any variable caused the entire subject to be excluded from analysis, unless otherwise stated. Chi-squared analysis assumed independent sample and individual cell counts > 5.

Sample Creation

The study sample used for analyses consisted of African-Americans born in the Stroke Belt who did not have missing data for age or gender. Study participants missing location data were also eliminated. Based on these criteria, a study sample of 6,983 persons was selected. To prevent further reduction of the study sample size, missing values were recoded as a categorical response. SAS will not evaluate a record if the response to any variable under consideration is missing. Missing values were recoded as a response category, so SAS would treat them just as any other response. The missing counts were as follows: education category, 6; possess health insurance, 9; television, 1,506; exercise category, 100; smoking status, 36; BMI category, 66; general health, 15. The states of origin from the Great Migration fully overlap with those of the Stroke Belt. Thus, a sample from the REGARDS study of African-Americans born in the Stroke Belt, therefore, also represents the population described in the Great Migration literature.

Location Data

Unlike the clinical and behavioral data which had one record for each study ID number, the location data had one record for each place lived across and ID number's life. In order for this data to be usefully integrated into the clinical data, each of these location records needed to be summarized so that each ID number only possessed one location record. To do this, Microsoft Excel was primarily used. First, if the record was the first place lived for a person (order equaling one), and the location was in the Stroke Belt, then the record was given a value of one for being born in the south. All other order and state combinations were given a value of zero. Thus, when the records are combined through summing, only persons with a value of one for the born in south variable would

have been born in the Stroke Belt and all others would have therefore not been born in the Stroke Belt.

A variable was created which only considered location data up to age 30. For each record, the number of years spent in the Stroke Belt and number of years spent outside the Stroke Belt up to a combined 30 years were recorded. Records were then evaluated if the person was born in the Stroke Belt and stayed there until at least age 30, in which case he or she received a response of 1 indicating they "stayed" in the Stroke Belt. All other respondents, those persons not born in the Stroke Belt or persons who left the Stroke Belt before age 30 were given a response of 0 indicating they "left" the Stroke Belt. Inclusion in the stroke-belt region was based on the Howard et al. definition which includes North Carolina, South Carolina, Georgia, Tennessee, Mississippi, Alabama, Louisiana, and Arkansas (Howard et al., 2007, p. 1).

Using data on the age at which a person left the Stroke Belt, the year in which the person left the Stroke Belt was recorded. This date was a calculation of the study interview date minus the age at the time of the interview, plus the age at which the person left the Stroke

Belt provided they were born there. The distribution of years in which respondents left the Stroke Belt were recorded and displayed in a histogram as well as in Table 2 along with other summary statistics.

Using a PivotTable in Microsoft Excel, these data were combined, stratifying by each ID number. In the end, only one record remained for each ID number, all variables having been summed across the numerous records relating to each ID number. These new data were then imported into SAS and merged with the non-location data within the

data step, merging based on matched ID number. Failure to match across ID number resulted in the record being excluded from analysis, which reduced the study population by 3,112 persons.

Data Filtering and Variable Selection

Each variable was evaluated for inclusion in the analyses. Many variables were collected on each study participant, ranging from variables on television watching activity, to locations lived, to the number of pillows used at night. Variables were chosen from this list based on their relation to the conceptual framework described earlier. Variables were classified in one of three groups: motivations and outcomes of migration, behavioral changes that could be brought about by migration, and health impacts from migration outcomes. This first group, those variables that indicate motivations and potential outcomes of migration, included education category (Some High School, High School Graduate, Some College, College Graduate+, Missing), income category (<$20k, $20-34k, $35-75k, $75k+, Refused), whether the patient possesses health insurance (Yes, No, Missing), and relationship status (Single, Married, Divorced, Widowed, Other). The second group, variables that captured the behavioral impacts of location on one's life, included hours of television watched (None, 1-6 hrs./wk., 1 hr./day, 2 hrs./day, 3 hrs./day, 4+ hrs./day, Missing), alcohol use (Never, Current, Past), exercise category (Never, 1-3 Times/Wk., 4+ Times/Wk.), and tobacco use (Never, Current, Past, Missing). The third and final group, looking to control for the health impact of migration or staying in the Stroke Belt, included body mass index category (Normal, Obese, Overweight, Underweight, Missing) and general health status (Poor, Fair, Good, Very Good, Excellent, Missing).

Data Cleaning and Coding

Once study variables were selected from the database, a process of data cleaning and recoding was undertaken. This process involved the elimination of implausible responses. First each variable underwent frequency or univariate analyses based on whether it was categorical or numerical respectively. For categorical variables, this was done to ensure no errors in data entry. For example, for each dichotomous variable, responses should be limited to "yes", "no", and missing responses, and a fourth option would obviously be erred. Based on initial analyses, none of the categorical variables contained any of these erred responses and therefore required no cleaning.

Categorical variables arrived coded in the "yes/no" format. To better allow SAS to interpret these variables the following were recoded to 1 for "yes" and 0 for "no." Some categorical variables had additional levels and were recoded accordingly.

Finally, each categorical study variable was dummy coded. To do so, conditional statements would identify the response of each variable and assign it to a variable specific to both that variable and that specific response. Thus, for a categorical variable with six possible responses, six dichotomous dummy variables were created.

Initial Analyses

Univariate Analyses

Table 3 contains descriptive univariate statistics for the numerical and categorical variables of the study. Variables are grouped by categories described above. Analyses were stratified by whether the participant stayed in the Stroke Belt until or left before age 30, and the table records means, standard deviations, and counts for each variable/level.

Bivariate Analyses

Table 4 contains means for each variable/level, stratified into four categories: stayed in Stroke Belt and had indications of stroke, stayed in Stroke Belt and did not have stroke, left Stroke Belt and had indications of stroke, and left Stroke Belt and did not have indications of stroke. Using persons who stayed in the Stroke Belt and had indications of stroke as the reference category, bivariate analyses were conducted to evaluate the statistical significance of shifts in means across strata. Superscript characters are used to denote the degree of significant associations. The full set of these analyses can be found in Table 4.

Modeling

Modeling and Diagnostics

To study the association of location on indications of stroke, several associative models were created. In each model, the outcome variable was stroke history as judged by self-report or stroke symptoms. Each model underwent diagnostics to check for collinearity and was ensured of proper calibration. The odds ratios and 95% confidence intervals for the variable of interest were outputted for each model. Shifts in the odds ratios were used to determine the impact of adjusting for different factors. Confidence intervals were used to determine if these shifts in associations were statistically significant.

Associative Models

To understand and study the effect of location from different perspectives, three sets of models were created. Each set included one of the three location variables created earlier: stayed in or left Stroke Belt by age 30, portion of life spent in the Stroke Belt, and the age bracket in which one left the Stroke Belt. Using the classification of variables

noted earlier as groupings, a set of 4 different models was created. In the first model, the location variable of interest was adjusted for age and gender. In the second model, the first model was further adjusted for variables that represent motivations of migration: education, income, health insurance, and relationship status. In the third model, television watching, exercise, tobacco use, and alcohol use were also controlled for. These variables represent behavior that could come as a result or indication of migration out of the Stroke Belt or lack there of. In the fourth and final model, we further adjusted for body mass index and general health status. This set of four models was conducted for each of the three location variables, producing twelve models in total. The odds ratios and 95% confidence intervals for each variable are shown in Table 5 – Table 10 below.

Analyses evaluated the impact of adjusting for additional variables on the significance and strength of the association between each location variable and indications of stroke outcome. Model 1 was considered the reference model for each set of models, and was therefore considered as the crude model. With each addition of more variables being adjusted for, the models were evaluated as to whether the effect of location on indications of stroke could be explained.

RESULTS

Descriptive Statistic Results

Location Findings

The histogram seen below in Graph 1 represents the distribution of years in which African-American respondents, who were born in the Stroke Belt, left the Stroke Belt region. Values are displayed as a percent of total study participants. The vast majority of migration of study participants took place between 1940 and 1970, which is very much in line with accounts from literature describing a third phase of the Great Migration taking place between early 1940s and the 1970s. Additional statistics on migrations, broken down by decade, is recorded below in Table 2.

Univariate Analyses

There exists a large disparity between education levels amongst persons who stayed in the Stroke Belt and those who left. Amongst those who stayed, only 21% had some college compared to 28% amongst persons who left the Stroke Belt. At the college grad+ level only 22% of persons who stayed had achieved this level compared to 25% of those who left the Stroke Belt. On the low end of the income scale, 34% of persons who stayed in the Stroke Belt reported earning less than $20k a year as opposed to only 26% of those persons who left. In terms of alcohol use, 46% of persons staying in the Stroke Belt had never consumed alcohol compared to 33% of those who left. Similarly, 54% of respondents who stayed in the Stroke Belt had never smoke compared to only 42% of those persons who left the Stroke Belt. Full univariate analyses are shown in Table 3.

Bivariate Analyses

In the bivariate t-test analyses, persons who were born in the Stroke Belt and stayed to at least age 30 and had indications of stroke (South-Stroke) served as the

reference group in comparing means against persons who stayed in Stroke Belt and did not have indications of stroke (South-No-Stroke), left Stroke Belt and had indications of stroke (North-Stroke), and left Stroke Belt and did not have indications of stroke (North-No-Stroke) groups. Using bivariate analyses, there exists a significant shift in means of the two gender variables levels (male, female) between the reference group and groups North-Stroke and North-No-Stroke, both at the 0.001 level. Education (Less than HS) had a significant shift between all groups at the 0.001 level. Income (<$20k) varied significantly between each group and the reference group. Shifts with groups South-No-Stroke and North-No-Stroke were at the 0.001 level while group North-Stroke was significant at the 0.01 level. Television (3 hr./day) also varied significantly across all groups at the 0.001 level except for group North-Stroke which was at the 0.05 level. Full bivariate analyses are shown in Table 4.

