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Impact of area social predictors of health on Black-White disparities in stroke mortality

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Impact of Area Social Predictors of Health on Black-White Disparities in Stroke Mortality

by

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ABSTRACT

This dissertation investigated the area social predictors of health (ASPoH) and Black-White disparities in stroke mortality relationship. Utilizing stroke mortality data obtained from the Florida Department of Health for years 1998-2002, and social and economic data obtained from the year 2000 Census of Population, this study examined the effect of resource availability at the census tract level on Black-White disparities in stroke mortality. The influence of social class on Black-White disparities in stroke mortality and effect modification by social class of the association between Black-White disparities and ASPoH variables was also investigated. Principal component analysis produced four ASPoH scores from economic and social measures. Multiple regression analysis assessed the predictive ability of these ASPoH variables on Black-White disparities.

Increases in the female Black-White ratio were significantly associated with increases in the magnitude of the ASPoH-1 and ASPoH-2 variables. When regression analyses were restricted (in terms of population count minimums) to a subset of census tracts, increases in the ASPoH-1 and ASPoH-2 variables were significantly associated with increases in all Black-White disparity measures for both males and females.

Assessment of the influence of social class on Black-White disparities in stroke

mortality was only feasible at the state level due to a lack of data at the census tract level. With the exception of the 65+ years age-group, Black males and females experienced higher age-group specific stroke mortality rates across each of the social class groups. Inconsistent with previous research findings, Black residents who attained a high school degree had the highest stroke death rates compared to all other educational attainment groups.

In the assessment of social class as a potential effect modifier, the study hypothesis stated that the ASPoH measures would have the greatest impact on those residents in the lowest social class category. This predicted effect was only supported when the Male Black-White ratio disparity score was examined.

Study findings support the conjecture that unknown and unmeasured processes influence the association between area social predictors and stroke mortality for Black Floridians. Identification of modifiable societal characteristics may be the key to unlocking the foundation of disparities in health outcomes.

Chapter One

Introduction

Quantification of neighborhood characteristics has become an important, and vastly utilized, research tool in determining influences on 'small geographic area' morbidity and mortality rates. The use of this tool is rendered possible by the availability of census tract population and social and economic data which are typically used to construct measures of neighborhood characteristics. Utilization of census data has proven to be an important resource for elucidating relationships between socioeconomic position and health outcomes for the U.S. as well as for the neighborhood level (census tract).[1,2] Furthermore, public health research has shown differential findings by race between socioeconomic status and health outcomes;[3] consequently, the role of neighborhood socioeconomic context in the contribution and exacerbation of racial inequalities in morbidity and mortality presents as the next logical question for public health researchers.

The challenge posed by this type of research is the identification and accurate definition of the aspects of the neighborhoods which are influential in health outcomes and opportunities for health promoting behaviors. Social and public health researchers have suggested using a framework of universal human needs as a basis for thinking about how places may influence health, and recommend testing of hypotheses about specific chains of causation that might link place of residence with health outcomes.[4] Research

on the resources that humans need in order to live a healthy life and the importance of the geographic distribution of these available resources as it relates to the distribution of health is needed.[4]

Stroke mortality, an adverse health outcome with specific socioeconomic status and geographical distributions, is an ideal health outcome that can be utilized when investigating the association between available neighborhood resources and the differential distribution of health outcomes. There are many critical aspects of understanding stroke outcomes. Various factors have been shown to be significant contributors to stroke mortality rates.[5,6] Understanding the many mechanisms leading to stroke mortality, determining the contributors to stroke mortality and, maybe most importantly, reconciling the relationship between these contributors is paramount in lessening the burden of this disease in our society. Deciphering these mechanisms may also lead to a better understanding of why certain groups experience a heavier disease burden.

Factors consistently identified as influential in stroke mortality include: ethnicity, age, gender, lower socioeconomic status (as defined by occupation only), social class (as defined by occupation and/ or education), and health risk behaviors (smoking, drinking, physical activity, diet), hypertension, diabetes, end-stage renal disease, and obesity.[7,8] In addition, disparities in stroke mortality, as well as other morbidities and causes of mortality, exist between specific ethnic and social subgroups within our society. The health disparities persist even after controlling for the majority of the aforementioned contributors.[7,8] Therefore, the potential relevant relationships between socioeconomic factors (education and income) and lifestyle, between lifestyle and ethnicity/race, as well

as the relationship between ethnicity/race and socioeconomic factors, must be taken into consideration as possible mediators in the pathway leading to stroke mortality. Reducing the prevalence of health risk behaviors, reducing the proportion of those living in poverty, and increased education would more than likely reduce the prevalence of stroke mortality in at risk subpopulations, as well as various other undesirable health outcomes. However, it is the premise of this study that differences in stroke mortality are due to a wider array of factors, many of which may be specific to contextual social and economic characteristics of small geographic areas. This study will explore the role of social and economic characteristics in racial disparities in stroke mortality (Refer to references 7 and 8 for rate difference between Blacks and Whites. Race specific rates are also presented in the following chapter). The specific contextual characteristics to be addressed in this study are 'area social predictors of health' (ASPoH) status at the census tract level. The ASPoH index will be fully developed within the methods chapter of this document.

Proposed Research Questions and Hypotheses

Research Question 1: Are Black-White disparities in stroke mortality elevated in those areas of low ASPoH status?

Hypothesis: Black-White disparities in stroke mortality will be greatest at lower levels of ASPoH.

Research Question 2: Are higher levels of Black-White disparities in stroke mortality associated with low levels of social class?

Hypothesis: Black-White disparities in stroke mortality will be greatest for those in the lowest social class group.

Research Question 3: Is there effect modification by social class of the ASPoH status and

Black-White disparities in stoke mortality relationship?

> Hypothesis: ASPoH status will have a greater impact on Black-White disparities in stroke mortality for the lower social class groups. However, the association between disparities and ASPoH status will persist after controlling for individual social class (educational attainment).

Study Purpose/Rationale

Stroke is the third leading cause of death after heart disease and cancer in the US.[9] Stroke mortality rates for Black Americans are substantially higher than those of White Americans.[9] Analogous to findings for other adverse health outcomes, there is consistent evidence of an unequal distribution of stroke deaths across social class. Generally, those individuals making up the higher social class group experience better health outcomes.[10] When these associations are examined separately for Whites and Blacks, worse outcomes are typically observed for Blacks at any given level of social class.[11] Similar results have been found for the association between socioeconomic status and stroke mortality;[12] not a surprising finding given that the same or related indices of social class are used as indices of socioeconomic status. Results from these studies have varied depending on the level of socioeconomic status being investigated (individual or community SES). Consistent associations are seen at the individual level;[13,14] however, results are mixed when the effect of SES is investigated at the community level.[15,16]

In an attempt to further our understanding of these disparities, current investigations now focus on aspects of the community in which we live as a contributing factor in these continued disparities. Whether these communities are created out of

natural growth or if the development of a community is determined by government acts/laws must be taken into consideration. Of concern is the lawfulness and appropriateness of land use zoning practices. Whether or not zoning practices lead to segregation by race and economic circumstance is the present question. If these practices lead to homogeneous groups of people residing in less healthy areas due to their economic circumstances and minority status, attention must be given to these issues in order to attempt to understand their role in geographic and racial health disparities. Research on zoning laws and practices reflect that harmful land uses tend to be disproportionately concentrated in poor and industrial neighborhoods which tend to have larger minority populations.[17,18] Authors have highlighted the injustice of these zoning practices and hypothesized about the underlying belief systems (overt or covert racism, putting economic profits over the health of people, or benign neglect) that lead to 'disproportionate risk.'[17,18]

Also of concern in proposing a causal pathway leading from area economic and social measures to racial disparities in stroke mortality is the potential influence of residential segregation. Residential segregation refers to the physical separation of the races in residential contexts. Residential segregation can lead to the formation of radically different environments for the segregated group and the rest of the population. The possibility exists that this study will capture elements of residential segregation at the census tract level. If this is the result, the influence of residential segregation on the study outcomes must be considered. At issue would be the question of whether those census tracts with greatest impact on study results are those tracts with relatively large Black populations. These particular census tracts may have greater impact on study

results because they contain sufficient numbers of Black residents allowing for the calculation of stroke mortality measures. The next consideration would be whether those census tracts with sufficient Black population exist as a result of residential segregation. If residential segregation processes are influential in determining racial residential groupings within the census tracts, this may affect the probability that people living in the same area (census tract) may not have access to the same amount and quality of resources. Unspoken norms and/or rules of behavior among residents within small geographic area may dictate the one carries out his/her daily activities within a restricted area within the census tract. Residents, who travel beyond the bounds of their 'designated' area, may experience discomfort within these locations. Therefore, residents may remain within their comfort zone and not take advantage of all seemingly available resources within their neighborhood.

Research investigating possible health effects of residential segregation show that even after controlling for important risk factors (such as education, income and occupational status), segregation may have a statistically significant effect on various health outcomes.[19,20] Residential segregation is to proposed influence racial disparities in health because of its capacity to capture some of the effects of racism. Researchers propose the community level effects of residential segregation as one potential reason for the persistence of racial differences in health status even after controlling for individual variations in socioeconomic status.[21] Residents of disadvantaged neighborhoods have a higher incidence of heart disease than people who do not reside in disadvantaged neighborhoods.[22] This effect persists after adjustment for education, income, occupational status, and biomedical and behavioral risk factors for coronary heart

disease. Residential segregation has also proven to be a significant predictor of mortality among adult African Americans[23] as well as among Black infants.[24]

Environmental factors, and their differential concentration within certain geographic areas, are closely related to personal behavior and lifestyle.[25] The need to "explicitly acknowledge the intimate connections between the social and economic conditions people live under and their biobehavioral risk factor profile" has been expressed.[25] How one interacts in his/her community (social interaction, participation, cohesion, and social networks) may have influences on risk factor exposure probability. This may be due to the presence of community characteristics that support or influence the probability of exposure to factors leading to adverse health outcomes. The "connections between these adverse {environmental} conditions and the adaptive responses affected communities must often make to them" needs further research.[25]

Investigation of economic aspects of the community (such as income inequality) in an attempt to demonstrate how relative deprivation may influence or increase the prevalence of adverse health behaviors (smoking, alcohol, sedentariness, unhealthy diet) have been undertaken.[26,27] The current study aims to continue this research by investigating the relationship between availability of and opportunity to obtain economic resources (captured by a measure of area social predictors of health) and racial disparities in stroke mortality. The construction of the 'Area Social Predictors of Health' measure incorporated dimensions of economy, employment, education, and housing conditions. Controlling for census tract level social position indicators through stratification is the technique utilized to analytically demonstrate an effect of the area in which one lives on health; therefore, potentially demonstrating an "independent" effect of ASPoH status.

Proposed Pathway: ASPoH and Black-White Disparities in Stroke Mortality

It is proposed that through the following theoretical causal pathway, resource availability, which is hypothesized to determine risk factor exposure potential, significantly contributes to racial disparities in stroke mortality.

A relationship between ASPoH status and Black-White disparities in stroke mortality is proposed. The proposed model depicts the effect of ASPoH status on the local environments contributing to these disparities (see Figure 1). In those areas of low ASPoH status, a concentration of Blacks living in poverty is expected. According to the literature, the quality of housing in these areas will be of low market value and more likely to be overcrowded.[28] Researchers have suggested that financial institutions determine the location of their establishments based on the credit worthiness of the local residents;[4] therefore, the theoretical pathway predicts that the availability of banks and other investment institutions will be in short supply in lower ASPoH areas. The commitment of monetary resources to the physical maintenance of these areas will be lacking, possibly due to the 'zoning' laws which influence the distribution of community maintenance funds.

It is believed that those residents living in lower ASPoH areas will have reduced access to private transportation and increased access to public transportation services.[29] Those residents without private transportation may be less likely to possess resources that permit travel beyond their immediate residence. This restriction would thereby limit their access to the full array of resources within the census tract.

The model presumes reduced availability of and/or access to medical care for residents of lower ASPoH areas. Among individuals with moderate or low incomes,

those without health insurance generally have less access to medical care than those with coverage.[30] The lack of annual physical exams may contribute to poor health, due to the inability to pay out-of-pocket medical expenses. Without periodic physical exams, early detection of disease may not be possible. Also, the availability of important information regarding preventive strategies for diseases in which the uninsured may be at increased risk will be lacking.[31] Additionally, African Americans are more likely to be uninsured than White Americans.[32] The aforementioned evidence coupled with the evidence that Black Americans are more likely to reside in more disadvantaged areas lends strength to the prediction that Black-White disparities in stroke mortality will be more pervasive in the lower ASPoH neighborhoods.

In lower ASPoH areas, the model theorizes reduced availability of emergency medical care facilities that are properly prepared to treat stroke victims. Investigations into the role of socioeconomic status on access to health services after stroke, found that patients in lower income categories were less likely to have access to hospitals with neurologists and imagery equipment necessary for the diagnosis and treatment of stroke.[33] Findings additionally suggest that the disparity in access to those hospitals properly staffed and equipped for stroke treatment is related to the distribution of specialized resources in more affluent neighborhoods.[33]

It is presumed that those outcomes listed above create an environment characterized by high unemployment rates and high poverty rates among residents. These conditions, and the lack of more positive circumstances, can in turn contribute to increases in the prevalence of adverse health behaviors such as smoking and drinking. This effect has been attributed to racial and neighborhood specific targeted marketing and

the increased availability and concentration of convenience stores which supply these products.[34,35,36]

Conditions associated with living in lower ASPoH neighborhoods may also lead to sedentary lifestyles. Physical inactivity may be due to a lack of recreational facilities and proper sidewalks in which to exercise.[37,38] Concern about personal safety issues may also lessen the probability of physical activity within these lower ASPoH areas.

The proposed model theorizes that health food stores will be scarce in lower ASPoH areas. Research shows a lower concentration of supermarkets and a higher concentration of locally owned food stores are found in less affluent areas and in areas in with large African Americans populations.[39] Additionally, the affordability and availability of recommended foods for healthy diets may be reduced in lower income areas.[40] The lack of supermarkets and health food stores with affordable and quality foods may contribute to consuming unhealthy diets. The importance of the differential distribution of these food stores is rendered even more significant for Black Americans when it coupled with the findings that the diet of Black Americans significantly improves as the number of supermarkets in their residential area (census tract) increases.[41]

The proposed model (see Figure 1) theorizes that living in an environment with fewer resources will have adverse health outcomes at varying degrees for Black and White Floridians. It is theorized that the low ASPoH environment will have negative effects on health behaviors accompanied by a higher prevalence of smoking, drinking, unhealthy diets and sedentary lifestyles. The model theorizes that these lower ASPoH groups will tend to experience a higher prevalence of stroke risk factors. Furthermore, because Black Americans are more likely to live in these disadvantaged areas,[42] the

model predicts Black-White disparities in the prevalence of stroke risk factors. It is theorized that all of the factors mentioned above contribute to higher incidences of hypertension, obesity, diabetes and atherosclerosis among black residents culminating in greater Black-White disparities in stroke mortality in low ASPoH level areas versus higher ASPoH level areas.