Multivariate Associative Modeling Results

Modeling–Left Stroke Belt By Age 30

In set A of associative models, the first model evaluated the association between being born in Stroke Belt and staying to at least age 30, age, and gender on stroke outcome: being born in Stroke Belt and staying to at least age 30 and Age were significantly associated with indications of stroke. Being Born in the Stroke Belt and staying there to at least age 30 resulted in an odds ratio of 1.18 (95%CI: 1.06, 1.32), staying in the Stroke Belt until age 30 resulted in an 18% higher odds of experiencing a stroke at some point in one's life as compared to persons who were born in the Stroke Belt but moved elsewhere before age 30. However, in model 2, with the introduction of variables representing the motivations for migration out of the Stroke Belt, the location variable grew statistically insignificant with an odds ratio of 1.06 (95%CI: 0.94, 1.18).

This means that when controlling for factors that would lead someone to migrate out of the Stroke Belt, the migration itself was no longer significantly associated with stroke indications. Instead, men, and persons who are HS grads, and persons who attended some College, & college grads, and persons earning between $20-75k+, and persons in relationships labeled as "other" all have significant associations with indications of stroke. In the third model, which also adjusted for a host of behavioral variables indicative of one's location and its impact on health, being born in and staying in the Stroke Belt remained statistically insignificant with an odds ratio of 1.06 (95%CI: 0.95, 1.19). It was hypothesized that adding in these additional behavioral factors would further diminish the association between indications of stroke history and migration out of the Stroke Belt by age 30. However, in not modifying the odds ratio, these behavioral factors fail to further explain the differences in indications of stroke seen across persons who migrated by age 30 and those who did not. A fourth and final model adjusted for body mass index and general health status, health outcomes of one's location, also demonstrated a statistically insignificant association between being born in Stroke Belt and staying to at least age 30 and indications of stroke history. Significant variables in this model were as follows: Gender (male), Education Category (Some College), Income Category ($35-74k & $75k+), Relationship Status (Other), Alcohol Use (Current), Exercise Category (1-3 Times/Wk.), Tobacco Use (Current), and General Health (Excellent, Very Good, Good, Fair, & Missing). Full listings of odds ratios and 95% confidence intervals are located in Table 5 and Table 6.

Modeling–Portion of Life Spent in Stroke Belt

In the next set of associative models, set B, the first model evaluated the association between the portion of total life spent in Stroke Belt, age, and gender on

stroke outcome. Like in set A, this first model demonstrated a statistically significant association between indications of stroke history and the location variable as well as age. The association between Portion of Life Spent in Stroke Belt and stroke history had an odds ratio of 1.26 (95%CI: 1.08, 1.48) meaning that for each percentage point increase in portion of life spent in the Stroke Belt, one's odds of stroke indications was 0.26% higher. In model 2, like in set A, the introduction of migration motivation variables caused the association between portion of life spent in the Stroke Belt and indications of stroke history to become statistically insignificant with an odds ratio of 1.14 (95%CI: 0.97, 1.34). Model 2 of set B displayed the same significance as model 2 of set A above. Like before, when controlling for factors that would lead someone to migrate out of the Stroke Belt, the time spent in the Stroke Belt itself was no longer robust to indications of stroke outcome . Adding additional behavior variables in model 3 resulted in a yet further insignificant association between portion of life spent in Stroke Belt and indications of stroke history, with an odds ratio of 1.16 (95%CI: 0.98, 1.36). Variables displaying significant associations were Gender (male), Education Category (HS Grad, Some College, & College Grad+), Income Category ($20-34k, $35-74k, & $75k+), Relationship Status (other), Alcohol Use (Current & Past), Exercise Category (1-3 Times/Wk. & 4+ Times/Wk.), and Tobacco Use (Current). In the fourth and final model, adding health outcome variables resulted in a similarly insignificant association between portion of life spent in the Stroke Belt and stroke history, displaying an odds ratio of 1.12 (95%CI: 0.94, 1.32). Model 4 did, however, display statistically significant associations between the following variables and indications of stroke history: Gender (male), Education Category (Some College), Income Category ($35-74k & $75k+), Relationship

Status (other), Alcohol Use (Current), Exercise Category (4+ Times/Wk.), Tobacco Use (Current), and General Health (Excellent, Very Good, Good, Fair, & Missing). Full listings of odds ratios and 95% confidence intervals are located in Table 7 and Table 8.

Modeling–Age At Which One Left Stroke Belt

In the next set of associative models, set C, the first model evaluated the association between the age bracket in which one left the Stroke Belt, age, and gender on stroke outcome. This first model demonstrated a statistically significant association between persons leaving the Stroke Belt during the 18-30 age bracket and stroke history. The other levels, however, were statistically insignificant. The association between leaving the Stroke Belt during this 18-30 age window and stroke history had an odds ratio of 0.75 (95%CI: 0.65, 0.86) compared to the reference category of those persons who stayed in the Stroke Belt. Meaning an African-American who leaves the Stroke Belt between the ages 18 and 30 has 25% lower odds of having a stroke later in life compared to someone who stayed in the Stroke Belt until at least age 30. In model 2, like in set A and set B, the introduction of migration motivators caused the association between age bracket in which one left the Stroke Belt and indications of stroke history to be come statistically insignificant at all levels. The odds ratio for the ages 18-30 level, which had been significant in model 1, became 0.872 (95%CI: 0.76, 1.002). Instead, Gender (male), Education Category (HS Grad, Some College, & College Grad+), Income Category ($20-34k, $35-74k, & $75k+), and Relationship Status (Other) displayed statistically significant associations with stroke history. Again, when controlling for factors that would lead someone to migrate out of the Stroke Belt, the age at which one left the Stroke Belt itself became statistically insignificant. Controlling for additional behavior variables in model 3 again resulted in insignificant associations between age at which one

left the Stroke Belt and stroke history at ever level. Variables displaying significant associations were Gender (male), Education Category (HS Grad, Some College, & College Grad+), Income Category ($20-34k, $35-74k, & $75k+), Relationship Status (other), Alcohol Use (Current & Past), Exercise Category (1-3 Times/Wk. & 4+ Times/Wk.), and Tobacco Use (Current). In the fourth and final model, adding health outcome variables resulted in a similarly insignificant association between each level of age bracket when one left the Stroke Belt and indications of stroke history. Variables Gender (male), Education Category (Some College), Income Category ($35-74k & $75k+), Relationship Status (other), Alcohol Use (Current), Exercise Category (4+ Times/Wk.), Tobacco Use (Current), and General Health (Excellent, Very Good, Good, Fair, & Missing) had statically significant associations with indications of stroke history in model 4. Full listings of odds ratios and 95% confidence intervals are located in Table 9 and Table 10.

DISCUSSION

This study draws upon the historical Great Migration, creating a quasi-natural experiment amongst African-Americans born in the Stroke Belt from the REGARDS study to evaluate if African-Americans who migrated out of the Stroke Belt experienced better stroke health outcomes than other African-Americans left behind in the Stroke Belt. Using historical accounts and studies of the Great Migration to identify motivations for migration amongst African-Americans migrating between 1940 and 1970, the same the same time frame in which the majority of the study sample migrated out of the Stroke Belt, this study sought to test whether the migration itself or the motivations and outcomes of the migration were the true risk modified for stroke. Using three sets of modeling, evaluating exposure to the Stroke Belt from three different perspectives, this study comprehensively evaluates this association. First, migration showed a significant association with stroke history; however, when the motivations and outcomes of migration were added to the picture, the migration itself no longer remained significant. Migrating out of the Stroke Belt afforded African-Americans more economic and educational opportunities. One must keep in mind that even with these findings, persons living in the Stroke Belt region do in fact still have higher odds of stroke, especially African-Americans. These odds, however, come not as a result of location but instead as a function of the economic and educational opportunity for African-Americans in the Stroke Belt.

Desiring to explain the mechanism behind this observation, this study hypothesizes that racism and organized disenfranchisement seen in the Stroke Belt did not exist in the north and west to the extent that it did in the Stroke Belt, allowing

African-Americans to garner higher wages and gain and education (Mathieu, 2009, pp. 19–20). Through this socioeconomic improvement, African-Americans outside the Stroke Belt had better access to healthcare as well as were able to maintain healthier behaviors as seen in the modeling involving behavioral factors. The combination of better access to health care and living a healthier lifestyle afforded African-Americans outside the Stroke Belt a stroke risk far improved over their counterparts who remained in the Stroke Belt.

If an African-American living in the Stroke Belt were afforded the same economic and educational opportunities that he or she would outside the Stroke Belt, he or she could be expected to experience same odds of stroke regardless of his or her location. Unfortunately, these economic and educational opportunities are often out of reach of African-Americans living in the Stroke Belt, either due to racism or organized disenfranchisement. Thus, in many cases, the only way that African-Americans can achieve these opportunities is through migration outside of the Stroke Belt. In doing so, they are able to reap the benefits in terms of reduced odds of stroke, but again this reduction comes not as a result of the migration itself but as a result of the opportunities made available by the migration (Tolnay, 1997).

These findings have widespread implications in how the United States and other nations address stroke risk in their populations. Traditionally, stroke risk has been managed through the management of those clinical risk factors for stroke. However, these indicators often emerge later in life. These factors are just as much a health outcome as the stroke they lead to. Management of these indicators, then, is akin to placing a Band-Aid on the problem, failing to address the root cause in the first place.

Using the findings of this study, which demonstrate that indications of potential stroke later in life are a function of the economic and educational opportunities afforded to a person early on, healthcare professionals can prioritize prevention over basic treatment. Ensuring that a person is afforded economic and educational opportunities no matter where he or she lives will result in a population that is healthier and experiences fewer of the clinical stroke risk indicators in the first place. Thus, the most effective way to address stroke is not by the treatment of risk factors for stroke once they arise, but rather to prevent those risk factors from ever occurring in the first place. In doing so, not only will stroke incidence go down, but so too will other negative health outcomes.

This study looked to evaluate the association between location and history of stroke. In many regards, this study entered uncharted territory in its study of indications of potential stroke and those factors that modify it. While a number of studies have attempted to evaluate location's impact on history of stroke, these studies only use exposure to the Stroke Belt as an intermediary for exposure to such factors as temperature, pollution, and neighborhood greenness. Hong et al. found that cold weather, especially cold weather during the winter months, was associated with ischemic stroke risk (Hong et al., 2003). Another study, however, found that there was no consistent relationship between temperature change and stroke risk, except for large day-to-day shifts (Kyobutungi et al., 2005, p. 696). Basu et al. hypothesized that stroke risk increased during periods of higher ambient temperatures, but only found a significant association of increased ambient temperature and stroke risk amongst persons aged 65 and younger. This population, though, is already less likely to have strokes (Basu et al., 2012, p. 817). On the pollutant front, Joubert et al. found exposure to a set of air

pollutants resulted in a 5% increase in stroke incidence risk, with a 3-6% increase in stroke mortality on top of that (2007, p. 74). As one can see, these studies draw upon location only as an intermediary determinant of other factors be it temperature or pollution. Further, these studies only evaluate short-term consequences of these exposures, evaluating associations between events within days or weeks of the stroke event and the stroke event.

Rather than evaluate short-term impacts of migration, the study evaluated migrations out of the Stroke Belt taking place before age 30, years in advance of any possible stroke event. This was driven by two factors. One, stroke most often occurs later in life and migrations are unlikely to take place around the same age or even decade of one's life. Two, this study wished to evaluate not only the impact of location but also the impact of factors motivating or resulting from migration. While someone migrating in his or her early twenties may do so for economic and educational opportunity, a person in his or her eighties is unlikely to do so for the same motivations. Additionally, the impact of economic or educational improvement to a person in his or her twenties will be far larger than for someone in his or her eighties, given that for the eighty year old, much of his or her health impacts from events earlier in life will already be set in stone. In studying only migrations that took place before age 30, this study was able to evaluate the long-term impacts of economic and educational opportunity on history of stroke. One should note that the exact motivation for migration are unknown but can be hypothesized on based on those migration outcome variables.

The strength of this study came from its ability to using historical information on the Great Migration to create a massive natural experiment. The Great Migration, the

giant internal migration of African-Americans out of the American South to places elsewhere in the United States, prompted a vital and yet unanswered question: did African-Americans leaving the South have better health outcomes than those who stayed in terms of stroke history? Drawing upon African-Americans born in the Stroke Belt, an area lining up with the southern region described in Great Migration literature, from the REGARDS study, this study selected a sample of persons whose migration fell into the Great Migration context. This study then evaluated the association between migration out of the Stroke Belt and history of stroke, later adjusting for factors that motivated and resulted from migration out of the Stroke Belt, as determined by Great Migration literature. In drawing a connection between the Great Migration and the REGARDS study, this study was able to strengthen its findings though the collective knowledge on both indications of potential stroke and the Great Migration history.

Limitations

The lack of access to the "age at first stroke" variable limited analyses based on time spent in the Stroke Belt across one's life. Analyses that only took into account the first 30 years of life were likely unaffected as stroke is extremely uncommon in these young ages. However, the portion of life spent in the Stroke Belt variable was likely affected. Without knowing the age when a stroke occurred, one will unknowingly include location data that occurred after the stroke event. As expected, this information should not be part of analyses as they violate temporality in the study of associations between location and stroke history. While the variable does exist in the study, the inability to establish rights to the specific variable made access impossible.

Future Research

Having demonstrated that the association between living in the Stroke Belt and stroke history can explained by adjusting for variables representing motivations for migration, the concept of the Stroke Belt should be reconsidered. Differences between life inside and outside can be reduced to a difference in opportunity and location itself is not what affects one's stroke risk. These findings, however, do not eliminate the possibility of location affecting one's stroke risk. Instead, these data show that the Stroke Belt as it is described (North Carolina, South Carolina, Georgia, Tennessee, Mississippi, Alabama, Louisiana, and Arkansas) should be re-evaluated (Howard et al., 2007, p. 1). Given that analyses depended on categorizing each participant as in the Stroke Belt or out of the Stroke Belt based on this list, modifying the list could have a large impact on study finds. Thus, future research should evaluate different states of the Stroke Belt separately. True associations may have been masked by others states displaying no or opposite associations. Further research could also investigate not only location but also the years in which each person lived in each location. As analyzed in this study, all location data was evaluated in a cross-sectional fashion. It is possibly that associations between location and stroke risk change over time and that without analyzing both the location and the years, it would be difficult to identify this trend.

CONCLUSIONS

In the study of ischemic stroke risk, clinical indicators have long been the center of attention and, as a result, the focus of many analyses. While clinical indicators certainly play a large role in one's eventual stroke risk, they serve perhaps only as intermediaries in the chain of events that terminate with a stroke event. Rather than focus on these clinical factors, which themselves are health outcomes just as a stroke is, this study evaluated the impact of one's location over the course of his or her life on stroke outcome. This study sought to evaluate if perhaps larger societal forces that may be at play in determining indications of potential stroke later in life, even from an early age. These findings may indicate ways in which policies and investments could in the wellbeing of persons from an early age could make a difference before any clinical outcomes is even established. The Stroke Belt, which includes North Carolina, South Carolina, Georgia, Tennessee, Mississippi, Alabama, Louisiana, and Arkansas, served as the center of these analyses. In a study sample of African-Americans born in the Stroke Belt, multivariate analyses determined a statistically significant association between the time spent in the Stroke Belt, as judged by various different variables (born in and migrated out of Stroke Belt by age 30 or stayed, portion of life spent in the Stroke Belt, and age at which one migrated out of the Stroke Belt, provided they were born there), and stroke history. We found that African-Americans who migrated out of the Stroke Belt experienced less stroke outcomes; however, when controlling for the factors that could motivate one to migrate, we saw that the migration itself no longer explained the decrease in indications of stroke. Rather the improvement in quality of life that comes from migration could explain the reduction in indications of stroke. Each initial model

indicating that moving out of the Stroke Belt or spending more of one's life outside the Stroke Belt was protective against stroke. Using a historical African-American migration know as the Great Migration as a natural study, information on the motivations that prompted many to migrate out of the Stroke Belt were subsequently used as controlled factors. When the multivariate models were adjusted for variables that represented motivations for migration, location in all of the studied forms grew statistically insignificant. The act of living in or moving out of the Stroke Belt was not the true link to stroke outcome, rather it was the opportunities both economic and education afforded to African-American only if they moved out of the Stroke Belt that proved to be the true indicator of protection against potential stroke later in life. Economic and educational opportunity impacted access to healthcare and knowledge of healthy behavior, which in turn affected those clinical indicators as well as stroke risk. An African-American, if afforded these economic and educational opportunities in the Stroke Belt, would likely have the same health outcomes as one living outside the Stroke Belt. However, be it racism or institutional, organized disenfranchisement taking place in the Stroke Belt, African-Americans are only able to achieve these opportunities if they are to migrate out of the Stroke Belt. This gives the appearance that migration out of the Stroke Belt itself is protective against stroke risk, but in truth, it is the economic and educational opportunities that come as a result of migration that truly modifies indications of stroke. Using this hypothesized information, health officials and policy makers hold great power to reduce stroke incidence. Rather than focus on treatment of clinical indicators of stroke, a point in time that is often too late to truly impact stroke incidence, treatment should give way to prevention. In ensuring that economic and educational opportunity is

afforded to African-Americans regardless of location, there will be profound changes in stroke outcomes as well as a reduction in many other negative health outcomes.

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TABLES

Table 1: Change In African-American Populations By Decade (In Thousands)

		1920	1930	1940	1950	1960	1970	1980	1990
Stroke Belt	Alabama	900.7	+44.2	+38.5	-3.7	+0.7	-76.8	+92.9	+24.4
	Arkansas	472.2	+6.2	+4.1	-55.9	-37.9	-36.3	+21.3	+0.1
	Louisiana	700.3	+76.1	+73.0	+33.1	+156.8	+47.6	+151.4	+61.0
	Mississippi	935.2	+74.5	+64.9	-88.1	-70.8	-100.0	+71.4	+27.9
	Tennessee	451.8	+25.9	+31.1	+21.9	+56.3	+34.4	+104.7	+52.1
Buckle	Georgia	1,206.4	-135.2	+13.8	-22.2	+59.8	+64.6	+278.0	+281.4
	N. Carolina	763.4	+155.2	+62.7	+66.1	+68.7	+10.5	+192.4	+137.5
	S. Carolina	864.7	-71.0	+20.5	+7.9	+7.2	-40.3	+159.6	+91.3
	Non-SB	4,168.6	+1,252.1	+665.9	+2,217.7	+3,588.7	+3,804.8	+2,843.0	+2,815.4

Source: U.S. Census Bureau (Gibson & Jung, 2002)

Table 2: Distribution of Years In Which One Left The Stroke Belt By Decade Amongst African-Americans Born in the Stroke Belt

Year	Gender	Average Age	Age At Move	Count Moved	Percent
1910s	Female	91.3	3.8	4	0%
1920s	Female	81.6	2.8	25	1%
	Male	82.2	3.9	20	
1930s	Female	75.9	7.6	71	3%
	Male	76.0	7.9	49	
1940s	Female	70.8	11.1	375	19%
	Male	72.7	13.2	271	
1950s	Female	66.2	16.1	639	32%
	Male	67.2	17.2	467	
1960s	Female	60.6	20.0	649	32%
	Male	59.9	19.6	451	
1970s	Female	55.7	23.4	194	10%
	Male	54.4	22.3	139	
1980s	Female	54.6	31.9	54	2%
	Male	53.7	31.6	27	
1990s	Female	57.5	44.8	17	1%
	Male	56.0	44.4	5	
2000s	Female	65.2	64.8	5	0%
	Male	54.0	55.0	2	