The primary purpose of this study is to identify contextual area characteristics that may be related to health outcomes independently of and/or in conjunction with social class status (see Figure 2). Specifically, the relationship between ASPoH level and Black-White disparities in stroke mortality will be investigated. In addition, it will be determined whether this association varies across levels of social class. The effect of social class is examined due to the established relationship between social class and health disparities in published literature. Consequently, a thorough examination of the effect of area resource measures on Black-White disparities in stroke mortality requires that the potential influence of social class be assessed. Many questions remain regarding the basis for racial differences in stroke mortality. This study is an attempt to answer a number of these questions, and to suggest directions for future research.

Figure 1: Theoretical Causal Model

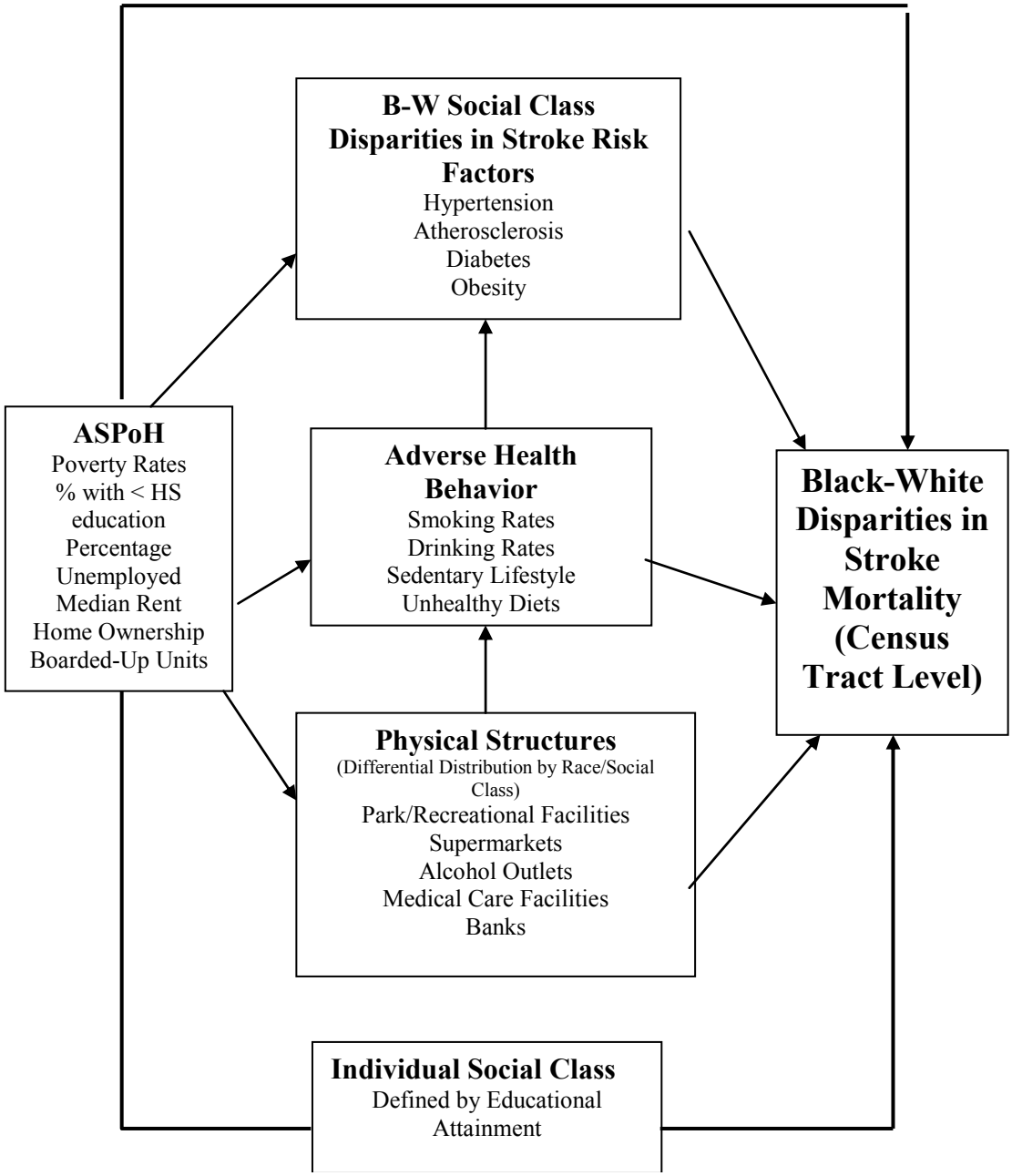
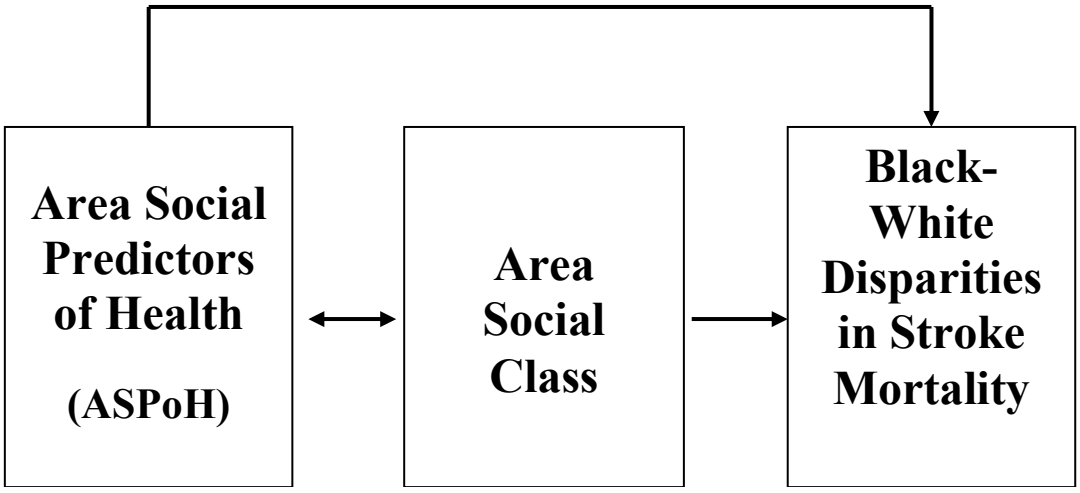


Figure 2: **Examined Theoretical Model**



Chapter Two

Review of the Literature

Stroke Definition

Stroke occurs when there is a sudden complication affecting the blood vessels of the brain.[43,44] There are two major categories of stroke, ischemic and hemorrhagic, and several stroke types within each of these categories. The three major stroke types include: ischemic stroke, intracerebral hemorrhage and subarachnoid hemorrhage. Of all strokes, 88 percent are ischemic, 9 percent are intracerebral hemorrhage and 3 percent are subarachnoid hemorrhage.[45] The following section describes the similarities and differences of these stroke types.

Ischemic Stroke

Ischemic stroke, the most common type of stroke, results from closure or blockage of an artery leading to the brain. There are several causes of ischemic stroke. The most common cause is excessive narrowing of the arteries in the neck or head, usually resulting from atherosclerosis (a gradual cholesterol deposition). This narrowing of the arteries can lead to the formation of blood clots with thrombotic stroke and embolic stroke as possible consequences.

Thrombosis occurs when blood clots block the artery where they are formed. A thrombotic stroke is clinically referred to as cerebral thrombosis or cerebral infarction and is responsible for almost 50% of all strokes. There are two categories of cerebral

infarction, large-vessel and small-vessel thrombosis, each correlating to the location of the blockage within the brain. Large-vessel thrombosis occurs when blockage is located within one of the brain's larger blood supplying arteries, whereas small-vessel thrombosis occurs when there is blockage in one of the brain's smaller and deeper penetrating arteries.

An *embolism* results when the blood clot dislodges and becomes trapped within arteries closer to the brain. In instances of embolic stroke, the clot (or embolus) was formed somewhere other than in the brain itself. These emboli, which often are released from the heart, travel the bloodstream until they become trapped. This blockage restricts the flow of blood to the brain, and results in almost immediate physical and neurological deficits.

Hemorrhagic Stroke

Hemorrhagic strokes, intracerebral (within the cerebrum, or brain) and subarachnoid (area of skull surrounding the brain) hemorrhagic, occur when there is bleeding of ruptured blood vessels in the brain. Intracerebral hemorrhage occurs when a weakened blood vessel within the brain bursts, allowing blood to leak inside the brain. A sudden increase in pressure within the brain can cause damage to the brain cells and possibly lead to unconsciousness or death. Intracerebral hemorrhage usually occurs in selected parts of the brain, including the basal ganglia, cerebellum, brainstem, or cortex. The most common cause of intracerebral hemorrhage is high blood pressure (hypertension). Less common causes include trauma, infections, tumors, blood clotting deficiencies, and abnormalities in blood vessels.

Subarachnoid hemorrhage occurs when a blood vessels just outside the brain

ruptures. The subarachnoid space rapidly fills with blood, possibly resulting in loss of consciousness or death. Subarachnoid hemorrhage is often caused by abnormalities of the arteries at the base of the brain, called cerebral aneurysms. These are small areas of rounded or irregular swellings in the arteries, with the most severe swelling resulting in weakening and rupturing of the arterial wall.

Public Health Importance of Stroke

Approximately 700,000 people experience a new or recurrent stroke each year, establishing stroke as one of the major public health problems in the United States today.[46] About 500,000 of these are first attacks, and 200,000 are recurrent attacks. Eight to 12 percent of ischemic strokes and 37-38 percent of hemorrhagic strokes result in death within 30 days. The age-adjusted stroke incidence rates (per 100,000) for first-ever strokes are 167 for White males, 138 for White females, 323 for Black males and 260 for Black females.[46]

Stroke is the leading cause of serious, long-term disability in the United States, resulting in mounting economic costs.[45] In 2004, it was estimated that Americans would pay $54 billion in direct and indirect cost of stroke. The mean lifetime cost of ischemic stroke in the United States is estimated at $140,048. These costs included inpatient care, rehabilitation, and follow-up care.[47] Lifetime costs per patient are estimated at between $59,000 and $230,000.[48]

In the United States, stroke is the third leading cause of death behind diseases of the heart and cancer.[45] Stroke accounted for more than 1 of every 15 deaths in the United States in 2001. Stroke kills nearly 168,000 people a year, an average of one stroke death every three minutes. Women experience three of every 5 deaths from stroke.

The overall (crude) stroke death rate for 2002 was 56.2 (per 100,000 US population). The 2002 stroke death rates per 100,000 population for specific groups were 54.2 for White males, 53.4 for White females, 81.7 for Black males and 71.8 for Black females.[45] Blacks have higher stroke mortality rate than whites. Higher stroke mortality rates among Blacks can be attributed to a greater incidence of stroke in Blacks, given that thirty-day case-fatality rates, regardless of stroke subtype, compared between the two races is similar (see Table 2.1).[49]

Table 2.1. Thirty-day stroke case-fatality rates by race and stroke subtypes

1999: 30-day case fatality	**All**	**Black**	**White**
All Stroke subtypes	14.7%	12.8%	16.9%
Ischemic stroke	10.2%	9.1%	11.5%
Intracerebral Hemorrhage	37.6%	36.2%	39.0%
Subarachnoid Hemorrhage	31.3%	28.2%	34.7%

Stroke Risk Factors

Non-traditional contributors (e.g., community economic measures, social participation, social cohesion) to stroke incidence and mortality continue to emerge as a greater amount of literature becomes available. However, investigations have identified a consistent grouping of important risk factors for stroke. These risk factors may or may not be modifiable through actions initiated by the individual. Stroke risk factors identified by the American Heart Association include: (1) being African American, (2) older than 55 years of age, (3) male (although more women die from stroke than males), (4) high blood pressure (5) heart disease, (6) diabetes mellitus, (7) prior stroke, (8) heredity and (9) cigarette smoking. Secondary risk factors for stroke include: high blood cholesterol, physical inactivity, and being overweight or obese.[50]

<u>High Blood Pressure and Stroke</u>

A review of published literature of the relationship between blood pressure and

stroke showed that the risk of stroke increases continuously in association with blood pressure levels greater than 115/75 mm Hg.[51] Results also demonstrated that the epidemiologically expected benefits of blood pressure lowering for stroke risk reduction are broadly consistent across a range of different population subgroups. Depending on age, Blacks have 2 to 5 times the prevalence of hypertension of Whites. The great bulk of the adult health differential between Blacks and Whites can be ascribed to this factor.

Diabetes Mellitus

Diabetes is an independent risk factor for stroke, and is strongly correlated with high blood pressure. While diabetes is treatable, the presence of the disease still increases the risk of stroke,[46] with the relative risk ranging from 1.8 to almost 6.0.[52] Diabetes is one of the most important risk factors for stroke in women. In the Framingham Heart Study and in several European studies, the impact of diabetes on stroke risk is greater in women than in men.[52,53]

Cigarette Smoking

The relative risk of stroke among heavy smokers (more than 40 cigarettes a day) is twice that of light smokers (less than 10 cigarettes per day). Stroke risk decreases significantly after two years of smoking cessation and is at the level of nonsmokers by five years after cessation of cigarette smoking.[45,54] Among Americans age 18 and older, 25.2 percent of men and 20.7 percent of women are smokers, putting them at increased risk of heart attack and stroke.[45]

In 1950 Blacks smoked less than Whites, but as a result of migration to large urban centers, this pattern began to change.[45] Although a decline has been reported for all groups, Blacks continue to smoke more than Whites, particularly Black males. There

is an inverse relationship between social class and prevalence of smoking in the US. Smoking prevalence is higher among those with 9-11 years of education (35.4 percent) compared with those with more than 16 years of education (11.6 percent). It is highest among persons living below the poverty level (33.3 percent) compared with other income groups.[55] The higher prevalence of smoking among lower social class and socioeconomic groups undoubtedly contributes to higher stroke mortality rates for these groups.

High Blood Cholesterol and Other Lipids

The higher a person's high density lipoprotein level (HDL), the better the chance of that person not experiencing stroke or heart disease. HDL carries at least one-third of blood cholesterol away from the arteries and back to the liver, where it is passed from the body. Research posits that HDL removes excess cholesterol from plaque in arteries, which slows plaque buildup, and lessens the risk of stroke or heart disease. Low HDL cholesterol (less than 40 mg/dL in adults) is a risk factor for heart disease and stroke. The mean level of HDL cholesterol for American adults age 20 and older is 50.7 mg/dL.[45] The mean level of LDL cholesterol for American adults age 20 and older is 127 mg/dL. Levels of 130-159 mg/dL are considered borderline high. Levels of 160-189 mg/dL are classified as high, and levels of 190 mg/dL and higher are very high.[45] Among non-Hispanic Whites, 20.4 percent of men and 17.0 percent of women have an LDL cholesterol level of 160 mg/dL or higher. Among non-Hispanic Blacks, 19.3 percent of men and 18.8 percent of women have an LDL cholesterol level of 160 mg/dL or higher. Results demonstrate that there is very little racial difference in the prevalence of this particular risk factor.