Source: REasons for Geographic And Racial Differences in Stroke (REGARDS) study
Sample: African-Americans Born in Stroke Belt Region (N: 6,983)

Table 3: Descriptive Statistics for Those Who Stayed in Stroke Belt v. Moved Out of Stroke Belt Amongst African-Americans Born in the Stroke Belt

		Born in Stroke Belt and stayed to at least age 30			Born in Stroke Belt and moved before age 30		
		Mean	SD	N	Mean	SD	N
Model 1 Bio-Demographic							
History of Stroke		0.27	0.45	838.00	0.25	0.43	972.00
Portion of Life Spent in Stroke Belt		0.98	0.09	3014.4	0.53	0.34	2055.2
Age One Left Stroke Belt	Stayed in SB	1.00	0.00	3072.0	(–)	(–)	(–)
Age One Left Stroke Belt	Age < 5	(–)	(–)	(–)	0.22	0.41	848.00
Age One Left Stroke Belt	Age 5 – 18	(–)	(–)	(–)	0.28	0.45	1095.0
Age One Left Stroke Belt	Age 18 – 30	(–)	(–)	(–)	0.50	0.50	1968.0 0
Age		63.14	9.34	3072	64.71	8.67	3911
Gender	Female	0.73	0.44	2255	0.58	0.49	2285
Gender	Male	0.27	0.44	817	0.42	0.49	1626
Model 2 Socio-Economic							
Education Category	Less than HS	0.28	0.45	860	0.18	0.39	721
Education Category	HS Grad	0.29	0.45	888	0.29	0.45	1121
Education Category	Some College	0.21	0.41	635	0.28	0.45	1090
Education Category	College Grad+	0.22	0.42	686	0.25	0.43	976
Education Category	Missing	0.00	0.03	3	0.00	0.03	3
Income Category	< $20k	0.34	0.48	1057	0.26	0.44	1010
Income Category	$20-34k	0.24	0.43	734	0.28	0.45	1079
Income Category	$35-74k	0.21	0.41	648	0.27	0.44	1037
Income Category	$75k+	0.06	0.24	183	0.09	0.28	345
Income Category	Refused	0.15	0.35	450	0.11	0.32	440
Possess Health Insurance	No	0.13	0.34	396	0.09	0.28	333
Possess Health Insurance	Yes	0.87	0.34	2671	0.91	0.28	3574
Possess Health Insurance	Missing	0.00	0.04	5	0.00	0.03	4
Relationship Status	Single	0.08	0.27	239	0.07	0.25	265
Relationship Status	Married	0.44	0.50	1366	0.47	0.50	1857
Relationship Status	Divorced	0.18	0.38	552	0.19	0.39	728
Relationship Status	Widowed	0.25	0.43	776	0.23	0.42	885
Relationship Status	Other	0.05	0.21	139	0.05	0.21	176
Model 3 Behavioral							
Television	None	0.00	0.06	13	0.00	0.07	17
Television	1-6 Hrs./Wk.	0.11	0.31	330	0.11	0.31	415
Television	1 Hr./Day	0.03	0.17	97	0.03	0.16	106
Television	2 Hrs./Day	0.09	0.29	291	0.13	0.33	502
Television	3 Hrs./Day	0.18	0.39	556	0.19	0.40	761

		Born in Stroke Belt and stayed to at least age 30			Born in Stroke Belt and moved before age 30		
		Mean	SD	N	Mean	SD	N
Television	4+ Hrs./Day	0.34	0.47	1053	0.34	0.47	1336
Television	Missing	0.24	0.43	732	0.20	0.40	774
Alcohol Use	Never	0.46	0.50	1406	0.33	0.47	1307
Alcohol Use	Current	0.32	0.47	987	0.41	0.49	1586
Alcohol Use	Past	0.22	0.42	679	0.26	0.44	1018
Exercise Category	Never	0.35	0.48	1084	0.37	0.48	1442
Exercise Category	1-3 Times/Wk.	0.25	0.43	768	0.26	0.44	1023
Exercise Category	4+ Times/Wk.	0.38	0.49	1175	0.36	0.48	1391
Exercise Category	Missing	0.01	0.12	45	0.01	0.12	55
Smoking Status	Never	0.54	0.50	1653	0.42	0.49	1644
Smoking Status	Current	0.15	0.36	468	0.18	0.38	686
Smoking Status	Past	0.30	0.46	932	0.40	0.49	1564
Smoking Status	Missing	0.01	0.08	19	0.00	0.07	17
Model 4 Health							
BMI Category	Normal	0.16	0.36	485	0.17	0.37	651
BMI Category	Obese	0.52	0.50	1606	0.47	0.50	1831
BMI Category	Overweight	0.30	0.46	921	0.35	0.48	1362
BMI Category	Underweight	0.01	0.09	23	0.01	0.10	38
BMI Category	Missing	0.01	0.11	37	0.01	0.09	29
General health	Poor	0.05	0.22	157	0.04	0.19	151
General health	Fair	0.25	0.43	768	0.21	0.41	829
General health	Good	0.41	0.49	1274	0.41	0.49	1608
General health	Very good	0.21	0.41	639	0.24	0.43	928
General health	Excellent	0.07	0.26	226	0.10	0.30	388
General health	Missing	0.00	0.05	8	0.00	0.04	7

Source: REasons for Geographic And Racial Differences in Stroke (REGARDS) study
Sample: African-Americans Born in Stroke Belt Region (N: 6,983)

Table 4: Descriptive statistics by Stayed in Stroke Belt v. Moved and Stroke History amongst African-Americans Born in the Stroke Belt

		Born in Stroke Belt and stayed to at least age 30		Born in Stroke Belt and moved before age 30	
		Stroke	No Stroke	Stroke	No Stroke
Model 1 Bio-Demographic					
Age		63.86	62.86[b]	65.67[a]	64.4
Gender	Female	0.70	0.75[c]	0.58[a]	0.59[a]
Gender	Male	0.30	0.25[c]	0.42[a]	0.41[a]
Model 2 Socio-Economic					
Education Category	Less than HS	0.35	0.25[a]	0.27[a]	0.15[a]
Education Category	HS Grad	0.32	0.28[c]	0.28[d]	0.29
Education Category	Some College	0.17	0.22[b]	0.26[a]	0.29[a]
Education Category	College Grad+	0.16	0.25[a]	0.19	0.27[a]
Education Category	Missing	0.00	0.00	0.00	0.00[d]
Income Category	< $20k	0.41	0.32[a]	0.34[b]	0.23[a]
Income Category	$20-34k	0.24	0.24	0.28[c]	0.27[c]
Income Category	$35-74k	0.16	0.23[a]	0.18	0.29[a]
Income Category	$75k+	0.03	0.07[a]	0.05[b]	0.1[a]
Income Category	Refused	0.17	0.14[d]	0.14	0.1[a]
Possess Health Insurance?	No	0.14	0.13	0.1[b]	0.08[a]
Possess Health Insurance?	Yes	0.86	0.87	0.9[c]	0.92[a]
Possess Health Insurance?	Missing	0.00	0.00	0.00[d]	0.00
Relationship Status	Single	0.07	0.08	0.06	0.07
Relationship Status	Married	0.43	0.45	0.43	0.49[b]
Relationship Status	Divorced	0.16	0.19	0.19	0.19[d]
Relationship Status	Widowed	0.27	0.25	0.27	0.21[b]
Relationship Status	Other	0.07	0.04[a]	0.05	0.04[b]
Model 3 Behavioral					
Television	None	0.01	0.00	0.00	0.00
Television	1-6 Hrs./Wk.	0.11	0.11	0.09[d]	0.11
Television	1 Hr./Day	0.03	0.03	0.03	0.03
Television	2 Hrs./Day	0.08	0.1[c]	0.11[c]	0.14[a]
Television	3 Hrs./Day	0.14	0.2[a]	0.17[c]	0.2[a]
Television	4+ Hrs./Day	0.40	0.32[a]	0.38	0.33[a]
Television	Missing	0.24	0.24	0.23	0.19[a]
Alcohol Use	Never	0.46	0.46	0.33[a]	0.34[a]
Alcohol Use	Current	0.28	0.34[b]	0.33[b]	0.43[a]
Alcohol Use	Past	0.26	0.21[b]	0.34[a]	0.23
Exercise Category	Never	0.34	0.36	0.32	0.39[c]
Exercise Category	1-3 Times/Wk.	0.22	0.26[b]	0.23	0.27[b]
Exercise Category	4+ Times/Wk.	0.43	0.37[b]	0.43	0.33[a]
Exercise Category	Missing	0.02	0.01	0.02	0.01

		Born in Stroke Belt and stayed to at least age 30		Born in Stroke Belt and moved before age 30	
		Stroke	No Stroke	Stroke	No Stroke
Smoking Status	Never	0.49	0.56[b]	0.38[a]	0.43[b]
Smoking Status	Current	0.19	0.14[a]	0.2	0.17
Smoking Status	Past	0.31	0.3	0.41[a]	0.4[a]
Smoking Status	Missing	0.01	0.01	0.00	0.00
Model 4 Health					
Body Mass Index Category	Normal	0.17	0.15	0.17	0.17
Body Mass Index Category	Obese	0.52	0.52	0.47[c]	0.47[c]
Body Mass Index Category	Overweight	0.29	0.3	0.35[b]	0.35[a]
Body Mass Index Category	Underweight	0.01	0[b]	0.01	0.01
Body Mass Index Category	Missing	0.01	0.01	0.00[c]	0.01
General health	Poor	0.10	0.03[a]	0.09	0.02[a]
General health	Fair	0.35	0.21[a]	0.33	0.17[a]
General health	Good	0.38	0.43[c]	0.38	0.42[c]
General health	Very good	0.13	0.24[a]	0.15	0.27[a]
General health	Excellent	0.04	0.09[a]	0.06	0.11[a]
General health	Missing	0.00	0.00	0.00	0.00