Physical Inactivity

Based on data from the 1997-2003 NHIS surveys of the CDC/NCHS, 31.3 percent of U.S. adults age 18 and older engaged in regular leisure-time activity.[56] For age groups 18-24 and 25-64, women were less likely than men to engage in regular leisure-time physical activity. The age-sex-adjusted percent of adults who engaged in regular leisure-time physical activity was 34.0 percent for non-Hispanic Whites, 26.4 percent for non-Hispanic Blacks and 21.1 percent for Hispanics. Physical inactivity is more prevalent among women than men, among Blacks and Hispanics than Whites, among older than younger adults and among the less affluent than the more affluent.[56] A recent study of over 72,000 female nurses indicates that moderate-intensity physical activity such as walking is associated with a substantial reduction in risk of ischemic stroke as well as all stroke types combined.[57]

Overweight and Obesity

The age-adjusted prevalence of overweight (BMI of 25.0 or higher) increased from 55.9 percent in NHANES III (1988-94) to 64.5 percent in NHANES IV (1999-2000).[45] The prevalence of obesity (BMI of 30.0 or higher) also increased during this period from 22.9 percent to 30.5 percent. Extreme obesity (BMI of 40.0 or higher) increased from 2.9 percent to 4.7 percent (all prevalence measures were age-adjusted).[58] Increases occurred for both men and women in all age groups and for non-Hispanic Whites, non-Hispanic Blacks and Mexican Americans. Racial and ethnic groups did not differ significantly in the prevalence of obesity or overweight for men. Among women, obesity and overweight prevalences were highest among non-Hispanic Black women. More than half of the women age 40 and older were obese, and more than 80 percent

were overweight.

The prevalence of obesity (BMI 30 or higher) in 2001 increased 5.6 percent between 2000 and 2001(BRFSS, CDC/NCHS). Research suggests that overweight men have a greater risk of developing stroke than those with normal levels of total body fat.[59] A comparison of risk factors in both the Honolulu Heart Program and Framingham Heart Study showed a BMI increase around 3 kg/m^2 raised the risk of hospitalized thromboembolic stroke 10-30 percent.[60] The Health Professionals Follow-up Study examined the association of body mass index and abdominal obesity (waist/hip ratio) with stroke incidence in over 26,000 males aged 40-75. Results suggest that for men, abdominal obesity is more closely related to stroke risk (rather than BMI).[61] A prospective cohort study of middle-aged Israeli men sought to clarify the relationship between excess weight, its distribution, and stroke mortality. The ratio of subscapular to triceps skinfold thickness, an indicator of trunk versus peripheral distribution of body fat, was found to be an independent predictor of long-term stroke mortality.[62] For women, BMI and weight gain are independent risk factors for stroke.[57]

Stroke Mortality Trends

Widespread declines in stroke mortality have been observed over the past several decades. The overall decline in US stroke mortality rate accelerated in the decades between 1950 and 1980, with a marked acceleration noted after 1973.[63] This reduction in stroke death rate occurred in both males and females for both White and Black Americans. Researchers have hypothesized that the decline in stroke death rates may have been due to either decreased incidence of stroke, improved survival of stroke patients, or a combination of these effects. This downward trend in stroke mortality rates

has also been attributed to improved treatment and control of hypertension.[64] Widespread control of hypertension began to take place in the early 1970s. The role of increased detection and control of hypertension in the dramatic reduction in mortality in this time period received support from several studies.

Klag et al (1989) utilized US vital statistics during 1950-1972 and 1973-1981 to gather evidence of the validity of the putative association between the accelerated decline in stroke mortality and increased use of antihypertensives.[65] Researchers propose the 1973 establishment of the National High Blood Pressure Education Program as a candidate to explain the increase in controlled hypertension, thereby resulting in the accelerated decline in stroke mortality shortly thereafter. Stroke mortality declined throughout the study period, however, after 1973, acceleration in the rate of decline was consistently seen in all age-race-sex groups. The rates of decline increased with age. Except in the 75-84 year olds, Blacks had greater rates of decline than Whites. Authors attributed the age- and race-related differences in the rate of decline in stroke mortality to the much higher baseline stroke mortality. However, results of the study lend no support to the proposed link between antihypertensive therapy and decline in stroke mortality. Finding no significant association, authors suggest that treatment of hypertension may not be the principal reason for the decline in stroke mortality after 1973. Alternative proposed candidates include: (1) some widespread environmental agent (2) the targeting of hypertensives and (3) decreased lead exposure (lead exposure has been linked to hypertension and increased stroke incidence) that occurred in 1973-1980 in the US population.

Expanding upon studies of the proposed antihypertensive and stroke mortality

decline link, Capser et al (1992) considered the influence of hypertension prevalence and socioeconomic profile (education, income and occupation indicators) on the proposed association.[66] Results showed that larger changes in both stroke mortality and controlled hypertension occurred during the post-1972 years than during the pre-1972 years. When the two study periods were combined, results showed an association between antihypertensive use and decline in stroke mortality. However, when time period was taken into consideration, no association was observed between treatment and mortality decline pre-1972. Additionally, groups with larger accelerations in stroke mortality declines did not show larger changes in controlled hypertension. During the post-1972 years, the groups with the largest increases in prevalence of controlled hypertension experienced slightly slower rates of decline in stroke mortality. Posing a challenge to the strength of the treatment-mortality decline hypothesis, results showed a consistent association between accelerated declines in stroke mortality and improvements in socioeconomic factors. Pre-1972, groups with the largest increases in education and income profiles experienced the slowest rates of decline in stroke mortality. Post-1972 the trend was reversed. The authors suggest that other factors may operate at the population level that either add to or detract from the effectiveness of increased antihypertensive pharmacotherapy on declines in stroke mortality or that influence the rates of stroke mortality directly.

Geographical Differences in Stroke Epidemiology

Large differences in cerebrovascular disease mortality among geographic areas of the United States have been reported.[67,68,69] Death rates were higher in the southeastern states, and lower in the plains and Rocky Mountain regions. A study of hospitalized

patients was performed to determine whether the mortality differences were due to a higher incidence or case fatality following a stroke in areas with high stroke death rates.[70] Investigators found that the incidence of stroke was higher in the high stroke death rate areas especially for men. The distribution of the specific types of stroke was similar among the areas. No consistent pattern in frequency of symptoms of stroke on admission to hospital was seen. Possible differences in the percentage of all stroke cases that were hospitalized might explain the variations in incidence among the areas. If a high percentage of all stroke cases were admitted to the hospital in the high areas, the incidence based on only hospitalized cases would be inflated relative to the other areas. Blacks have a much higher rate than Whites. Investigators could not determine whether race, sex and geographical differences were due to one specific stroke type.

An epidemiologic study was conducted of geographic differences in stroke mortality between areas (high, intermediate and low stroke rate areas) within the United States.[71] Population samples of 35-54 years of age were drawn for interview and medical examination. Population samples were compared with emphasis on possible risk factors for stroke: serum cholesterol and glucose tolerance test determinations, weight and height measurements, blood pressure and cigarette smoking. The study did not explain the geographic variations in stroke mortality among the high, low and intermediate areas of the United States. Black females showed the expected stepwise progressive increase in severe hypertension from the low to the high stroke areas. White males also showed this pattern, however the differences were not as great. No other consistent pattern of increasing prevalence risk factors for stroke was evident.

Various studies have reported considerable geographic variation of stroke

mortality rates in the United States. The quality of diagnoses on death certificates is questionable, however, certification studies have suggested that large differences in stroke mortality between high and low rate areas are real and reflect differences in the same direction in incidence and possibly case fatality.[72]

At the level of state economic areas (SEAs) changes in the geographic distribution of stroke during 1962 –1982 (period of decline) for White men and White women, aged 35-74 are presented.[71] A cluster of SEA rates in the highest decile is observed in the Southeast (Mississippi, Alabama, Georgia, North and South Carolina). Most SEAs in the highest deciles were in the South. Lowest rates occurred in the western half of the US, particularly in the Plains and Rocky Mountain states. There were SEAs for which stroke mortality rates either increased or did not change. Patterns of higher stroke mortality rates in the eastern US and lower rates in the western US were observed.

Socioeconomic status and living conditions have improved in the United States during the period of the decline. SES is negatively associated with the prevalence of hypertension and with stroke mortality. These associations are consistent with the concentration of high stroke rates in the South, an area economically underdeveloped in relation to the rest of the nation. This region known as the "Stroke belt" became less concentrated over the 2 decades (1962-1982).[71]

Stroke mortality rates, from 1970-2000, for White Floridians tend to be lower than rates for Black Floridians and for the nation as a whole.[72,73] Contrastingly, not only are stroke mortality rates for Blacks slightly higher than national stroke mortality rates, the stroke mortality rates for Black Floridians is 1.5 to 1.9 times higher than rates for White Floridians. Florida stroke mortality rates, by race, compared to US rates can be

seen in Table2.2 below.

Table 2.2. Trends in Stroke Death Rates per 100,000 Population (all ages), Florida and US

	1970		1980		1990		2000	
	Florida	**US**	**Florida**	**US**	**Florida**	**US**	**Florida**	**US**
All Races	138.0	147.7	87.3	96.2	53.5	65.3	48.6	60.9
White	131.4	143.5	83.0	93.4	50.2	62.9	46.1	58.8
Black	199.3	197.1	142.5	129.3	97.5	91.7	81.6	81.9

Black-White Disparities in Mortality

The issue of Black-White disparities in health and mortality is an established concern within the United States. These disparities are consistent across many different health outcomes. African Americans die disproportionately because of higher rates of infant mortality, cancer, substance abuse, asthma, heart disease, diabetes, AIDS, and homicide.[72] Experts continue to debate the origin of these disparities with a decisive focus on socioeconomic influences.

Given that Black Americans experience an excess burden of the majority of the adverse health outcomes, it is appropriate to begin addressing the issue of disparities with a thorough examination of the health of the US Black population. A multitude of health and mortality outcomes depict the disadvantages experienced by the African American population. Infant mortality rates are often used as gauges of the quality of life of populations. In 1998, infants born to African American women have more than twice the rate of death as infants born to non-Hispanic White women.[75] Black Americans are sicker and die younger than Whites.[76] The status of Black health is in decline as evidenced by several indicators. Blacks experience poorer nutrition, more untreated mental illness, more environmental exposure to toxins, and lack of quality health care for

the elderly population.[72] Black women aged forty-five to sixty-four are ten times more likely than White women of the same age to die of diseases of the heart and are five times more likely to die of diabetes. Black women are three to six times more likely than White women to die from complications of pregnancy. African American men in every age group up to age sixty-five and over experience higher mortality rates than that of White males.[77]

Black Americans have higher age-adjusted rates than Whites for the majority of the leading causes of death. In the instance of diseases of the heart, Black Americans have a higher age-adjusted rate (308.4 per 100,000) than White Americans (236.7 per 100,000) (Health US, 2004).[78] Similar disparities are also present for cerebrovascular disease death rates. For Black males (85.4 per 100,000), the age-adjusted death rate for cerebrovascular diseases is about 1.5 times that of White males (54.2 per 100,000), and the death rate for Black females (73.7 per 100,000) exceeds that of White females (54.5 per 100,000) by a similar extent (1.4 times).[79] Black men experience a shorter life expectancy than do any other racial or ethnic minority subgroup (National Vital Statistics Report). At birth, there is a difference of 5.2 years in life expectancy between Black and White Americans (both sexes).

There are also extensive differences between Black and White Americans across various health indicators. Black adults 20 years of age and older are more likely to suffer from hypertension (40 percent) than White adults (28 percent).[75] Black females are much more likely to be overweight (77.7 percent) or obese (50.4 percent) than White females (57.2 percent overweight, and 30.4 percent obese). Compared to 39.1 percent of White females, only 22 percent of Black females achieved a healthy weight. In 1998, the

primary and secondary syphilis case rate for Black Non-Hispanics (16.9 per 100,000) was 34 times the rate for White Non-Hispanics.[75] While many studies have reported significant improvements in mortality and overall health for both Black and White Americans, the Black-White disparity persists with no clear sign of convergence.

Black-White Disparities in Stroke Mortality

As early as the 1960's, investigators began to report geographic differences in the distribution of stroke deaths.[80] Regionally, areas in the Southeastern United States were found to experience higher stroke death rates with lower rates occurring in the Great Plains and Rocky Mountain areas. Later observations demonstrated that not only do these geographic differences exist, however there are concomitant variations in the distribution of stroke deaths among racial groups.[81] In the United States, Blacks were found to experience higher death rates than Whites, a phenomenon even more pronounced in the younger age groups.

Large racial disparities in health status and health care exist between majority Whites and minority racial/ethnic groups in the United States. Data representing US cerebrovascular disease death rates, age-adjusted using the year 2000 standard population, demonstrate that there has been a significant decline in stroke death rates since the 1960's.[82] Generally, White females experience the most "favorable" stroke mortality rates, with Black males experiencing the worst rates. For example, 1990 stroke death rates for White females and Black males were 60.3 and 102.2 per 100,000 resident population, respectively. Although these rates declined in 2000 for each sex-race category, these racial disparities in stroke mortality persist. Strides have been made in the effort to account for some of the Black-White disparities in health outcomes.

Research examining the association between social class and premature stroke mortality demonstrated excess mortality among Blacks compared to Whites at every level of social class.[11] Black-White stroke mortality ratios ranged from 3.9 to 4.9 for social class categories demonstrating that social class (as defined by occupation) accounts for some, but not all, of Black-White disparities in stroke mortality.

African Americans are disproportionately affected by high blood pressure and related morbidity and mortality.[83] In the United States, the prevalence of hypertension increases with age, and is greater for African Americans (32.4%) than non-Blacks (23.3%) and Mexican Americans (22.6%).[84] The complications of uncontrolled high blood pressure, including cerebrovascular accident, are up to four times more prevalent among African Americans than among Whites and there are increases at any given level of blood pressure.[85] Approximately 20% to 30% of deaths among African Americans is directly attributable to hypertension.

Blacks develop high blood pressure at an earlier age and have more severe cases of hypertension than Whites.[84] In addition, Blacks have a 1.3-fold greater rate of nonfatal stroke, a 1.8-fold greater rate of fatal stroke, a 1.5-fold greater rate of heart disease deaths, and a fivefold greater rate of end-stage renal disease,[86] each for which hypertension is a serious risk factor. Compared to the general public, African Americans have 80% higher rate of stroke mortality, 50% higher rate of heart disease mortality and 320% greater rate of hypertension-related end-stage renal disease.

The elimination of these disparities will require a composite of strategies including enhanced efforts at preventing disease, promoting overall health, and delivering appropriate care. When many variables, including income, are held constant, a difference

between Black and White health status still surfaces.[72] The evidence that race correlates with persistent health disparities among different populations in the United States rightly demands the attention of the policymakers and local, state, and national health and human service heads.[72]

Socioeconomic Status and Health

Influences of individual characteristics and neighborhood economic structure on health has been the focus of several studies over the past two decades. Education, income or occupation (or a combination of two or more of these measures) is typically used as a measure of individual social class or socioeconomic status, while an area-based socioeconomic indicator (composed of various area/neighborhood level economic and social measures obtained from census data) often represents the economic structure. Findings from this type of research frequently support the hypothesis that living in economically deprived areas and being a member of a lower SES group are both associated with increased prevalence of negative health outcomes.

Athersclerosis Risk in Communities study (ARIC) data and 1990 US Census data were examined for relatedness of neighborhood (census block group) characteristics to coronary heart disease prevalence and to the distribution of three major CHD risk factors: blood cholesterol, smoking, and systolic blood pressure.[87] Results supported a relationship between living in deprived neighborhoods and increased CHD prevalence and increased levels of risk factors with results persisting after adjustment for individual-level indicators of social class (income, education and occupation).