[a] Significant at the .001 level – compared to stroke victims born and stayed in Stroke Belt
[b] Significant at the .01 level – compared to stroke victims born and stayed in Stroke Belt
[c] Significant at the .05 level – compared to stroke victims born and stayed in Stroke Belt
[d] Significant at the .10 level – compared to stroke victims born and stayed in Stroke Belt

Source: REasons for Geographic And Racial Differences in Stroke (REGARDS) study
Sample: African-Americans Born in Stroke Belt Region (N: 6,983)

Table 5: Odds Ratios For **Model 1a** And **Model 2a** Based On Born In Stroke Belt And Stayed To At Least Age 30 Amongst African-Americans

		Model 1a	**Model 2a**
	Level	OR (95% CI)	OR (95% CI)
Model 1 Bio-Demographic			
Born in Stroke Belt and stayed to at least age 30		1.18 (1.06, 1.32)*	1.06 (0.94, 1.18)
Age		1.01 (1.01, 1.02)*	1 (0.99, 1.01)
Gender	Female	Reference	Reference
	Male	0.9 (0.8, 1)	0.81 (0.72, 0.92)*
Model 2 Socio-Economic			
Education Category	Less than HS	Omitted	Reference
	HS Grad		0.76 (0.66, 0.89)*
	Some College		0.69 (0.58, 0.81)*
	College Grad+		0.65 (0.54, 0.78)*
	Missing		3.79 (0.69, 20.86)
Income Category	< $20k	Omitted	Reference
	$20-34k		0.78 (0.67, 0.9)*
	$35-74k		0.53 (0.44, 0.63)*
	$75k+		0.40 (0.3, 0.54)*
	Refused		0.93 (0.78, 1.11)
Possess Health Insurance	No	Omitted	Reference
	Yes		1.00 (0.83, 1.2)
	Missing		1.76 (0.47, 6.65)
Relationship Status	Single	Omitted	Reference
	Married		1.15 (0.91, 1.44)
	Divorced		1.05 (0.82, 1.34)
	Widowed		1.18 (0.93, 1.51)
	Other		1.47 (1.08, 2.02)*

* Significant

Source: REasons for Geographic And Racial Differences in Stroke (REGARDS) study
Sample: African-Americans Born in Stroke Belt Region (N: 6,983)

Table 6: Odds Ratios For **Model 3a** And **Model 4a** Based On Born In Stroke Belt And Stayed To At Least Age 30 Amongst African-Americans

		Model 3a	**Model 4a**
	Level	**OR (95% CI)**	**OR (95% CI)**
Model 1 Bio-Demographic			
Born in Stroke Belt and stayed to at least age 30		1.06 (0.95, 1.19)	1.04 (0.93, 1.17)
Age		1.00 (0.99, 1.01)	1.00 (1.00, 1.01)
Gender	Female	Reference	Reference
	Male	0.81 (0.71, 0.93)*	0.80 (0.70, 0.92)*
Model 2 Socio-Economic			
Education Category	Less than HS	Reference	Reference
	HS Grad	0.81 (0.70, 0.94)*	0.88 (0.75, 1.02)
	Some College	0.74 (0.63, 0.87)*	0.84 (0.71, 0.99)*
	College Grad+	0.73 (0.60, 0.87)*	0.85 (0.71, 1.03)
	Missing	3.40 (0.61, 18.9)	3.91 (0.69, 22.2)
Income Category	< $20k	Reference	Reference
	$20-34k	0.82 (0.7, 0.95)*	0.87 (0.75, 1.02)
	$35-74k	0.57 (0.47, 0.68)*	0.65 (0.54, 0.78)*
	$75k+	0.46 (0.34, 0.61)*	0.56 (0.41, 0.75)*
	Refused	0.96 (0.8, 1.14)	0.98 (0.82, 1.18)
Possess Health Insurance	No	Reference	Reference
	Yes	1.03 (0.85, 1.23)	1.04 (0.87, 1.26)
	Missing	1.92 (0.5, 7.34)	2.13 (0.55, 8.26)
Relationship Status	Single	Reference	Reference
	Married	1.15 (0.91, 1.45)	1.19 (0.94, 1.51)
	Divorced	1.05 (0.82, 1.34)	1.11 (0.86, 1.42)
	Widowed	1.17 (0.92, 1.5)	1.24 (0.96, 1.59)
	Other	1.46 (1.06, 2)*	1.46 (1.06, 2.02)*
Model 3 Behavioral			
Hours of Television	None	Reference	Reference
	1-6 Hrs./Wk.	0.95 (0.41, 2.21)	0.9 (0.39, 2.13)
	1 Hr./Day	1.12 (0.46, 2.70)	1.05 (0.43, 2.59)
	2 Hrs./Day	0.93 (0.40, 2.15)	0.89 (0.38, 2.10)
	3 Hrs./Day	0.93 (0.40, 2.13)	0.89 (0.38, 2.08)
	4+ Hrs./Day	1.16 (0.51, 2.66)	1.07 (0.46, 2.47)
	Missing		
Alcohol Use	Never	Reference	Reference
	Current	0.8 (0.69, 0.93)*	0.82 (0.71, 0.95)*
	Past	1.18 (1.02, 1.37)*	1.13 (0.97, 1.31)

	Level	Model 3a OR (95% CI)	Model 4a OR (95% CI)
Exercise Category	Never	Reference	Reference
	1-3 Times/Wk.	0.80 (0.71, 0.92)*	0.89 (0.78, 1.02)*
	4+ Times/Wk.	0.73 (0.63, 0.84)*	0.86 (0.74, 0.99)
	Missing	1.08 (0.70, 1.67)	1.11 (0.71, 1.73)
Tobacco Use	Never	Reference	Reference
	Current	1.33 (1.13, 1.57)*	1.28 (1.08, 1.52)*
	Past	1.08 (0.95, 1.24)	1.04 (0.91, 1.19)
	Missing	1.00 (0.46, 2.16)	0.93 (0.42, 2.07)
Model 4 Health			
Body Mass Index Categories	Normal	Omitted	Reference
	Underweight		0.95 (0.81, 1.13)
	Overweight		1.04 (0.87, 1.23)
	Obese		1.2 (0.68, 2.12)
	Missing		0.6 (0.33, 1.12)
General health	Poor	Omitted	Reference
	Fair		0.56 (0.43, 0.72)*
	Good		0.33 (0.26, 0.42)*
	Very Good		0.21 (0.16, 0.28)*
	Excellent		0.19 (0.14, 0.26)*
	Missing		0.3 (0.09, 0.97)*

* significant

Source: REasons for Geographic And Racial Differences in Stroke (REGARDS) study
Sample: African-Americans Born in Stroke Belt Region (N: 6,983)

Table 7: Odds Ratios For **Model 1b** And **Model 2b** Based On Portion of Life Spent in Stroke Belt Amongst African-Americans Born in the Stroke Belt

		Model 1b	**Model 2b**
	Level	OR (95% CI)	OR (95% CI)
Model 1 Bio-Demographic			
Portion of Life Spent in Stroke Belt		1.26 (1.08, 1.48)*	1.14 (0.97, 1.34)
Age		1.02 (1.01, 1.02)*	1.00 (1.00, 1.01)
Gender	Female	Reference	Reference
	Male	0.91 (0.81, 1.02)	0.81 (0.72, 0.91)*
Model 2 Socio-Economic			
Education Category	Less than HS	Omitted	Reference
	HS Grad		0.77 (0.66, 0.89)*
	Some College		0.69 (0.58, 0.81)*
	College Grad+		0.65 (0.54, 0.77)*
	Missing		3.8 (0.69, 20.95)
Income Category	< $20k	Omitted	Reference
	$20-34k		0.78 (0.67, 0.91)*
	$35-74k		0.53 (0.44, 0.63)*
	$75k+		0.41 (0.30, 0.55)*
	Refused		0.93 (0.78, 1.11)
Possess Health Insurance	No	Omitted	Reference
	Yes		1 (0.83, 1.2)
	Missing		1.76 (0.47, 6.64)
Relationship Status	Single	Omitted	Reference
	Married		1.14 (0.90, 1.43)
	Divorced		1.05 (0.82, 1.34)
	Widowed		1.18 (0.92, 1.5)
	Other		1.47 (1.08, 2.02)*

* significant

Source: REasons for Geographic And Racial Differences in Stroke (REGARDS) study
Sample: African-Americans Born in Stroke Belt Region (N: 6,983)

Table 8: Odds Ratios For **Model 3b** And **Model 4b** Based On Portion of Life Spent in Stroke Belt Amongst African-Americans Born in the Stroke Belt

		Model 3b	**Model 4b**
	Level	**OR (95% CI)**	**OR (95% CI)**
Model 1 Bio-Demographic			
Portion of Life Spent in Stroke Belt		1.16 (0.98, 1.36)	1.12 (0.94, 1.32)
Age		1.00 (1.00, 1.01)	1.01 (1.00, 1.01)
Gender	Female	Reference	Reference
	Male	0.81 (0.71, 0.93)*	0.8 (0.70, 0.92)*
Model 2 Socio-Economic			
Education Category	Less than HS	Reference	Reference
	HS Grad	0.81 (0.70, 0.94)*	0.88 (0.75, 1.02)
	Some College	0.74 (0.63, 0.87)*	0.84 (0.71, 0.99)*
	College Grad+	0.72 (0.60, 0.87)*	0.85 (0.70, 1.03)
	Missing	3.41 (0.61, 18.97)	3.91 (0.69, 22.21)
Income Category	< $20k	Reference	Reference
	$20-34k	0.82 (0.71, 0.95)*	0.88 (0.75, 1.02)
	$35-74k	0.57 (0.48, 0.69)*	0.66 (0.54, 0.79)*
	$75k+	0.46 (0.34, 0.62)*	0.56 (0.41, 0.76)*
	Refused	0.96 (0.8, 1.14)	0.99 (0.82, 1.18)
Possess Health Insurance	Yes	Reference	Reference
	No	1.02 (0.85, 1.23)	1.04 (0.86, 1.26)
	Missing	1.92 (0.50, 7.33)	2.13 (0.55, 8.25)
Relationship Status	Single	Reference	Reference
	Married	1.05 (0.82, 1.34)	1.11 (0.86, 1.42)
	Divorced	1.14 (0.91, 1.44)	1.18 (0.94, 1.50)
	Widowed	1.46 (1.06, 2.00)	1.46 (1.05, 2.02)
	Other	1.16 (0.91, 1.49)*	1.23 (0.96, 1.58)*
Model 3 Behavioral			
Hours of Television	None	Reference	Reference
	1-6 Hrs./Wk.	0.95 (0.41, 2.21)	0.90 (0.38, 2.12)
	1 Hr./Day	1.12 (0.46, 2.70)	1.05 (0.43, 2.58)
	2 Hrs./Day	0.93 (0.40, 2.15)	0.89 (0.38, 2.10)
	3 Hrs./Day	0.93 (0.40, 2.13)	0.89 (0.38, 2.08)
	4+ Hrs./Day	1.16 (0.51, 2.66)	1.06 (0.46, 2.47)
	Missing	1.20 (0.52, 2.74)	1.12 (0.48, 2.6)
Alcohol Use	Never	Reference	Reference
	Current	0.80 (0.70, 0.93)*	0.82 (0.71, 0.95)*
	Past	1.19 (1.02, 1.38)*	1.13 (0.97, 1.32)

	Level	Model 3b OR (95% CI)	Model 4b OR (95% CI)
Exercise Category	Never	Reference	Reference
	1-3 Times/Wk.	0.80 (0.71, 0.92)*	0.89 (0.78, 1.02)
	4+ Times/Wk.	0.73 (0.63, 0.84)*	0.86 (0.74, 0.99)*
	Missing	1.08 (0.70, 1.67)	1.11 (0.71, 1.73)
Tobacco Use	Never	Reference	Reference
	Current	1.34 (1.13, 1.58)*	1.28 (1.08, 1.52)*
	Past	1.08 (0.95, 1.24)	1.04 (0.91, 1.19)
	Missing	0.99 (0.46, 2.16)	0.93 (0.42, 2.07)
Model 4 Health			
Body Mass Index Categories	Normal	Omitted	Reference
	Underweight		0.96 (0.81, 1.13)
	Overweight		1.04 (0.88, 1.23)
	Obese		1.19 (0.67, 2.11)
	Missing		0.61 (0.33, 1.12)
General health	Poor	Omitted	Reference
	Fair		0.56 (0.43, 0.72)*
	Good		0.33 (0.26, 0.42)*
	Very Good		0.21 (0.16, 0.28)*
	Excellent		0.19 (0.14, 0.26)*
	Missing		0.29 (0.09, 0.96)*

* significant

Source: REasons for Geographic And Racial Differences in Stroke (REGARDS) study
Sample: African-Americans Born in Stroke Belt Region (N: 6,983)

Table 9: Odds Ratios For **Model 1c** And **Model 2c** Based On Age One Left Stroke Belt Amongst African-Americans Born in the Stroke Belt

		Model 1c	**Model 2c**
	Level	OR (95% CI)	OR (95% CI)
Model 1 Bio-Demographic			
Age One Left Stroke Belt	Stayed	Reference	Reference
	Before Age 5	0.98 (0.82, 1.16)	1.04 (0.87, 1.24)
	Age 5 – 18	0.93 (0.80, 1.09)	1 (0.85, 1.17)
	Age 18 – 30	0.75 (0.65, 0.86)*	0.87 (0.76, 1.00)
Age		1.01 (1.01, 1.02)*	1 (1.00, 1.01)
Gender	Female	Reference	Reference
	Male	0.89 (0.79, 0.99)	0.80 (0.71, 0.91)*
Model 2 Socio-Economic			
Education Category	Less than HS	Omitted	Reference
	HS Grad		0.77 (0.66, 0.89)*
	Some College		0.69 (0.59, 0.82)*
	College Grad+		0.66 (0.55, 0.79)*
	Missing		3.69 (0.67, 20.30)
Income Category	< $20k	Omitted	Reference
	$20-34k		0.78 (0.67, 0.90)*
	$35-74k		0.53 (0.45, 0.64)*
	$75k+		0.40 (0.30, 0.54)*
	Refused		0.93 (0.78, 1.11)
Possess Health Insurance	No	Omitted	Reference
	Yes		1.01 (0.84, 1.21)
	Missing		1.76 (0.47, 6.64)
Relationship Status	Single	Omitted	Reference
	Married		1.15 (0.91, 1.45)
	Divorced		1.05 (0.82, 1.34)
	Widowed		1.18 (0.93, 1.51)
	Other		1.47 (1.07, 2.02)*

* significant

Source: REasons for Geographic And Racial Differences in Stroke (REGARDS) study
Sample: African-Americans Born in Stroke Belt Region (N: 6,983)

Table 10: Odds Ratios For **Model 3c** And **Model 4c** Based On Age One Left Stroke Belt Amongst African-Americans Born in the Stroke Belt

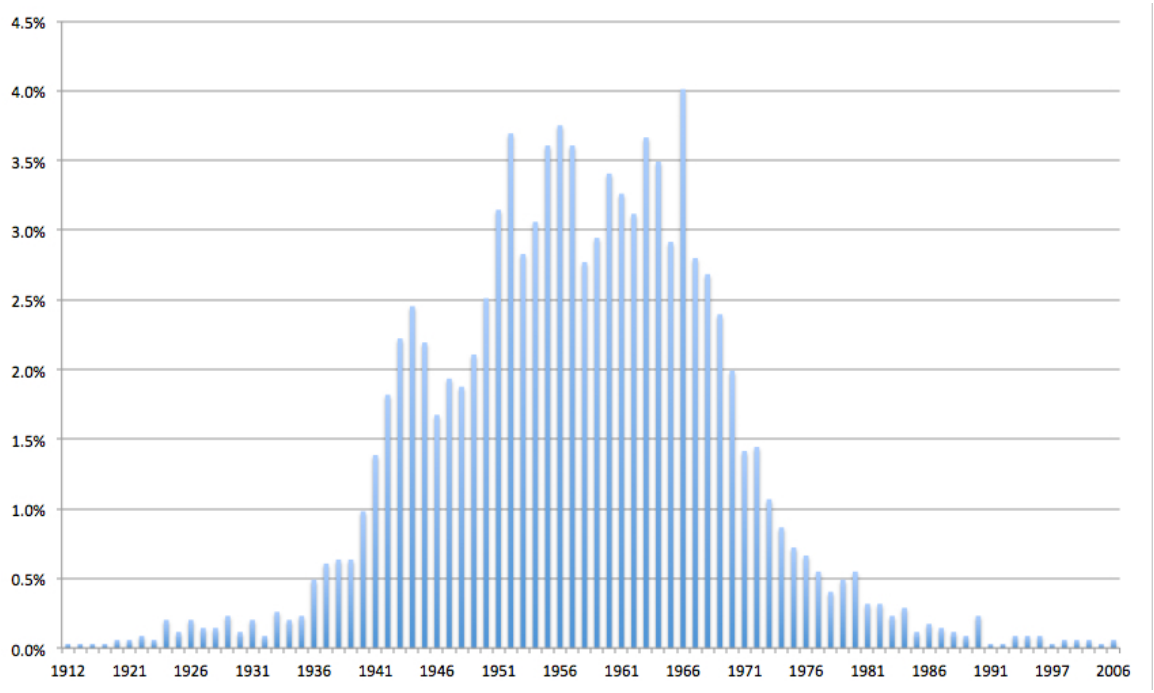
		Model 3c	**Model 4c**
	Level	**OR (95% CI)**	**OR (95% CI)**
Model 1 Bio-Demographic			
Age One Left Stroke Belt	Stayed	Reference	Reference
	Before Age 5	1.02 (0.86, 1.22)	1.01 (0.84, 1.21)
	Age 5 – 18	0.99 (0.84, 1.17)	1.01 (0.86, 1.2)
	Age 18 – 30	0.88 (0.77, 1.01)	0.91 (0.79, 1.04)
Age		1.00 (1.00, 1.01)	1.01 (1, 1.01)
Gender	Female	Reference	Reference
	Male	0.81 (0.71, 0.92)*	0.8 (0.69, 0.91)*
Model 2 Socio-Economic			
Education Category	Less than HS	Reference	Reference
	HS Grad	0.81 (0.70, 0.95)*	0.88 (0.75, 1.03)
	Some College	0.74 (0.63, 0.88)*	0.84 (0.71, 1.00)*
	College Grad+	0.73 (0.61, 0.88)*	0.86 (0.71, 1.04)
	Missing	3.32 (0.6, 18.42)	3.82 (0.67, 21.7)
Income Category	< $20k	Reference	Reference
	$20-34k	0.82 (0.71, 0.95)*	0.87 (0.75, 1.02)
	$35-74k	0.57 (0.48, 0.69)*	0.66 (0.54, 0.79)*
	$75k+	0.46 (0.34, 0.62)*	0.56 (0.41, 0.75)*
	Refused	0.96 (0.80, 1.14)	0.98 (0.82, 1.18)
Possess Health Insurance	Yes	Reference	Reference
	No	1.03 (0.86, 1.24)	1.05 (0.87, 1.26)
	Missing	1.92 (0.50, 7.31)	2.13 (0.55, 8.27)
Relationship Status	Single	Reference	Reference
	Married	1.16 (0.92, 1.46)	1.20 (0.94, 1.51)
	Divorced	1.04 (0.82, 1.34)	1.10 (0.86, 1.42)
	Widowed	1.17 (0.92, 1.50)	1.24 (0.96, 1.59)
	Other	1.46 (1.06, 2.00)*	1.46 (1.06, 2.02)*
Model 3 Behavioral			
Hours of Television	None	Reference	Reference
	1-6 Hrs./Wk.	0.96 (0.41, 2.22)	0.91 (0.39, 2.13)
	1 Hr./Day	1.12 (0.46, 2.72)	1.06 (0.43, 2.6)
	2 Hrs./Day	0.93 (0.4, 2.16)	0.90 (0.38, 2.11)
	3 Hrs./Day	0.93 (0.41, 2.15)	0.90 (0.39, 2.10)
	4+ Hrs./Day	1.17 (0.51, 2.68)	1.07 (0.46, 2.49)
	Missing	1.20 (0.52, 2.75)	1.12 (0.48, 2.62)