The relationship between neighborhood characteristics and mortality (all-cause, CVD, and cancer) was investigated for African American and white participants aged 45-

64.[88] The age- and gender-adjusted mortality rate was highest among those who lived in disadvantaged neighborhoods and who were of lower SES. All cause and CVD mortality rates decreased with increasing neighborhood SES advantage and family income in all race-gender groups. Although the pattern generally persisted after adjustment for individual socioeconomic factors, statistically significant associations persisted for CVD mortality in whites only. The lack of significant statistical association after adjustment for individual socioeconomic factors for black participants may be due to their insufficient representation within higher SES neighborhoods.

A prospective study of the associations of individual occupational social class and area-based socioeconomic indicators with mortality revealed that both all cause and cardiovascular mortality rates showed an inverse relationship with socioeconomic position (both at the individual and area based level).[89] Additionally, less favorable socioeconomic position, both individually assigned and area based, were associated with cardiovascular risk disease factors including shorter height, worse lung function, and higher prevalence of bronchitis and coronary heart disease. Interaction between social class and deprivation score were not statistically significant; however, social class differences in all cause and CVD mortality were slightly attenuated but remained substantial and statistically significant after adjustment for area deprivation score. Additionally, all cause and CVD mortality retained sizeable and significant associations with area deprivation after adjustment for social class.

After controlling for personal income, education, and occupation, a prospective study found that living in a disadvantaged neighborhood is associated with an increased incidence of coronary heart disease.[22] Hazard ratios for coronary heart disease among

low-income persons living in the most disadvantaged neighborhoods, as compared with high-income persons in the most advantaged neighborhoods, were 3.1 among Whites and 2.5 among Blacks. Additionally, these associations remained unchanged after adjustment for established risk factors for coronary heart disease.

An inverse association for all cause mortality with both individual and area level indicators of socioeconomic status was found for an American Cancer Society cohort.[90] When both variables were included simultaneously in the analysis, the effect of individual level SES remained, while area level effects were somewhat diminished.

Neighborhood affluence, as measured by the percentage of neighborhood residents with a household annual income of $50,000 and over, was shown to be positively correlated with the self-rated health of adult residents of the metropolitan Chicago area.[91] The positive health effect of neighborhood affluence continued even after controlling for individual-level socioeconomic (income, education), demographic and health-related background factors.

A cross-sectional study of women from British electoral wards found that adverse area-level socioeconomic characteristics, over and above individual life-course socioeconomic position (SEP), are associated with increased coronary heart disease.[92] After adjustment for age and 10 indicators of individual life-course SEP, the odds of coronary heart disease was 27% greater among those living in wards with a deprivation score above the median compared with those living in a ward with a deprivation score. Additionally, the size of the association between neighborhood unemployment rates (as a measure of deprivation) and all cause mortality from samples across six countries (US, Netherlands, England, Finland, Italy, Spain) demonstrated that living in more deprived

neighborhoods is associated with increased all cause mortality independent of individual socioeconomic characteristics.[93]

Socioeconomic Status and Stroke

Stroke incidence, survival and mortality and their relation to individual and area socioeconomic status has been investigated. A prospective study in the United Kingdom found an association of higher area deprivation with stroke at younger age, severe stroke, higher baseline systolic blood pressure, and with higher rates of stroke risk factors.[94] Stroke mortality was associated with area deprivation after correction for age, sex and stroke risk factors. A cohort study in the Netherlands demonstrated a statistically significant association between area socioeconomic status (postcode areas) and stroke incidence.[16] Residents of postcode areas with below average socioeconomic status experienced a significantly higher incidence of stroke than residents of postcode areas with average or above average socioeconomic status. Scottish hospital patients from the most socially deprived areas were shown to be significantly more likely to be dead or dependent 6 months after admission for an acute stroke.[95] No adjustments were made for individual level socioeconomic measures. Similarly, follow-up studies of stroke patients found an association between survival and individual level socioeconomic status (occupation, occupational status and income)[96] and between risk of fatal stroke and having less than 12 year of education.[97] The less educated, the lower level employees, the unemployed, and the lower income groups, experienced higher risk of death compared to their counterparts.

It is well established that the socioeconomic position of individuals, groups and places is a defining characteristic of their level of health and disease.[98] Scientific

understanding of the processes through which neighborhood SES influences lifestyle, health promoting opportunities, morbidity and mortality is essential. The goal of future research in this area must be to advance our understanding of these effects and their policy implications.

Theories of Causation

A multitude of theories have been proposed to address the issue of how specific aspects of society, and the people who live within, work together to influence population health. When researchers began to realize the importance of the environment in the promotion of health and illness, many took on the task of identifying specific quantifiable aspects of society that could be scientifically related to health outcomes. Although major steps forward have been made in reducing morbidity and mortality, the social class and racial divide continues to widen. The connection between individual level behaviors with conditions at the societal level, results in complexities which have led to the lack of more substantial improvements in health.[99]

Social epidemiology holds that we embody or incorporate biologically the world around us. It attempts to answer the question of who and what it is that is responsible for population patterns of health, disease and well-being, as manifested in present, past and changing social inequalities in health. The three main theories of social epidemiology (psychosocial theory, social production of disease and ecological theory) are described below.

The psychosocial theory is based on the host-agent-environment relationship. The psychosocial framework directs attention to endogenous biological responses to stress and on stressed people in need of psychosocial resources. Researchers following

this framework believe that in order to explain disease distribution we must investigate factors influencing susceptibility.[100] The belief is that the social environment alters host susceptibility by affecting neuroendocrine function. Relevant psychosocial factors include social disorganization, rapid social change, bereavement, social support (which is believed to be a buffer to all of the above). Because of the believed ability of social support to buffer the effects of the psychosocial factors, the most feasible and promising interventions to reduce disease will be to improve or strengthen the social supports rather than reduce the exposure to stressors. This theory dedicates no attention to: (1) who or what generates psychosocial insults and buffers to these insults, (2) how their distribution is shaped by social, political and economic policies or (3) time.

The social production of disease theory, with its Marxist origin, is also known as the political economy of health.[76,101] This theory is an advocacy of materialist analysis of health. These materialist analyses address economic and political determinants of health and disease including structural barriers to people living healthy lives. At issue are priorities of capital accumulation and their enforcement by the state so that few can stay rich while the many are poor. In this theoretical framework, determinants of health are analyzed in relation to who benefits from specific policies and practices, at whose cost. The theory posits that economic and political institutions and decisions that create, enforce and perpetuate economic and social privileges and inequality are root or fundamental causes of social inequalities in health. The theory attempts to determine the health impacts of rising income inequality, and the experience of economic and non-economic forms of racial discrimination. The call for action is for healthy public policies, especially redistributive policies to reduce poverty and income inequality. This

includes an attempt to change unjust social and economic policies and norms and to provide systematic framework for delineating government accountability to promote and protect health.

The ecological theory presents an analysis of current and changing population patterns of disease, health and well being in relation to each level of biological, ecological and social organization as manifested at each and every scale.[101,102]The theory embraces a social production of disease perspective while aiming to bring in a comparably rich biological and ecological analysis. It elucidates population patterns of health, disease and well being as biological expressions of social relations and proposes multilevel pathways linking expressions of stressors, for example (racial discrimination) and their biological consequences across the lifecourse. The theory embodies biological expressions of racism and emphasizes accountability. It extends beyond psychosocial explanations focused on anger and hostility to the social phenomena (interpersonal and institutional discrimination) eliciting these responses, as mediated by material pathways. There is an interplay between exposure, susceptibility and resistance and it advances beyond social production of disease analyses typically focused on racial/ethnic disparities in socioeconomic position to highlight discrimination within class strata plus ongoing biological impact of economic deprivation in early life.

The social determination movement, which embodies the social production of disease and the ecosocial theory, studies the inequality of health within a nation or among nations.[103] It sees steep gradients of education, income, and social position as adversely affecting the health of a population, not only at the bottom but throughout the entire range of the social structure. This theory holds that inequality rather than absolute deprivation

in developed economies undermines the capacity of people to resist disease. The psychological concomitants of steep social positioning are emphasized both at the individual (self-esteem, hopefulness) and collective (community efficacy, social capital) levels. The task is to integrate the insights of these most perspective efforts and to confront health, society, and habitat as a whole, in their full complexity.

Stallones (1973) indicated a need for a broader view of the disease processes when attempting to address the issue of causation.[104] The burden of disease on human populations is seen as part of an environmental system. The disease process is depicted as an interaction of biologic, social and physical factors. Stallones proposed that the interrelatedness of the components of the system cannot be understood by pursuing research whose rationale is to divide and isolate the components in even greater detail. The belief was that disease is embedded in the environment of man, and that diseases of a society characterize the environment. It was suggested that physical environmental characteristics, demographic and social characteristics, and disease (total mortality and morbidity) need to be brought together in order to obtain a deeper understanding of disease as a community phenomenon.

Cassel (1976) proposed the 'social environment' as environmental factors capable of changing human resistance to disease and of making subsets of people more susceptible to ubiquitous agents in the environment.[100] Pyschosocial processes were presented as agents capable of altering the endocrine balance in the body, increasing susceptibility to disease. Stress, defined as either a dynamic state within the individual or as a stimulus assault (any aspect of the environment), was presented as one of these psychosocial factors. It is believed that psychosocial factors should be regarded as

predisposing to disease and not causal. The stressor could be unfamiliarity with cues and expectations of society (immigration) leading to higher susceptibility (under conditions of social disorganization). Authors offered social support as a potential protective factor, buffering the individual from the physiologic or psychological consequences of exposure to the stressor situation. Empirical evidence suggests that Black males living in high stress areas have higher blood pressures. Results were not the same for Whites. Authors suggest that these results may reflect a subservient role of Blacks. The lack of association between high stress areas of Whites and blood pressure levels was explained as Whites potentially having more resources in the face of social disorganization. Additional examples of psychosocial risks include racism, low income, physical abuse, and psychological abuse.

Crawford (1977) addressed the victim blaming ideology which emphasizes individual responsibility for health.[105] Crawford asserted that this ideology serves to re-order expectations and to justify a retreat from the language of rights and the policies of entitlement (to medical care). The common theme of the victim blaming ideology emphasizes the need to reduce expectations and utilization of ineffective and costly medical services and instead to increase the necessity for individual responsibility. This ideology instructs people to be individually responsible at a time when they are becoming less capable as individuals of controlling their health environment. Crawford believed that this blaming ideology obscures the class structure of work, removing the focus away from influence of place in society on health and well being.

Stallones (1980) expressed the need for the development of theories of causation.[106] Development of epidemiologic theory would involve the arrangement of

facts into an orderly chain of inference. It was believed that this epidemiologic theory, which is likely to be unique, would guide the collection of data and the organization of information. During this time period, ecologically based epidemiology was commonly utilized to characterize communities simultaneously by both physical and social circumstances. Individual traits were measured and related to the overall morbidity and mortality patterns of the communities. Instead, Stallones proposed that the community be viewed as a social organization. The distribution of disease in communities would therefore be considered a social phenomenon, and as such, might be expected to have social causes. It was believed that physical and social environmental factors affect the specific etiological agents, along with the likelihood of exposure and the degree of susceptibility of the exposed persons.

Why Blacks Have Higher Stroke Mortality: Influence of Social Environment

Why are Black Americans more vulnerable to adverse health than White Americans who reside in the same area? This study theorizes that increased vulnerability to adverse health among Black Americans is differentially mediated by various environmental factors and conditions. These environmental factors, measured in terms of resource availability for purposes of this study, in turn influence individual lifestyle choices that may be detrimental to health. However, the availability of resources may not be truly representative of the degree of access to these resources. Compromised access will result in underutilization of "available" utilization. This is a circumstance witnessed more often in the Black population. This underutilization of resources by Black Americans may be viewed in terms of unhealthy "lifestyle choices" when in reality the choices may have been extremely limited. This process possibly culminates in Black-

White health disparities within geographic areas where Black and White residents supposedly share resources.

To investigate this problem, this study utilizes a theoretical focus on social and economic characteristics at the census tract level and the influence of these characteristics beyond those typically observed at the individual level. The environments in which people live their lives afford them a certain amount of opportunities for utilization of available resources. Explanations for illness and mortality are typically limited to the individual behaviors of Blacks and few studies address the social context in which these behaviors occur. This study aims to direct focus on social influences of Black-White health disparities, stroke mortality in particular.

This study is carried out in a psychosocial context, conceding that the study does not directly measure the influence of specific psychosocial risk factors on Black-White disparities in stroke mortality. This study builds on the perception that psychosocial influences, specifically racism, directly and indirectly influence access to community resources. The study hypotheses contend that research on Black health (and the Black-White disparities that result) should conceptualize Black health as a complex interaction of psychosocial risks which influence access that have a profound effect on that health. Laws based on racist ideals created these situations in which Blacks are more likely to experience environmental influences disadvantageous to health.

Conclusion

The fundamental attribute differentiating social class categories relates to differences in the power to access material resources. Material factors therefore seem an obvious candidate for consideration as an explanation of social health inequalities.[107]

Because of the unequal distribution of Black and White Americans within these social class groups, the influence of race on access to material resources should be assessed. While Black Americans have made health status improvements, a multitude of racial disparities still exist. A major reason is that, although legislation may have made health care relatively more available and affordable, the fundamental and unequal structure of American society, which is primarily responsible for racial disparities in health, remains unchanged. The social environmental conditions in which a large portion of Black Americans live may not be conducive to enduring implementation of lifestyles which promote cardiovascular health. The close connections between the socioeconomic conditions people live under and their adaptive behavioral profile must be acknowledged.

Chapter Three

Methods

Study Design

The study type is of a mixed design. It is fundamentally an ecological design, but this study does have elements of a retrospective cohort study design in that the study participants' community resource availability is utilized as a predictor of racial disparities in stroke mortality. However, given that the study outcome time period is 1998-2002, while the community resource indicator, or "predictor," was taken from the 2000 Census of Population and Housing, establishing that the exposure preceded the outcome cannot be achieved. A 5-year study period was chosen to increase the amount of data that would be available for small area analysis and the year 2000 chosen as the midpoint of the study period in order to use the population and socioeconomic data from the 2000 Census of Population and Housing.

Study Population

The geographic study area is the State of Florida. Our study population consisted of Non-Hispanic White and African American (both Hispanic and non-Hispanic) adults aged 35 and older who resided in the State of Florida during the years 1998-2002. As of the 2000 census, Florida had a total population of almost 16 million. White Americans made up seventy-eight percent of the population whereas African Americans make up 14.6 percent. Almost 17 percent of Florida residents were reported being of Hispanic

ethnicity. Study subjects included 43,945 Florida residents aged 35 and older, of which 13,605 were aged 35-74 years, who died from stroke during the 1998-2002 time period. Stroke incidence and stroke mortality is less common in those younger than age 35, while stroke incidence and mortality rates are considerably elevated for those beyond the age of 75. To lessen the effect of these extreme rates, very low and very high, on study results, only those decedents aged 35-74 were included in the study.