	Level	Model 3c OR (95% CI)	Model 4c OR (95% CI)
Alcohol Use	Never	Reference	Reference
	Current	0.80 (0.69, 0.93)*	0.82 (0.71, 0.95)*
	Past	1.18 (1.02, 1.37)*	1.13 (0.97, 1.31)
Exercise Category	Never	Reference	Reference
	1-3 Times/Wk.	0.81 (0.71, 0.92)*	0.89 (0.78, 1.02)
	4+ Times/Wk.	0.73 (0.63, 0.84)*	0.86 (0.74, 1)*
	Missing	1.08 (0.7, 1.66)	1.11 (0.71, 1.72)
Tobacco Use	Never	Reference	Reference
	Current	1.33 (1.13, 1.57)*	1.28 (1.08, 1.51)*
	Past	1.08 (0.94, 1.23)	1.04 (0.9, 1.19)
	Missing	1.00 (0.46, 2.17)	0.94 (0.42, 2.08)
Model 4 Health			
Body Mass Index Categories	Normal	Omitted	Reference
	Underweight		0.95 (0.81, 1.13)
	Overweight		1.04 (0.87, 1.23)
	Obese		1.21 (0.68, 2.13)
	Missing		0.60 (0.32, 1.11)
General health	Poor	Omitted	Reference
	Fair		0.56 (0.44, 0.72)*
	Good		0.33 (0.26, 0.42)*
	Very Good		0.21 (0.16, 0.28)*
	Excellent		0.19 (0.14, 0.26)*
	Missing		0.30 (0.09, 0.99)*

* significant

Source: REasons for Geographic And Racial Differences in Stroke (REGARDS) study
Sample: African-Americans Born in Stroke Belt Region (N: 6,983)

Graph 1: Distribution of Migrations Out of the Stroke Belt Amongst African-Americans Born in the Stroke Belt Amongst Study Population



APPENDIX

General Etiology and Prevention of Strokes

Established Risk Factors

ATRIAL FIBRILLATION

Atrial Fibrillation, a heart condition that causes stagnation of blood flow within the left atrial appendage, is an independent risk factor for stroke with a three-fold to five-fold higher risk. When blood stagnates in the heart, it can coagulate and clot forming a thrombus or embolus. A thrombus or embolus is a clot of blood cells that block blood flow in a blood vessel, the distinction in name referring to where the blockage occurs in the body. If this clot is to travel to and block blood flow in the brain, a stroke occurs. Studies indicate that about 30% of strokes are attributable to atrial fibrillation in patients 80 years or older. Atrial Fibrillation not only increases the risk of stroke morbidity but also increases the risk of stroke mortality by 1.5 to 1.9 times (Grysiewicz et al., 2008, p. 877).

DIABETES MELLITUS

Diabetes Mellitus, commonly referred to as simply diabetes, has an immense impact on one's stroke risk. Estimates of the impact vary from a relative risk of stroke of about 2 (Markus, 2004, p. 57) to a staggering six-fold higher risk of stroke (Grysiewicz et al., 2008, p. 876). A study of about 14,000 persons with coronary heart disease found that impaired glucose tolerance (an indication of diabetes) was associated with higher stroke risk. Overall, studies have estimated "nearly 40% of all ischemic strokes can be attributed to the effects of diabetes either alone or in combination with hypertension" (Grysiewicz et al., 2008, p. 876).

DEPRESSION

An Australian study by Simons et al., which followed 2805 people prospectively over 8 years, demonstrated that depression score was an indicator of stroke risk. Depression scores in the upper third of the scale had a 41% higher stroke risk than did those in the bottom third (Joubert et al., 2007, p. 71). Simons et al. also detected an interaction between depression's impact on stroke risk and age. In persons aged 70 years and older, high depression score led to a relative risk of stroke of 1.83. However, in persons aged 60-69 years, the association between depression score and stroke risk was not significant (Joubert et al., 2007, p. 72).

A later, related study, Simonsick et al., proposed that the link between depression score and stroke risk was due to poor blood pressure control caused by depression. However, despite this hypothesis, they were unable to find a consistent association between blood pressure control and depression. Researchers May et al. instead argued that depression leads to the adoption of unhealthy behaviors: smoking, poor diet, or lack of exercise. Jonas and Mussolino later disproved this hypothesis by demonstrating a significant association between depression and stroke remained, even after adjusting for low levels of exercise and other unhealthy, 'vice' behaviors. As of yet, the mechanism by which depression truly modifies stroke risk is unknown (Joubert et al., 2007, p. 73).

DIET

A patient's diet plays a direct and indirect role in one's stroke risk. Consumption of fruits and vegetables, the classic guidelines of a healthy diet, show a strong relationship with ischemic stroke risk. This relationship is especially true with cruciferous vegetables, green leafy vegetables, and citrus fruits, where a 6% reduction in risk for ischemic stroke is seen with each incremental serving increase per day

(Grysiewicz et al., 2008, p. 879). Cholesterol, a dietary component of many foods, also modifies one's stroke risk. Diets high in saturated fat can increase cholesterol in one's body. When levels of cholesterol pass a threshold of ideal levels (total cholesterol <200; low-density lipoprotein (LDL) <100 and high-density lipoprotein (HDL) >40), a patient's risk of stroke increases (Bergman et al., 2012, p. 37). Increases in total cholesterol and LDL cholesterol are also a strong risk factor for ischemic heart disease, which in turn further increases stroke risk (Markus, 2004, p. 57).

In persons already overweight, dietary intake plays an especially critical role in the modification of stroke risk. Higher sodium intake for an overweight individual is associated with an 89% increased risk for stroke mortality (Grysiewicz et al., 2008, p. 879). This association results from the increase in hypertensive symptoms as a result of increased dietary sodium (Bergman et al., 2012, p. 37). Conversely, an increase of 10 mmol of daily potassium consumption is associated with a 40% drop in stroke mortality, even when controlling for other cardiovascular risk factors. Increasing potassium consumptions decreases mean systolic and diastolic blood pressures, addressing hypertension and hypertension's subsequent impact on stroke risk (Grysiewicz et al., 2008, p. 879).

Family History and Genetics

Family history, which has long been considered a factor in stroke risk, is gaining new research interest following the sequencing of the human genome and the proliferation of personal genetic sequencing. First, though, consider the observed influence of family history on stroke risk. Grysiewicz et al. states, "parental history of stroke, TIA, or myocardial infarction is associated with 1.4 to 3.3 fold increased risk for stroke" (2008, p. 875). Baird, citing the variation in study finds and some degree of

methodological weaknesses, argues that a family history of stroke increases a subject's stroke risk by 2- to 3-fold. Using three generations of data, the Framingham study found a parental history of stroke was associated with an approximately double risk of stroke. Stratifying paternal and maternal stroke history, the Framingham study found a paternal history of stroke to give a relative risk of stroke of 2.4 and a maternal history with a relative risk of 1.4 (Baird, 2010, p. 246).

Currently, the contribution of genetics on stroke risk appears to be small due to the fact that few genetic factors have been identified for stroke. However, as more research is conducted on the human genome, it is highly likely that there will be a number of discoveries of biomarkers for stroke (Baird, 2010, p. 245). According to Baird, modifiable risk factors account for approximately 60% of the population-attributable risk (PAR) for stroke. Baird argues, "the mechanisms for over 30% of ischemic strokes are not known, even after extensive workup" (2010, p. 245). Assuming 60% of PAR for stroke is in fact accounted for by modifiable risk factors, 40% of the PAR for stroke has yet to be accounted for. As further research is conducted into the human genome, researchers will look to explain where the other 40% of PAR for stroke comes from and potentially how to address that risk. Aside from this 40% unaccounted for, genetics could also play a role in the other major stroke risk factors themselves. Conditions such as hypertension, atrial fibrillation, depression, and other vascular disease may all have genetic links. Thus, genetics could contribute far more to stroke risk than may be initially apparent (Baird, 2010, p. 248).