Data Sources

The level of analysis utilized in this study is the census tract. Census tracts are small, relatively permanent statistical subdivisions of a county or statistically equivalent entity.[108] A primary purpose of census tracts is to provide a stable set of geographic units for the presentation of decennial census data. For Census 2000, the entire United States was covered by approximately 65,000 census tracts, while the State of Florida consisted of 3154 census tracts. The specific number of census tracts utilized for study analyses within each research question was dependent upon the specific study outcome. The number of census tracts utilized, in addition to a discussion of the loss of census tracts, is presented along with the appropriate analyses results.

Stroke Mortality Data

Stroke mortality data was obtained from the Florida Department of Health. The Florida Department of Health provided a data set containing information on all 1998-2002 decedents in the study population for whom the underlying cause of death was coded as stroke. Information was not obtained for decedents who died from causes other than stroke; therefore, the files only contained Florida stroke decedents. Data on age, gender, race, Hispanic ethnicity, educational attainment, and census tract of usual

residence were the only data included in the files obtained. Each stroke death was point located (geocoded) within its proper census tract by the Florida Department of Health (see Appendix A). Stroke deaths were identified as those with the International Classification of Diseases (ICD)-9 (for 1998 decedents) and ICD-10 (for 1999-2002 decedents) codes indicating 'cerebrovascular diseases' as the category for cause of death. Specific coding used for the death certificates is presented in Table 3.1

A total of 10,799 stroke decedents, ages 35-74 years, for the study area and time period were included in the study. Data for census tract of residence, age, gender and cause of death were available for 100% of the stroke decedents included in the study. Race data was available for 99.96% of the decedents, while Hispanic ethnicity data was

Table 3.1. International Classification of Diseases (ICD) Codes for Cerebrovascular Diseases

	ICD 9 (430-438)
430	Subarachnoid hemorrhage
431	Intracerebral hemorrhage
432	Other and unspecified intracranial hemorrhage
433	Occlusion and stenosis of precerebral arteries
434	Occlusion of cerebral arteries
435	Transient cerebral ischemia
436	Acute but ill-defined cerebrovascular disease
437	Other and ill-defined cerebrovascular disease
438	Late effects of cerebrovascular
	ICD 10 (I60- I69)
I60	Subarachnoid haemorrhage
I61	Intracerebral haemorrhage
I62	Other nontraumatic intracranial haemorrhage
I63	Cerebral infarction
I64	Stroke, not specified as haemorrhage or infarction
I65	Occlusion and stenosis of precerebral arteries, not resulting in cerebral infarct
I66	Occlusion and stenosis of cerebral arteries, not resulting in cerebral infarction
I67	Other cerebrovascular diseases
I68	Cerebrovascular disorders in diseases classified elsewhere
I69	Sequelae of cerebrovascular disease

available for 99.72% of the decedents. For educational attainment, 96.57% of the stroke decedents had available data.

Population Data

Population counts were obtained for the year 2000 US census from the United States Census website. The 2000 census year served as the midpoint for the five-year study period. Summary Files 3 (SF3) and 4 (SF4) data were obtained at the census tract level for population count purposes. These counts were multiplied by 5 as death data were available for a five-year time period, 1998-2002. This methodology is utilized since it is the most accurate method to estimate the total population since the population typically increases each year. Utilizing this method, possibly overestimated the population for the years of 1998 and 1999. However, the population is more than likely underestimated for the years of 2001 and 2002. This provides for the calculation of average annual age-adjusted rates.

Area Social Predictors of Health (ASPoH) Data

Census tract data on population and socioeconomic characteristics were obtained from the 2000 Census of Population and Housing Summary File 3 (SF3). Summary File 3 contains information compiled from the questions asked of a sample of all people and housing units. Specific information in the SF3 population files includes: population total, urban or rural denotation, households and family types, marital status, educational attainment, poverty status and many other factors. Summary File 3 contains a total of 813 unique tables, a subset of which is repeated by race and Hispanic or Latino ethnicity. Local area indicators of the ASPoH variable were calculated from these summary files.

Statistical Products

SAS computer statistical package, Version 9.1 (SAS Institute Inc, Cary, NC) was used in the analyses of study data.

Analytic Methods

Race and Sex Specific Mortality Rates

Race and sex specific age-adjusted stroke mortality rates were calculated utilizing the direct method of standardization. An age-adjusted rate is a weighted average of the age-specific rates. The standard population distribution used to adjust the stroke death rates was the 2000 US Standard Population (35-74 years of age for Research Question 1 and 35 years of age and up for Research Questions 2 and 3). The stroke death rate for each census tract was calculated for each race-sex specific age group. The number of stroke deaths in that race-sex specific age-group was divided by the population of the same race-sex specific age-group within that census tract. The race-sex age-group-specific stroke death rate was then multiplied by the proportion of the standard population (see Table 3.2) for that specific age-group. The weighted age-specific rates are then summed for the census tract to calculate the age-adjusted rate. Direct adjustment reduces the potential for confounding by age; therefore, comparison of death rates across racial group with different age distributions is possible.

Table 3.2. Year 2000 Standard population weights

Age Group	2000 Proportion
35-44 years	0.363877
45-54 years	0.297873
55-64 years	0.191597
65-74 years	0.146652

Black-White Disparity Scores

Black-White disparity scores were calculated both on an absolute scale (difference score) and on a relative scale (ratio and percent difference scores). The absolute measure of disparity is expressed simply as the arithmetic difference between the Black stroke death rate and the Non-Hispanic White stroke death rate (reference point). The difference score provides information on the excess number of stroke deaths among Black Floridians. The ratio score is interpreted as the relative magnitude of the Black stroke death rate compared to the Non-Hispanic White stroke death rate (expressed as a quotient). The ratio score is an index of how serious the stroke mortality risk is for Blacks relative to Non-Hispanic Whites. The percentage difference score is expressed as the difference between rates (Black minus Non-Hispanic White) as a percentage of the Non-Hispanic White death rate. Absolute and relative measures of disparity calculated from the same reference point (Non-Hispanic White rate) should lead to the same conclusion about stroke mortality disparities between these groups. Utilization of both absolute and relative measures allows for a check of consistency between the disparity measures.[109] Methods of calculation of the disparity scores are presented in Table 3.3.

Table 3.3. Disparity score calculation methods

Black-White Disparity Measure	Formula
Male Ratio Score	BMAAdeathrate[1] ÷ NHWMAAdeathrate[2]
Female Ratio Score	BFAAdeathrate[3] ÷ NHWFAAdeathrate[4]
Male Difference Score	BMAAdeathrate - NHWMAAdeathrate
Female Difference Score	BFAAdeathrate - NHWFAAdeathrate
Male Percent Difference Score	(BMAAdeathrate - NHWMAAdeathrate) ÷ NHWMAAdeathrate
Female Percent Difference Score	(BMAAdeathrate - NHWMAAdeathrate) ÷ NHWMAAdeathrate

[1] Black Male Age-Adjusted stroke death rate, [2] NH- White Male Age-Adjusted stroke death rate
[3] Black Female Age-Adjusted stroke death rate, [4] NH-White Female Age-Adjusted stroke death rate

Statistical Methodology

This study attempted to create a variable that would properly reflect the contextual characteristics of an area possibly affecting Black-White disparities in stroke mortality. Area Social Predictor of Health (ASPoH) describes features of social organization, structure, and stratification of the environment, such as socioeconomic deprivation, economic inequality, resource availability, and opportunity structure. Specifically, this study attempted to compile a set of indicators that would closely reflect both the study residents' economic resource availability and their probability of obtaining these resources. In order to construct this area socioeconomic status measure, several indicators were statistically transformed into a smaller number of variables known as principal components.

The decision to include specific measures was based on a core set of 12 dimensions of social determinants of health. This core set grew out of a University of Michigan School of Public Health project funded by the Centers for Disease Control and Prevention.[110] Investigators representing a wide range of disciplines participated in a workshop to review dimensions important in understanding social determinants of health. Participants were able to arrive at a consensus on a core set of 12 dimensions (4 of which are assessed in this study). The directory contains an extensive list of available types of data sets. Workshop participants generated suggestions for possible data sources and specific variables that *might be* used to measure the components of each dimension. Researchers may choose to utilize certain elements from this list in order to evaluate how the social environment impacts the health of populations. The data sets are organized according to the 12 dimensions specified in Table 3.4.

Table 3.4. Twelve core dimensions to understanding social determinants of health.

1	Economy	7	Medical
2	Employment	8	Governmental
3	Education	9	Public Health
4	Political	10	Psychosocial
5	Environmental	11	Behavioral
6	Housing	12	Economy

For this current project, data were available for measures representing four of the twelve core dimensions. The census tract level measures used in the construction of the ASPoH variables for this study are as follows:

Economy Dimension

1. Poverty Rate

2. Median Income

Employment Dimension

3. Percent Unemployed

4. Percent of workers aged 16 years or older using private transportation to work

5. Full vs. part-time employment

Education Dimension

6. High School Graduation rates for those 25 years of age and older

Housing Dimension

7. Median Rent

8. Median value of owner occupied housing units

9. Vacancy rates

10. Home Ownership

11. Overcrowded Housing

The methods of calculation for these 11 variables is reported in Appendix C.

Principal Component Analysis Methodology

Principal Component Analysis (PCA) involves a mathematical procedure that transforms a number of possibly correlated variables into a smaller number of uncorrelated variables called principal components. This is accomplished by first identifying patterns in the data, followed by expressing the data in such a way as to highlight their similarities and differences. The first principal component produced by the mathematical procedure accounts for as much of the variability in the data as possible, and each succeeding component accounts for as much of the remaining variability as possible.[111]

Direct uses of Principal Component Analysis include: (1) identification of new "meaningful" underlying variables and (2) reduction of number of variables.[112] To obtain reliable results, the minimal number of subjects providing usable data for the analysis should be the larger of 100 census tracts or five times the number of variables being analyzed. In these analyses, the minimum sample size requirement was met with 3154 census tracts contributing usable data.

Generated components are thought to be representative of the underlying processes that have created the correlations among variables.[112] Variables that are correlated with one another which are also largely independent of other subsets of variables are combined into components. Components may either be associated with 2 or more of the original variables (common factors) or associated with an individual variable (unique factors).

The number of components extracted in a principal component analysis cannot exceed the number of observed variables being analyzed. The *principal component* is a linear combination of optimally weighted observed variables. The first component extracted accounts for a maximal amount of total variance in the observed variables. The second component extracted accounts for a maximal amount of variance in the dataset that was not accounted for by the first component, and it will be uncorrelated (r=0) with the first component. Each remaining component accounts for a maximal amount of variance in the observed variables that was not accounted for by the preceding components and is also uncorrelated with all the preceding components. The resulting components (all extracted components) will display varying degrees of correlation with the observed variables, but are completely uncorrelated with one another.

Loadings relate the specific association between factors and original variables. Therefore, it is necessary to find the loadings, then solve for the factors, which will approximate the relationship between the original variables and underlying factors. The loadings are derived from the magnitude of eigenvalues associated to individual variables.[112]

Steps in Conducting Principal Component Analysis

(1) Initial Extraction of the Components

The number of components extracted is equal to the number of variables being analyzed. An eigenvalue table is presented. The eigenvalue represents the amount of variance that is accounted for by a given component. The first components extracted will account for relatively large amounts of variance, while the later components account for relatively smaller amounts.

(2) Determining the number of "Meaningful" Components to Retain

There are four techniques that may be used to determine the number of principal components that may be retained for further analyses. One criterion involves retaining any component with an eigenvalue greater than 1.00. The rationale for utilizing this technique evolves from the fact that each variable contributes one unit of variance to the total variance in the dataset. Any component that displays an eigenvalue greater than 1.00 is accounting for a greater amount of variance than had been contributed by one variable. Such a component is therefore accounting for a meaningful amount of variance, and is worthy of being retained.

A second criterion involves the use of the Scree test. This test involves plotting the eigenvalues associated with each component and looking for a break between the components with relatively large eigenvalues and those with small eigenvalues. The components that appear before the break are assumed to be meaningful and are retained for rotation.

A third criterion is the interpretability criteria. This techniques involves interpreting the substantive meaning of the retained components and verifying that this interpretation makes sense in terms of what is known about the constructs under investigation. There are four rules to follow in doing this: (1) Are there at least three variables with significant loadings on each retained component? (2) Do the variables that load on a given component share the same conceptual meaning? (3) Do the variables that load on different components seem to be measuring different constructs? (4) Does the rotated factor pattern demonstrate "simple structure?"

The final criterion, takes into account the proportion of variance accounted for by

a specific set of components. This criterion requires that components are retained if the cumulative percent of variance accounted for is equal to some minimal value (70 to 80%).

The decision was made to retain four principal components for this study. The retention of these four components satisfied each of the four criterion suggested for determining the number of principal components to retain for inclusion in further analyses. All components with an eigenvalue greater than one were included in this study (components 1, 2 and 3). The Scree test resulted in a break between principal components 3 and 4. A substantive interpretation for each of the four retained components was accomplished. Finally, the first four principal components accounted for approximately 76% of the variance in the data.

Preliminary Analyses

Dividing ASPoH Measures into Quartiles

There is no gold standard for assessing the predictability of the ASPoH index; therefore, the association between the ASPoH index and Black-White disparities in stroke mortality was examined in two ways. First, ASPoH categories were created. Census tracts were categorized based on group distribution of the ASPoH index. Therefore, categorization was as follows: (1) below the 25^{th} percentile, (2) between the 25^{th} and 50^{th} percentile, (3) between the 50^{th} and 75^{th} percentile or (4) above the 75^{th} percentile. The values were assigned to groups in ascending order, with the smallest value assigned to the first quartile and so on. These methods resulted in 25% of the census tracts being contained within each category. Therefore, each ASPoH category contains either 788 or 789 (3154 census tracts divide 4 groups) census tract values each. Black-White disparity

scores for stroke mortality were calculated for each of the ASPoH categories and compared. Because of the limited range in value of each ASPoH variable, quartiles, instead of quintiles, were used.

Each census tract was assigned the best-fitting category of ASPoH based on the homogeneity of the ASPoH indicators. This process allowed for the calculation of age-adjusted stroke death rates (separately for Blacks and Whites) for each of the ASPoH categories. SF3 data from the 2000 US Census was utilized for denominator purposes in order to obtain population counts at the census tract level for race*sex*age. The age-adjusted stroke death rates were used to calculate Relative Risks, with the "most favorable" ASPoH category as the referent group.

Research Question One Analyses

Research Question 1

Do lower levels of ASPoH status result in greater black-white disparities in stroke mortality?