HORMONE THERAPIES

Hormone therapies have demonstrated themselves to be important modifiers of stroke risk. While many hormone therapies exist, these analyses focus on hormone

replacement therapies used in postmenopausal women and oral contraceptive pills. Initially, hormone replacement therapy was believed to have a positive impact on vascular disease risk, with observational trials reporting a possible beneficial impact of hormone replacement therapy. However, the Women's Health Initiative trial failed to link hormone replacement therapy with any reduction in coronary heart disease. Instead, the study showed an increase in risk of both CHD and heart attack, bearing in mind that a larger proportion of the study population was aged over 70, and therefore already at increased risk for CHD and heart attack. The study, along with the United Kingdom General Practice Research Database (GPRD) showed a higher stroke risk following hormone replacement therapy in all ages (Katsiki, Ntaios, & Vemmos, 2011, p. 240). Grysiewicz et al. notes that a clinical trial demonstrated that women who receive estrogen replacement therapy (a type of hormone replacement therapy) had a relative risk for stroke mortality of 2.9 and a worsening of outcomes even if the stroke did not prove fatal (Grysiewicz et al., 2008, p. 880). Oral contraceptive pills also modify stroke risk, particularly estrogen-containing preparations (Markus, 2004, p. 58) This risk is further magnified when the patient is positive for certain genetic markers, though this genetic link has yet to be exhaustively studied (Baird, 2010, p. 246).

HYPERTENSION

Hypertension, commonly referred to as high blood pressure, is perhaps the most significant modifiable risk factor for both ischemic and hemorrhagic stroke. As expected, the treatment of hypertension is one of the most effective tools in reducing first-ever and recurrent stroke incidence.(Bejot et al., 2007, p. 85). Lim, Kim, and Hong report that hypertension along with other risk factors such as current smoking, abdominal obesity,

diet and physical activity account for over 80% of strokes, with hypertension taking a larger share of this association than other components (Lim et al., 2013, p. 146).

Observational studies find that at systolic blood pressures as low as 115 mm Hg and upwards, risk for death from either ischemic heart disease (IHD) or stroke continuously and consistently increases. Beyond this threshold systolic blood pressure value of 115 mm Hg, the risk of mortality due to stroke or heart disease doubles with every 20 mm Hg increase (Grysiewicz et al., 2008, p. 876). Diastolic blood pressure also serves as an indication of stroke risk. Markus describes the relationship between diastolic blood pressure and stroke as log linear throughout the normal range of blood pressures. Also, there is no value at which blood pressures lower experience stable stroke risk. Markus also notes that for ever 7.5 mm Hg increase in diastolic blood pressure, the risk of stroke nearly doubles (Markus, 2004, p. 57). These increases in stroke risk due to systolic and diastolic blood pressure levels are independent of other risk factors for stroke and is strongly supported by randomized control trials showing that stroke can be prevented by lowering blood pressure, even at 'normal' levels (Markus, 2004, p. 57). Hypertension further increases stroke risk by accelerating the development of atherosclerosis, a condition that leads to the formation of fatty plaque deposits in blood vessels. These deposits ultimately leading to an increased number of blood vessel blockages, further increasing blood pressure (Grysiewicz et al., 2008, p. 876).

INFECTION

Infection, like hypertension, has long been considered a cause of atherosclerosis, a condition leading to fatty plaque deposits in blood vessels. Infection in the body may also increase cytokine expression, causing pro-coagulant effects in the blood stream. This association between infection and stroke risk extends to both acute bacterial or viral

infections, especially increasing risk for ischemic stroke. The effect of infection on stroke weakens over time after the infection, with the most profound effect on stroke risk coming from infections taking place within a week before a stroke event. In addition, a number of other infectious agents such as herpes and cytomegalovirus have been implicated in specially modifying stroke risk (Grysiewicz et al., 2008, p. 882).

VASCULAR DISEASE ELSEWHERE

Patients who currently have or have had vascular disease elsewhere in their body are at significantly higher risk for stroke. A study by Grysiewicz et al. found that in the first five years following a heart attack, a person has an 8.1% chance of an ischemic stroke (2008, p. 878). In many ways, this association is to be expected. Many of the conditions that directly or indirectly affect stroke risk also modify heart attack risk or other vascular diseases. In many ways, vascular disease elsewhere is an indication of conditions that lead to a higher stroke risk rather than the cause of higher stroke risk itself. While not a direct cause, vascular disease elsewhere is useful as a mechanism to prompt intervention to reduce stroke risk (Markus, 2004, p. 58).

Other Possible Factors

Citing that existing knowledge on stroke risk factors cannot explain the totality of stroke risk, a number of researchers have sought to identify new risk factors and associations for stroke risk and incidence. Noted below are a handful of studies that tested proverbial knowledge about stroke risk factors, but did not find significant associations.

TAP WATER HARDNESS

Health officials have long been concerned about the impact of trace minerals in drinking water on stroke risk. The logic went that tap water hardness, an indication of

levels of dissolved calcium carbonate ($CaCO_3$), would introduce the substance into one's blood stream, which could in turn crystalize and create blockages in blood vessels. A study by Leurs, Shouten, Mons, Goldbohm, and Brant sought to answer this exact question, testing the relationship between water hardness and mortality due to ischemic heart disease or stroke. According to the study, there was no relationship between tap water hardness and either IHD mortality or stroke mortality. Further, there was no significant association between magnesium levels in the tap water and stroke mortality (2010, p. 414).

TEA CONSUMPTION

Some evidence suggests that tea consumption also plays a role in the modification of stroke risk. Larsson, Virtamo, and Wolk demonstrated that high tea consumption (consumption of four cups or more of tea per day) was associated with a statistically significant lower risk of total stroke. Both men and women who consumed high amounts of tea each day had a 21% reduced risk of stroke when compared to persons who never consume tea. While the results were significant, the findings are of little relevance to stroke prevention overall. Consuming four or more cups to tea each day may exceed what typical tea drinkers consume. Further, consumption at this level is only significant when compared to people who never consume any tea. The study concedes that consuming only one to three cups of tea per day was not associated with any significant modification in stroke risk. Further muddling significance, the study authors note, "the association seemed to be confined to cerebral [ischemic stroke] and intracerebral [hemorrhagic stroke], but results for stroke types were not significant" (Larsson, Virtamo, & Wolk, 2013, p. 159). While the study adjusted for the impact of fresh red meat, processed meat, fish, and chocolate consumption, there are likely further confounders

causing the association between high tea consumption and stroke risk modification. Likely, persons who consume a large amount of tea are more health conscious and therefore have lower stroke risks to begin with. Without any indication as to the mechanism by which tea could reduce stroke risk or any explanation as to the lack of dose response between cups of tea consumed and risk reduced, this study fails to be very persuading. However, the association is certainly one of interest and would benefit from additional study (Larsson et al., 2013, p. 158).

Prevention of Strokes

The primary and most apparent way to prevent stroke morbidity and mortality is the modification of modifiable variables (Bergman et al., 2012, p. 2). While many characteristics that increase ones risk for stroke such as age and gender cannot be changed, there are a host of medical and behavioral variables whose modification can greatly impact one's risk of suffering a stroke. According to Joubert, Cumming, and McLean, "if traditional vascular risk factors are correctly managed, up to 75% of cerebrovascular disease can be prevented" (2007, p. 71). To do so, patients should focus on a host of medical and behavioral interventions. The Heart Protection Study demonstrated that the use of simvastatin, a cholesterol lowering statin drug, at the 40mg indication reduces the risk of stroke and other vascular events amongst high-risk patients. High-risk in this case refers to patients with a previous ischemic stroke, coronary or peripheral vascular disease, or diabetes. The study found this association to hold even amongst patients with normal levels of cholesterol (LDL < 3.5 mmol/liter). Amongst patients with pre-existing cerebrovascular disease, simvastatin reduced the risk of vascular events by 20% over a five year period, with no increased risk of a hemorrhagic stroke (Pendlebury & Rothwell, 2004, p. 66)

Blood pressure reduction is also an influential component of stroke risk reduction. The PROGRESS study showed that the use of Perindopril and Indapamide, both blood pressure drugs, after a first stroke reduces the risk of subsequent strokes, even amongst patients whose blood pressure is deemed normal (Pendlebury & Rothwell, 2004, p. 66).

Antiplatelet drugs, medicines that prevent blood cells from sticking together, are a useful tool for the reduction of recurrent stroke and overall vascular death. A study over three years found that the treatment of one thousand patients with antiplatelet drugs resulted in 36 fewer serious vascular event and 15 fewer deaths per thousand patients. Many of the trial patients were given aspirin, but some were given new antiplatelet agents such as Clopidogrel and Dipyridamole. The extra benefit of these newer drugs over aspirin was not able to justify the increase in cost. However, there is interest in combination antiplatelet therapy, using aspirin in conjunction with one of these two advanced drugs. For persons at high risk of recurrent ischemic events, the use of aspirin and Dipyridamole versus aspirin alone led to a relative risk reduction for stroke of 30% (9.5% versus 12.5%). These same effect was not found to be true of Clopidogrel and aspirin versus Clopidogrel alone (Pendlebury & Rothwell, 2004, p. 66).

Anticoagulation medicines are also an important method for preventing stroke in patients in sinus rhythm (normal heart function). Anticoagulation medicines work by preventing the patients blood from clotting and potentially creating embolus, clusters of clotted blood cells that block blood flow in veins and arteries. While anticoagulants can help prevent ischemic strokes, anticoagulants such as the drug warfarin should be used with caution as preventing coagulation can actually increase the harm caused by a

hemorrhagic stroke, as bleeding will continue and the body is unable to stop the blood loss in the brain (Pendlebury & Rothwell, 2004, p. 66).

In addition medical interventions, lifestyle changes can have a profound impact on one's stroke risk. While medical interventions serve as a powerful tool in the fight against strokes, their gains are often undone by poor health decisions undertaken by a patient. A number of observational studies indicate that lifestyle modification such as smoking cessation, regular physical activity, healthy diet, and abandonment of heavy alcohol consumption all help reduce one's risk of stroke (Neuhauser, 20, p. 2121). Additionally, for those female patients deemed at high risk of stroke, cessation of oral contraceptive medicine can further reduce stroke risk. In essence, while medical interventions are an important component of stroke risk management, persons at high stroke risk should also work with their physicians on behavior modifications to further reduce his or her stroke risk.