The multiple linear regression model was used to test the predictability of Black-White disparity in stroke mortality, as well as, race-sex specific age adjusted stroke mortality rates (ages 35-74), by the ASPoH measures (4 principal components) at the census tract levle. ASPoH scores and Black-White disparity measures were calculated for each of the individual census tracts. During these analyses, the census tracts were not categorized into ASPoH quartiles as in the previous analyses. The strength of predictability was determined at the individual census tract level. The models tested in this phase of the analyses include:

(1) Black Female Age-Adjusted Stroke Mortality Rate = β_0 + β_1ASPoH1 + β_2ASPoH2 + β_3ASPoH3 + β_4ASPoH4 + ε

(2) Black Male Age-Adjusted Stroke Mortality Rate = β_0 + β_1ASPoH1 + β_2ASPoH2 + β_3ASPoH3 + β_4ASPoH4 + ε

(3) Non-Hispanic White Female Age-Adjusted Stroke Mortality Rate = β_0 + β_1ASPoH1 + β_2ASPoH2 + β_3ASPoH3 + β_4ASPoH4 + ε

(4) Non-Hispanic White Male Age-Adjusted Stroke Mortality Rate = β_0 + β_1ASPoH1 + β_2ASPoH2 + β_3ASPoH3 + β_4ASPoH4 + ε

(5) Male Black-White Disparity Ratio = β_0 + β_1Prin1 + β_2Prin2 + β_3Prin3 + β_4Prin4 + ε

(6) Male Black-White Disparity Difference = β_0 + β_1Prin1 + β_2Prin2 + β_3Prin3 + β_4Prin4 + ε

(7) Male Percent Difference = β_0 + β_1Prin1 + β_2Prin2 + β_3Prin3 + β_4Prin4 + ε

(8) Female Black-White Disparity Ratio = β_0 + β_1Prin1 + β_2Prin2 + β_3Prin3 + β_4Prin4 + ε

(9) Female Black-White Disparity Difference = β_0 + β_1Prin1 + β_2Prin2 + β_3Prin3 + β_4Prin4 + ε

(10) Female Percent Difference = β_0 + β_1Prin1 + β_2Prin2 + β_3Prin3 + β_4Prin4 + ε

Research Question Two Analyses

Research Question 2

Question: Does low social class result in greater Black-White disparities in stroke mortality?

Social class categorization was determined by educational attainment information extracted from the death certificates. Five categories of social class were defined in the following manner:

(1) Social Class 1 (High): College graduates (with degree) and beyond

(2) Social Class 2: Some college education and/or Associates Degree

(3) Social Class 3: High School graduates/12 years completed

(4) Social Class 4: 9-11 years of school completed

(5) Social Class 5 (Low): 0-8 years of school completed

Population counts were obtained from Summary File 4 data (from the 2000 US Census). Population counts stratified by race, social class, sex, and 10-year age groups (ages 35 and up) were used to calculate stroke death rates for the years 1998-2002 by social class. To be retained in the study, the individual census tract must have a population count of at least one within each race-gender-10yr age group category. A total of 2156 out of the original 3154 census tracts met this criterion. Each stroke decedent was assigned to the appropriate social class category and race specific stroke mortality rates calculated for each social class category. Black-White disparity scores were calculated for each social class group at the census tract level. Limitations of the data resulted in the calculation of the disparity scores at the state level only.

Research Question Three Analyses

Research Question 3

Question: Is there effect modification by social class of the ASPoH and Black-White disparities in stroke mortality relationship?

For each social class category, linear regression analyses were performed to access the association between Black-White disparity in stroke mortality and ASPoH variables (ages 35 and up). Whether the relationship (as measured by parameter estimates) between ASPoH and Black-White disparity is stroke mortality varied across social class categories was investigated.

Separate analyses were conducted for each social class group. For each social class category, the predictability of Black-White disparities in stroke mortality by the ASPoH variable was determined. These separate analyses were examined to determine whether the magnitude of the parameter estimates (accessing the association between the particular disparity score and the ASPoH variable) varied across different categories of social class.

Chapter Four

Results

Part I. Area Social Predictors of Health

Descriptive Statistics

Principal component analysis methodology was utilized to construct the 'Area Social Predictors of Health' variables. Summary statistics for each of these census tract level variables subjected to principal component analysis are presented in Table 4.1. The median employment rate at the census tract level is 94.19%. This results in an unemployment rate close to 4.5%, which is very close the national unemployment rate of 5%. Fifty-one percent of those employed residents are full-time employees. The average percent above poverty rate for Florida census tracts was 90% for the 2000 census year. The variability of the data is not unreasonable given that we are dealing with data at a small geographical unit (census tract). On average, 12% of the homes in each census tract were vacant and less than 1 percent of these owner-occupied homes were overcrowded. Seventy percent of these occupied housing units were owner occupied. Median homes values averaged around $105,000, while renters paid an average of $677 per month.

On average, nearly 80% of residents 25 years and older had received their high school diploma. The study average poverty rate of 10.04% was slightly lower than that of the United States in 2001 (12.1%) and slightly lower than the poverty level for the

state of Florida which is 11.5% (1999-2000 average). The study median household income of $43322.50 is almost identical to that of the United States in 2003 which was $43, 381.[113]

Table 4.1. Summary Statistics: Area Social Predictors of Health Variables, 2000 US Census, 3154 Census Tracts

ASPoH Variables	Mean	Median	Std Dev	Minimum	Maximum	Skew
Percent Employed	94.19	95.46	5.53	3.33	100.00	-6.45
Percent Above Poverty Rate	89.96	92.96	9.25	26.22	100.00	-2.08
Percent of Occupied Homes	88.19	91.01	10.07	0.00	100.00	-2.33
Percent of Non-crowded Homes	99.07	99.88	1.85	79.17	100.00	-3.54
Percent Using Private Transport	90.63	92.74	7.93	17.63	100.00	-3.70
Percent 25yr+ with High School Diploma	79.24	82.06	13.29	20.46	100.00	-1.01
Percent of population Employed Full-time	51.33	53.00	11.74	4.38	100.00	-0.73
Median Income	47697.00	43322.50	20334	0.00	200001.00	1.82
Percent Home Ownership	69.75	75.42	21.04	0	100.00	-1.06
Median Rent	677.78	636.00	248.75	0	2001.00	1.52
Median Home Value	105417.70	87050.00	72984.00	0	1000001.00	4.66

In the correlation matrix (Table 4.2), the majority of the correlations were in the expected direction given that the variables were calculated in such a manner that the higher the score the more positive the area economic situation. The strongest statistically significant correlations were between the 'median home value' and 'median income' variables and between the 'percentage of the population 25 years and older who earned a high school diploma' and the 'percentage of census tract households above the poverty rate'

Table 4.2. Pearson Correlation Coefficients: ASPoH Variables, 2000 US Census

	% Employed	% Above Poverty Rate	Occupied Home Rate	Non-Crowded Rate	% Using Private Transport	% High School Diploma	% Employed Full-time	Median Income	% Home Ownership	Median Rent	Median Home Value
% Employed	1.0										
% Above Poverty Rate	0.547 *	1.0									
Occupied Home Rate	-0.036 *	-.006	1.0								
Non-Crowded Rate	0.347 *	0.521 *	-0.074 *	1.0							
% Using Private Transport	0.412 *	0.415 *	0.265 *	0.267 *	1.0						
% High School Diploma	0.461 *	0.737 *	-.029	0.627 *	0.225 *	1.0					
% Employed Full-time	0.178 *	0.118 *	0.498 *	-0.020	0.265 *	0.127 *	1.0				
Median Income	0.415 *	0.622 *	-.021	0.339 *	0.077 *	0.674 *	0.119 *	1.0			
% Home Ownership	0.391 *	0.617 *	-0.047 *	0.434 *	0.445 *	0.385 *	-0.147 *	0.459 *	1.0		
Median Rent	0.296 *	0.467 *	0.045 *	0.169 *	0.113 *	0.499 *	0.151 *	0.634 *	0.299 *	1.0	
Median Home Value	0.259 *	0.359 *	-.134 *	0.140 *	-.174 *	0.441 *	0.019	0.823 *	0.216 *	0.486 *	1.0
***= significant at .05 level**											

variables, at 0.823 and 0.737 respectively. Variables in which there appeared to be almost no correlation, -0.006, include the 'census tract occupied home rate' and the 'percentage of census tract households above the poverty rate' variables.

Principal Component Analyses Results

Three of the principal components had eigenvalues above the value of one (Table 4.3). This tells us that these three components account for more than one point of variance within the data. Although it is common practice to only retain those variables with an eigenvalue greater than one, others have also chosen to keep as many components as you need to have a cumulative amount of variance in the data accounted for. In these analyses, four principal components are retained. These components account for a total of 76.29% (range of 70-80 typically used) of the variance in the data.

Table 4.3. Eigenvalues of the correlation matrix, Principal Components Analyses

Principal Component	Eigenvalue	Difference	Proportion	Cumulative
1	4.4566	2.7578	0.4051	0.4051
2	1.6987	0.2600	0.1544	0.5596
3	1.4387	0.6404	0.1308	0.6904
4	0.7982	0.0410	0.0726	0.7629
5	0.7571	0.2519	0.0688	0.8318
6	0.5052	0.0735	0.0459	0.8777
7	0.4317	0.1092	0.0392	0.9169
8	0.3224	0.0267	0.0293	0.9463
9	0.2957	0.0909	0.0269	0.9731
10	0.2047	0.1141	0.0186	0.9918
11	0.0906		0.0082	1.0000

The two variables with the largest factor loadings for each of the components are presented as the description for the principal components (The magnitude of all contributing variables, both positive and negative, can be seen in Table 4.4).

The two variables representing principal component 1 are (1) the median household income and (2) the percent of households within the census tract that were above the poverty rate. The two variables representing principal component 2 are (1) Percent of occupied homes and (2) the percent of residents employed fulltime. The two variables representing principal component 3 are (1) Median home value and percent of home ownership. The two variables representing principal component 4 are (1) Percent of census tract residents who are employed and (2) the percent of census tract residents 25 years and older who are high school graduates.

Table 4.4. Factor Loadings for Principal Components Retained in Further Analyses

Census Tract Level Variables	Principal Component 1	Principal Component 2	Principal Component 3	Principal Component 4
Pct Employed	0.283	-0.114	-0.105	0.493
Pct Above Poverty Rate	0.402	-0.054	-0.166	-0.036
Occupied Home Rate	0.007	-0.582	0.243	-0.138
Non-Crowded Rate	0.274	-0.004	-0.393	-0.616
Pct Private Transport Use	-0.194	0.503	0.357	-0.236
Pct High School Diploma	-0.393	-0.092	-0.061	0.409
Pct Employed Full-time	-0.093	0.537	-0.386	0.158
Median Income	0.424	0.132	0.223	0.015
Pct Home Ownership	-0.308	-0.034	0.384	-0.249
Median Rent	0.319	0.047	0.328	0.199
Median Home Value	0.324	0.272	0.403	0.084

Throughout this dissertation, further definition of the ASPoH-1, ASPoH-2, ASPoH-3, and ASPoH-4 variables can be found in Appendix B: Definition of Study Variables.

Part II. Research Question One

*Question***:** Are Black-White disparities in stroke mortality elevated in those areas of low socioeconomic status?

Hypothesis: Black-White disparities in stroke mortality will be greatest at lower

levels of ASPoH.

Descriptive Statistics

Numerator Data: Stroke Death Counts

Within the 1998-2002 time period, there was a total of 43,945 stroke deaths for Florida residents aged 35 years and older. These deaths were distributed across 3064 census tracts. There were no stroke deaths reported for the study time period for the remaining ninety census tracts. The distribution of the stroke deaths by race and gender are as follows: Black males 5%, Black females 7%, NH-White males 36%, NH-White Females 52%. The median age of the Black decedents was 62 years, a slightly younger age than that of White decedents, 68 years.

Stroke data included in the subsequent analyses were restricted to those decedents between the age of 35 and 74 years. These remaining 10,799 deaths were distributed across these 3064 census tracts. Twenty-four percent of these decedents are Black Americans (Hispanic and non-Hispanic) and 76% are White Americans (non-Hispanic). Males constituted the majority of the decedents with 52.5 %. The effect of excluding deaths in the oldest age groups (75+ years) was to increase the percentage of deaths represented by Black males. This occurred because Blacks, on average, die earlier; therefore, compared to non-Hispanic White decedents, fewer Black decedents were excluded when age restrictions (35-74 years) were utilized. The percentage of female decedents decreased because females in the oldest age group constituted a large percentage of the original subject pool. When the age restriction is introduced, the percent contribution of females to the stroke death count decreases.

As expected, decedents in the oldest age group, 65-74 years, made up the greatest

proportion of stroke deaths (Table 4.5). The proportion of stroke deaths decreases with younger age groups. In each of the younger age groups (35-44, 45-54 and 55-64), Blacks consistently contributed a higher proportion of stroke deaths than did White decedents. A particularly striking finding was that the proportion of Black males in the 45-54 year age group was almost twice that of White males, at 22.2% and 11.8%, respectively. In the 35-44 year age group, the proportion of Black female stroke decedents was more than twice that of White female decedents in the same age group, 11% and 4.2%, respectively.

Table 4.5. Percentage of Stroke Deaths by Race-Sex-Age group, Florida 1998-2002

	35-44 years	**45-54 years**	**55-64 years**	**65-74 years**	**Total**
Black Males (N= 1326)	7.7	22.2	30.0	40.1	100 **(12.3%)***
Black Females (N= 1265)	11.0	17.9	26.9	44.2	**100 (11.7%)***
NH-White Males (N= 4345)	4.8	11.8	22.0	61.4	100 **(40.2%)***
NH-White Females (N= 3863)	4.2	10.3	20.9	64.5	100 **(35.8%)***
Total (N= 10,799)	**5.7**	**13.2**	**23.2**	**57.9**	100 **(100%)***
*** = % of total study population**					

Denominator Data: Florida Population Counts

The median age for Florida residents was 38.7 years. Median age for White residents was slightly higher than that of Black residents, 42.0 and 29.0 respectively. A large proportion of these retirees are White, resulting in a higher median age value for this group. There is an equal distribution of males and females within the State of Florida. Black residents were less likely than White residents to have ever been married, 16.8% and 6.9 % respectively. The widow/divorce rate was very similar for both race

groups. Black residents were less likely than White residents to have an education above the high school graduate level and were more likely to have not completed high school.

To be retained in the study for further analysis, the individual census tract must have a population count of at least one within each race-gender-10yr age group category. This resulted in the following population distribution: Black males 7%, Black females 9%, NH-White Males 40%, NH-White females 44% (Table 4.6). These proportions are

Table 4.6. 2000 US Census Population counts by Race-Sex-Age group

2156 Census Tracts	**35-44 years**	**45-54 years**	**55-64 years**	**65-74 years**	**Total**
Black Males	155,437	109,076	61,739	38,261	**364,513**
Black Females	173,975	124,368	72,719	50,280	**421,342**
NH-White Males	597,962	515,601	377,336	351,721	**1,842,620**
NH-White Females	584,177	526,671	414,975	405,504	**1,931,327**
Total	**1,511,551**	**1,275,716**	**926,769**	**845,766**	**4,559,802**

similar to those observed when all of the original census tracts are included in the study. When exclusion / inclusion criteria are applied in the selection of census tracts for this study, only 2156 of the original 3154 census tracts remain in the study. A total population count of 4,559,802 was distributed across these 2156 census tracts (Table 4.6). These population counts were multiplied by 5 (years) before being utilized as denominators in the calculation of all study rates.

The 35-44 year age group contributed the greatest percentage of person years to the study at 33.1% (Table 4.7). Compared to the Non-Hispanic White population, a higher proportion of the Black population made up the younger age groups. These data

reflect the age distribution of Blacks within the State of Florida. In the State of Florida, Black residents are younger than white residents with median ages of 29.0 and 42.0 years, respectively. This trend was also seen in the study data. For example, the 35-44 year age group, constituted 42.6% of the Black male study population. In comparison to White males in the same age group at 32.4%, Black males have a younger age distribution than do White males. The same trend was also seen for females aged 35-44 years. The percentages for Black females and White females are 41.3% and 30.2%, respectively. The age distributions are similar between the race-sex groups within the 45-54 and 55-64 year age groups. The percentage of the White residents in the oldest age group, 65-74 years, is almost double the percentage of Black residents falling into this age category. Within the State of Florida, White residents tend to live longer than do Black residents. Overall, Black residents comprised 17.2% of the total study population, with White residents making up 82.8% of the study population.

Table 4.7. 2000 US Census population percent distribution by race-sex-age group

	35-44 years	**45-54 years**	**55-64 years**	**65-74 years**	**Total**
Black Males	42.6	29.9	16.9	10.5	100 **(8.0%)***
Black Females	41.3	29.5	17.3	11.9	100 **(9.2%)***
NH-White Males	32.4	28.0	20.5	19.1	100 **(40.4%)***
NH-White Females	30.2	27.3	21.5	21.0	100 **(42.4%)***
Total	**33.1**	**28.0**	**20.3**	**18.5**	100 **(100%)***
*** = percent of total study population (summed across 2156 census tracts)**					

Stroke Mortality Rates

Race-Sex-10 year Age Group Specific Stroke Mortality Rates

When the age-group specific stroke death rates by race and gender are examined, the trends are consistent with what is expected (Table 4.8). For each of the race-sex groups, the 35-44 year age-group has the lowest stroke death rate. The rates increase across successive 10-yr age-groups. The highest stroke death rates are observed for those in the 65-74 year age group. Across racial groups, males typically have the higher stroke death rates.

Table 4.8. Race-sex 10-year age group specific stroke mortality rates*: Census tract

N=2156	**35-44 yrs**	**45-54 yrs**	**55-64 yrs**	**65-74 yrs**
Black Males	8.68	47.26	116.76	273.42
Black Females	12.85	35.47	73.21	211.56
NH-White Males	6.66	20.43	46.52	137.79
NH-White Females	5.43	15.91	33.52	114.71
*Rates per 100,000				

In each of the age groups Blacks had higher stroke mortality rates than did Whites. In the 35-44 year age group, Black females had the highest stroke death rate at 12.85 per 100,000. This slightly higher rate for Black females, compared to Black males, at the younger age group is in accord with published data. In each of the succeeding 10 year age groups, Black males consistently had the highest rates, while Black females consistently have the second highest rates. White males and females experienced half the stroke death rate of their Black counterparts in each respective 10-year age categories. For males, the largest stroke mortality rate difference between Blacks and Whites is seen in the 55-64 year age group. In this age group, the Black male stroke death rate is 2.51 times higher than that of White males. For females, the largest stroke mortality rate

difference between Blacks and Whites is seen in the youngest age group, 35-44. In this age-group the Black female stroke death rate is 2.36 times higher than that of White females. For males, the smallest stroke mortality rate difference between Blacks and Whites is seen in the 35-44 year age group. In the 35-44 year age group Black males have a stroke death rate that is 1.30 times higher than that of White males. For females, the smallest stroke mortality rate difference between Blacks and Whites is seen in the 65-74 year age group. In the 65-74 year age group, Black females have a stroke death rate that is 1.84 times that of White females.

Race and Sex Specific Age-Adjusted Stroke Mortality Rates: Census Tract Level

Census tract level average annual age-adjusted stroke mortality rates for those aged 35-74 years were calculated for each of the four race-sex groups (Table 4.9). On average, Black stroke mortality rates were twice that of White residents. Black males and females experienced the highest average stroke death rate at 79.70 per 100,000 and 60.29 per 100,000, respectively. Non-Hispanic White males and females experienced lower rates of 37.63 and 29.97 per 100,000, respectively. The variability in these census tract level death rates is strikingly large as seen in the standard deviation values (Table 4.9). These calculated stroke death rates are considerably lower than expected given published US and state level stroke death rates. This finding is likely due to the comparatively smaller population size of census tracts (compared to US and state populations) and consequently lower number of stroke deaths (by race and sex groups) within each census tract. In instances of inadequate population and stroke death counts, calculations of race-sex specific stroke death rates would produce unstable results. Another possible contributor to these finding is the age-restrictions imposed by these study analyses.

These restrictions lower the number of study subjects included in the analyses.

Table 4.9. Race and Sex Specific Age-Adjusted (35-74 yrs) Stroke Mortality Rates*, Florida 1998-2002

N= 2156 Census Tracts	**Mean**	**Median**	**Std Dev**	**Min**	**Max**
Black Male	79.70	0.00	253.91	0.00	3831.94
Black Female	60.29	0.00	222.53	0.00	5957.46
Non Hispanic White Male	37.63	22.19	75.87	0.00	2157.04
Non Hispanic White Female	29.97	17.44	60.66	0.00	1277.31
* rates per 100,000					

Study Outcome Scores

Sex specific racial disparity measures, rate ratio and rate difference measures, were calculated at the census tract level (see Table 4.10). On average, Black stroke death rates were twice that of White residents, with average ratios of 2.28 and 2.02 for males and females respectively. The absolute racial difference scores were 42.07 for males and 30.33 for females. As was seen with the race-gender specific age-adjusted rates, there is tremendous variability within the census tract level disparity scores.

Table 4.10. Summary statistics for Black-White stroke mortality disparity measures

N=2156 Census Tracts	**Mean**	**Median**	**Std Dev**	**Min**	**Max**
Male Black-White Ratio	2.28	0.00	10.30	0.00	229.50
Female Black-White Ratio	2.04	0.00	10.33	0.00	245.77
Male Black-White Difference	42.07	-8.86	264.40	-1900.75	3796.87
Female Black-White Difference	30.33	-4.45	229.88	-1277.31	5906.17
Male Percent Difference	173.37	-100.00	1194.00	-100.00	22850.00
Female Percent Difference	147.15	-100.00	1223.00	-100.00	24476.98

Area Social Predictors of Health (ASPoH) Quartiles

Descriptive Statistics

Before progressing to census tract level analyses of the ASPoH and racial disparities in stroke mortality relationship, this potential association was investigated

utilizing ASPoH categories as indicators of disparity magnitude. The range of ASPoH values within each quartile are presented in Table 4.11. For example, if a census tract had a calculated value of -10 for the ASPoH1 variable, that census tract would be included in the First Quartile. If that census tract had a calculated value of 1.1 for the ASPoH1 variable, that census tract would be included in the Third Quartile, and so on. Each quartile contains either 788 or 789 census tracts (3154 total census tracts).

Table 4.11. Interquartile range of calculated census tract values for each ASPoH variable, 2000 US Census

Range of Values for each ASPoH Variable				
N=3154	**First Quartile**	**Second Quartile**	**Third Quartile**	**Fourth Quartile**
ASPoH1	-11.08 to -1.07	-1.07 to 0.19	0.19 to 1.35	1.35 to 6.38
ASPoH2	-7.26 to -0.82	-0.82 to -0.28	-0.28 to 0.49	0.49 to 9.46
ASPoH3	-8.37 to -0.78	-0.78 to 0.017	0.017 to 0.73	0.73 to 4.68
ASPoH4	-13.53 to -0.37	-0.37 to -0.003	-0.003 to 0.35	0.35 to 8.34

All race-sex specific stroke mortality rates are highest in the lowest quartile of the ASPoH-1 variable (see Table 4.12). The most favorable (lowest) stroke mortality rates occurred in the most affluent area as represented by the Fourth Quartile. With the exception of the Percent Difference scores, the remaining disparity measures followed a similar pattern of intensity. The data showed that the male Black-White disparity in stroke mortality was more pronounced for the lowest ASPoH-1 quartile which represented the most deprived area (Table 4.13). Results were inconsistent for the female disparity scores.

Table 4.12. Mean Race-sex specific stroke mortality by Quartile: ASPoH-1

ASPoH-1				
Quartile	**Black Male Age Adjusted Death rate**	**Black Female Age Adjusted Death rate**	**NH-White Male Age Adjusted Death rate**	**NH-White Female Age Adjusted Death rate**
1 (low)	89.629	66.663	42.264	32.804
2	69.120	56.668	31.675	25.234
3	55.444	44.450	26.761	20.673
4 (high)	38.715	37.588	18.890	15.329

Table 4.13. Mean Black-White Stroke Mortality Disparity by Quartile: ASPoH-1

ASPoH-1						
Quartile	**Male Black-White Ratio**	**Female Black-White Ratio**	**Male Black-White Difference**	**Female Black-White Difference**	**Male Percent Difference**	**Female Percent Difference**
1 (low)	2.121	2.032	47.365	33.859	112.070	103.218
2	2.182	2.246	37.446	31.435	118.219	124.574
3	2.072	2.150	28.682	23.777	107.179	115.015
4 (high)	2.049	2.452	19.825	22.258	104.954	145.200

Similar results were obtained for Non Hispanic Whites when the effect of the ASPoH-2 variable was examined, as seen in Table 4.14. For Blacks, however, the influence of the ASPoH-2 variable was in contrast to ASPoH-1 effects. The most favorable stroke mortality rates for Blacks were observed for the lowest quartile of the ASPoH-2 variable, which represents affluent areas, and the least favorable rates occurred in the highest quartile areas, which represents deprived areas. Accordingly, Table 4.15 demonstrates that each of the disparity scores is highest in those areas of affluence.

Table 4.14. Mean Race-sex specific stroke mortality by Quartile: ASPoH-2

ASPoH-2				
Quartile	**Black Male Age Adjusted Death rate**	**Black Female Age Adjusted Death rate**	**NH-White Male Age Adjusted Death rate**	**NH-White Female Age Adjusted Death rate**
1 (high)	60.121	47.680	33.042	25.966
2	73.755	53.520	31.012	24.634
3	79.590	62.809	28.629	23.443
4 (low)	92.879	73.520	21.636	16.529

Table 4.15. Mean Black-White Stroke Mortality Disparity by Quartile: ASPoH-2

ASPoH-2						
Quartile	**Male Black-White Ratio**	**Female Black-White Ratio**	**Male Black-White Difference**	**Female Black-White Difference**	**Male Percent Difference**	**Female Percent Difference**
1 (high)	1.819	1.836	27.079	21.714	81.953	83.623
2	2.378	2.173	42.743	28.886	137.828	117.263
3	2.780	2.679	50.961	39.366	178.006	167.920
4 (low)	4.293	4.448	71.243	56.990	329.273	344.783

The race-sex specific stroke mortality rates scores vary slightly in magnitude across the quartiles for the ASPoH-3 measure (Table 4.16). Consequently, the magnitude of this measure has limited influence on Black-White disparities in stroke mortality (Table 4.17).

Table 4.16. Mean race-sex specific stroke mortality by Quartile: ASPoH-3

ASPoH-3				
Quartile	**Black Male Age Adjusted Death rate**	**Black Female Age Adjusted Death rate**	**NH-White Male Age Adjusted Death rate**	**NH-White Female Age Adjusted Death rate**
1	69.895	60.231	27.120	21.957
2	86.859	59.958	28.990	23.262
3	79.929	56.064	28.760	21.772
4	67.704	60.565	25.226	19.763

Table 4.17. Mean Black-White Stroke Mortality Disparity by Quartile: ASPoH-3

ASPoH-3						
Quartile	**Male Black-White Ratio**	**Female Black-White Ratio**	**Male Black-White Difference**	**Female Black-White Difference**	**Male Percent Difference**	**Female Percent Difference**
1	2.577	2.743	42.775	38.275	157.729	174.319
2	2.996	2.577	57.869	36.696	199.617	157.750
3	2.779	2.575	51.169	34.292	177.921	157.504
4	2.684	3.064	42.479	40.802	168.394	206.458

Table 4.18. Mean race-sex specific stroke mortality by Quartile: ASPoH-4

ASPoH-4				
Quartile	**Black Male Age Adjusted Death rate**	**Black Female Age Adjusted Death rate**	**NH-White Male Age Adjusted Death rate**	**NH-White Female Age Adjusted Death rate**
1	88.846	63.895	31.325	25.238
2	81.718	63.131	28.899	23.096
3	68.667	53.039	25.290	20.026
4	68.743	56.243	26.176	20.143

The ASPoH-4 measure has similar influences as the ASPoH-1 measure. For Black and Non Hispanic White residents, the stroke mortality rates are lowest in the most affluent areas and highest in the most deprived areas. Table 4.18 demonstrates that the impact of the ASPoH-4 measure is slightly stronger for Black residents as compared to Non Hispanic White residents. In Table 4.19 we see that the magnitude of the disparities scores shows only slight variation across the ASPoH-4 categories.

Table 4.19. Mean Black-White Stroke Mortality Disparity by Quartile: ASPoH-4

ASPoH-4						
Quartile	**Male Black-White Ratio**	**Female Black-White Ratio**	**Male Black-White Difference**	**Female Black-White Difference**	**Male Percent Difference**	**Female Percent Difference**
1	2.836	2.532	57.521	38.657	183.627	153.169
2	2.828	2.733	52.819	40.035	182.772	173.344
3	2.715	2.648	43.377	33.013	171.516	164.854
4	2.626	2.792	42.568	36.100	162.622	179.221

Regression Findings: Census Tract Level Analyses

The multiple regression model which was utilized to test the predictive capability of the ASPoH variables (4 principal components) at the census tract level is the following: Racial_Disparity_Score = $ß_0 + ß_1$ASPoH1 + $ß_2$ASPoH2 + $ß_3$ASPoH3 + $ß_4$ASPoH4 + ε. This regression model was used in six instances, once for each of the six disparity outcome scores.

The ASPoH variables were shown to be significant predictors of the Female Ratio (Table 4.21) but were not significant predictors of the Male Ratio outcome (Tables 4.20).

Table 4.20. Regression model which measured the association between the male Black-White stroke mortality ratio and the "Area Social Predictors of Health' variables

Male Ratio F Value: 2.25 **	**Parameter Estimate**	**Standard Error**	**t-value**	**Pr> |t|**
ASPoH-1	0.168	0.135	1.24	0.2143
ASPoH-2	0.350	0.267	1.31	0.1898
ASPoH-3	-0.453	0.218	-2.07	0.0381
ASPoH-4	0.468	0.344	1.36	0.1744
** : not statistically significant, p value > 0.05				N=1909 Census Tracts

The F-Values were 2.25 and 2.38 for the Male ratio and Female ratio, respectively. The model accounted for 0.5% of the variance in the Male Ratio score and

0.5% of the variance in the Female Ratio score. ASPoH-2 was the only significant independent predictor of the Female ratio, with the Female ratio increasing 0.557 points with every one unit increase in the ASPoH-2 variable.

Table 4.21. Regression model which measured the association between the female Black-White stroke mortality ratio and the "Area Social Predictors of Health' variables

Female Ratio F Value: 2.38*	**Parameter Estimate**	**Standard Error**	**t-value**	**Pr> |t|**
ASPoH-1	0.199	0.136	1.46	0.1442
ASPoH-2	0.557	0.364	2.10	0.0355
ASPoH-3	-0.440	0.219	-1.84	0.0665
ASPoH-4	-0.023	0.345	-0.07	0.9463
* : statistically significant, p value ≤ 0.05				N=1894 Census Tracts

The ASPoH variables were not shown to be significant predictors of the Male or Female Difference scores (Tables 4.22 and 4.23). The F-Values were 1.90 and 1.10, respectively. The amount of variance accounted for by the models was minimal. The model accounted for 0.35% of the variance in the Male Difference score and 0.20% of the variance in the Female Difference score.

Table 4.22. Regression model which measured the association between the male Black-White stroke mortality difference score and the "Area Social Predictors of Health' variables

Male Diff F Value: 1.90 **	**Parameter Estimate**	**Standard Error**	**t-value**	**Pr> |t|**
ASPoH-1	-2.860	3.049	-0.94	0.3484
ASPoH-2	4.535	6.426	0.71	0.4805
ASPoH-3	-10.037	5.250	-1.91	0.0560
ASPoH-4	-12.695	7.969	-1.59	0.1113
** : not statistically significant, p value > 0.05				N= 2156 Census Tracts

Table 4.23. Regression model which measured the association between the female Black-White stroke mortality difference score and the "Area Social Predictors of Health' variables

Female Diff F Value: 1.10 **	**Parameter Estimate**	**Standard Error**	**t-value**	**Pr> |t|**
ASPoH-1	3.183	2.653	1.20	0.2305
ASPoH-2	2.965	5.591	0.53	0.5960
ASPoH-3	-4.479	4.568	-0.98	0.3269
ASPoH-4	-7.281	6.934	-1.05	0.2938
** : not statistically significant, p value > 0.05				N= 2156 Census Tracts

The ASPoH variables were not shown to be significant predictors of the Male or Female Percent Difference scores (Tables 4.24 and 4.25). The F-Values were 1.72 and 2.02, respectively. The amount of variance accounted for by the models was minimal. The model accounted for 0.49% of the variance in the Male Percent Difference (MPD) score and 0.60% of the variance in the Female Percent Difference (FPD) score.

Table 4.24. Regression model which measured the association between the male Black-White stroke mortality percent difference score and the "Area Social Predictors of Health' variables

MPD F Value: 1.72	**Parameter Estimate**	**Standard Error**	**t-value**	**Pr> |t|**
ASPoH-1	26.205	19.213	1.36	0.1728
ASPoH-2	40.643	36.506	1.11	0.2658
ASPoH-3	-37.522	30.619	-1.23	0.2206
ASPoH-4	77.139	50.574	1.53	0.1274
** : not statistically significant, p value > 0.05				N=1415 Census Tracts

Table 4.25. Regression model which measured the association between the female Black-White stroke mortality percent difference score and the "Area Social Predictors of Health' variables

FPD F Value: 2.02	**Parameter Estimate**	**Standard Error**	**t-value**	**Pr> |t|**
ASPoH-1	35.124	20.428	1.72	0.0858
ASPoH-2	81.423	38.943	2.09	0.0367
ASPoH-3	-33.890	31.935	-1.06	0.2888
ASPoH-4	-0.045	53.129	-0.00	0.9993
** : not statistically significant, p value > 0.05				N= 1346 Census Tracts

Restricted Subset of Census Tracts

Descriptive Statistics

Gender specific racial disparity measures, rate ratio and rate difference measures, were calculated at the census tract level (Table 4.10). On average, Black stroke death rates were twice that of White residents, with average ratios of 2.28 and 2.02 for males and females respectively. The absolute racial difference scores were 42 for males and 30 for females. As was seen with the race-gender specific age-adjusted rates, there is tremendous variability within the census tract level disparity scores. This is likely due to vast number of census tract with zero rates for black males and females. Dividing or subtracting by zeros (Black age-adjusted rates) leads to an attenuation of the calculated racial disparity scores. When these census tracts with zero rates for either Blacks or Whites are excluded, the calculated disparity scores are much larger (Table 4.26).

Table 4.26. Descriptive Statistics for Black-White stroke mortality disparity measures: Restricted Subset

	Mean	Median	Std Dev	Min	Max
Male Black-White Ratio	10.656	3.778	21.727	0.040	229.500
Female Black-White Ratio	10.299	3.314	23.315	0.075	245.770
Male Black-White Difference	249.572	117.880	495.838	-1900.755	3796.875
Female Black-White Difference	189.466	84.669	484.873	-662.725	5906.174
Male Percent Difference	965.629	277.778	2173.000	-95.952	22850.000
Female Percent Difference	929.915	231.434	2332.000	-92.470	24476.980

Multiple Regression Models

Additional analyses, utilizing the previous regression model (Disparity Score = $ß_0 + ß_1\text{Prin1} + ß_2\text{Prin2} + ß_3\text{Prin3} + ß_4\text{Prin4} + \varepsilon$), were performed only on those census tracts with non-zero age-adjusted rates for each of the race groups. This

restriction results in the utilization of only 363 and 323 census tracts in the regression analyses for male and female Black-White disparity scores, respectively.

The model with male ratio as the outcome was statistically significant with an F-Value of 17.78 and 17% of the variance accounted for by the model (Table 4.27). Both ASPoH-1 and 2 were statistically significant predictors of the Black-White male ratio score. With a one point increase in the ASPoH-1 score, the male ratio increases by 5.59. With a one point increase in the ASPoH-2 score, the male ratio increases 7.21.

Table 4.27. Regression model which measured the association between the male Black-White stroke mortality ratio and the 'Area Social Predictors of Health' variables, Restricted subset of census tracts

Male Ratio F Value: 17.78*	Parameter Estimate	Standard Error	t-value	Pr> |t|
ASPoH-1	5.588	0.705	7.92	<.0001
ASPoH-2	7.217	1.482	4.87	<.0001
ASPoH-3	-0.819	1.027	-0.80	0.4259
ASPoH-4	3.262	1.428	2.28	0.0230
* : statistically significant, p value ≤ 0.05				N=363 Census Tracts

The model predicting female ratio was statistically significant with an F-Value of 16.98 and 18% of the variance accounted for by the model (Table 4.28). Both ASPoH-1 and 2 were statistically significant predictors of the Black-White female ratio score. With a one point increase in the ASPoH-1 score, the female ratio increases by 6.18. With a one point increase in the ASPoH-2 score, the female ratio increases 10.12.

Table 4.28. Regression model which measured the association between the female Black-White stroke mortality ratio and the 'Area Social Predictors of Health' variables, Restricted subset of census tracts

Female Ratio F Value: 16.98*	Parameter Estimate	Standard Error	t-value	Pr> |t|
ASPoH-1	6.177	0.807	7.65	<.0001
ASPoH-2	10.125	1.711	5.92	<.0001
ASPoH-3	0.039	1.189	0.03	0.9737
ASPoH-4	2.512	1.744	1.44	0.1506
* : statistically significant, p value ≤ 0.05				N=323 Census Tracts

The ASPoH variables were significant predictors of the Male difference score (Table 4.29). The model was statistically significant with an F-Value of 12.42 and 12% of the variance accounted for by the model. ASPoH-1 is a statistically significant predictor of the Black-White male difference score. With a one point increase in the ASPoH-1 score, the male difference score increases by 107.53.

Table 4.29. Regression model which measured the association between the male Black-White stroke mortality difference score and the 'Area Social Predictors of Health' variables, Restricted subset of census tracts

Male Diff F Value: 12.42*	Parameter Estimate	Standard Error	t-value	Pr> |t|
ASPoH-1	107.531	16.511	6.51	<.0001
ASPoH-2	57.404	34.692	1.65	0.0989
ASPoH-3	-31.478	24.058	-1.31	0.1916
ASPoH-4	41.628	33.444	1.24	0.2141
* : statistically significant, p value ≤ 0.05				N=363 Census Tracts

The ASPoH variables were significant predictors of the Female difference score (Table 4.30). The model was statistically significant with an F-Value of 10.74 and 12% of the variance accounted for by the model. ASPoH-1 is a statistically significant predictor of the Black-White female difference score. With a one point increase in the ASPoH-1 score, the female difference score increases by 103.74.

Table 4.30. Regression model which measured the association between the female Black-White stroke mortality difference score and the 'Area Social Predictors of Health' variables, Restricted subset of census tracts

Female Diff F Value: 10.74*	Parameter Estimate	Standard Error	t-value	Pr> |t|
ASPoH-1	103.742	17.360	5.98	<.0001
ASPoH-2	50.118	36.799	1.36	0.1742
ASPoH-3	-1.510	25.569	-0.06	0.9529
ASPoH-4	-4.686	37.498	-0.12	0.9006
* : statistically significant, p value ≤ 0.05				N=323 Census Tracts

The ASPoH variables were significant predictors of the Male percent difference

score (Table 4.31). The model was statistically significant with an F-Value of 17.78 and 16.6% of the variance accounted for by the model. ASPoH-1 is a statistically significant predictor of the Black-White male percent difference score. With a one point increase in the ASPoH-1score, the male percent difference score increases by 558. ASPoH-2 is a statistically significant predictor of the Black-White male percent difference score. With a one point increase in the ASPoH-2 score, the male percent difference score increases by 721.

Table 4.31. Regression model which measured the association between the male Black-White stroke mortality percent difference score and the 'Area Social Predictors of Health' variables, Restricted subset of census tracts

F Value: 17.78*	Parameter Estimate	Standard Error	t-value	Pr> |t|
ASPoH-1	558.846	70.521	7.92	<.0001
ASPoH-2	721.666	148.173	4.87	<.0001
ASPoH-3	-81.908	102.751	-0.80	0.4259
ASPoH-4	326.172	142.842	2.28	0.0230
* : statistically significant, p value ≤ 0.05				N=363 Census Tracts

The ASPoH variables were significant predictors of the Female percent difference score (Table 4.32). The model was statistically significant with an F-Value of 16.98 and 17.6 % of the variance accounted for by the model. ASPoH-1 is a statistically significant predictor of the Black-White female percent difference score. With a one point increase in the ASPoH-1 score, the female percent difference score increases by 617. ASPoH-2 is a statistically significant predictor of the Black-White female percent difference score. With a one point increase in the ASPoH-2 score, the female percent difference score increases by 1012.

Table 4.32. Regression model which measured the association between the female Black-White stroke mortality percent difference score and the 'Area Social Predictors of Health' variables, Restricted subset of census tracts

F Value: 16.98*	Parameter Estimate	Standard Error	t-value	Pr> |t|
ASPoH-1	617.713	80.735	7.65	<.0001
ASPoH-2	1012.470	171.132	5.92	<.0001
ASPoH-3	3.928	118.908	0.03	0.9737
ASPoH-4	251.251	174.385	1.44	0.1506
* : statistically significant, p value ≤ 0.05				N=323 Census Tracts

Summary of Findings

ASPoH categories (quartiles) were created to assess the relationship between level of economic advantage/disadvantage and magnitude of Black-White disparities in stroke mortality. All race-sex-specific age-adjusted rates and disparity scores were lowest in the ASPoH-1 quartile (quartile 4) representing the highest values for economic advantage. In the assessment of the ASPoH-2 variable, Black males and females in the most economically advantaged census tracts experienced the lowest stroke mortality rates. This resulted in disparity scores being the greatest in these economically disadvantaged census tracts. Race-sex specific stroke mortality rates and disparity scores were very similar across quartiles for the ASPoH-3 variable and for the ASPoH-4 variable. No inferences can be made regarding the impact of the ASPoH-3 and ASPoH-4 variables on the magnitude of Black-White disparities in stroke mortality.

Multiple regression analysis was utilized to assess the predictive ability of the ASPoH variables on Black-White disparities in stroke mortality. Study results showed elevated age-adjusted stroke mortality rates for Black Floridians compared to Non-Hispanic White Floridians. For females, the Black-White ratio score was associated with significant changes in levels of the ASPoH variables. Increases in the magnitude of

ASPoH-1 and ASPoH-2, which accounted for the highest percentage of variance in the census tract level social and economic measures, were associated with higher Black-White stroke mortality ratios. Contrastingly, increases in the magnitude of the ASPoH-3 and ASPoH-4 variables, were associated with decreases in the Black-White stroke mortality ratios. These decreases in the magnitude of Black-White stroke mortality ratios in those areas of economic advantage support the study hypothesis which states that Black-White disparities in stroke mortality will be greatest at lower levels (magnitudes) of the ASPoH variables. None of the remaining multiple regression models testing the predictive ability of the ASPoH variables on Black-White stroke mortality (as measured by the disparity scores) were statistically significant. When regression analyses were restricted to a subset of these same census tracts, all of the regression models were found to be statistically significant. Increases in the ASPoH-1 and ASPoH-2 variables were associated with increases in the Black-White ratio score, difference score and percent difference score for both males and females. Inconsistent results were obtained for the ASPoH-3 and ASPoH-4 variables. Additionally, the hypothesis was only supported when the restricted analyses were performed accessing the predictability of the ASPoH-2 variable. In this instance, the Black-White disparity scores decreased with elevations in the ASPoH scores. The hypothesis was not supported when accessing the predictability of either of the remaining ASPoH variables.

Part III. Research Question Two

Question: Are higher levels of Black-White disparities in stroke mortality associated with low levels of social class?

Hypothesis: Black-White disparities in stroke mortality will be greatest for those

in the lowest social class group.

Descriptive Statistics

Social Class Groups Population Counts

Educational attainment data (used as a proxy measure for social class) were reported for a total of 3138 out of the 3154 Florida census tracts. The census reported educational attainment data for the following race, gender and age groups: Black males aged 35-44, Black males aged 45-64, and Black males 65 and up. This educational attainment information was presented for the same age-groups for Black females, Non Hispanic White males and Non Hispanic White females, resulting in educational attainment data for a total of twelve race-sex-age groups.

Population counts for each of the social class race-sex-age-groups are multiplied by 5(years) to estimate the population total for the 1998-2002 5-year study period. As a result, a total population of 31,884,280 was contributed to the study by all Florida residents 35 years of age and older (Table 4.33). Across all race-sex-groups, the 45-64 year age-group contributes the highest population percentage at 39.51%. The 35-44 year age-group and the 65 years and up age group contribute 25.42 and 35.07 percent of the total person years, respectively. NH-White females and males contributed the highest percentage of person years to the study, with approximately 47.47% and 41.76%, respectively. Black males contributed 4.85% of the population count and Black females contributed 5.91%% of the total population to the study. Within each race-sex group, the population distribution is slightly different for NH-White females. For NH-White females, the 65+ year age-group contributes the largest percentage to the population count (39.32%). For each of the remaining race-sex groups, the 45-64 year age-groups

contribute the largest percentages to the population counts. For Black males and Black females, the smallest percentage of the population is contributed by the 65 years and older age group. For the NH-White females and males, the smallest percentage of the population is contributed by the 35-44 year age group.

Table 4.33. 2000 US Census Percent Population by race, sex and age group, Florida, All educational attainment groups

	Age-Group			
	35-44	**45-64**	**65_up**	**Total**
Black Males N=1,547,955	39.67	43.84	16.49	4.85 (100)
Black Females N=1,885,735	37.10	41.80	21.10	5.91 (100)
NH-White Males N=13,316,065	25.66	39.95	34.39	41.76 (100)
NH-White Females N=15,134,525	22.30	38.38	39.32	47.47 (100)
Total	25.42	39.51	35.07	100%

Almost thirty-four percent of the total population contributed by Black males belonged to Social Class 3 category (Table 4.34). The second highest population percentage is contributed by Black males in Social Class 4 category at 25.01% (next to the last social class category). Only 7.94% of Black male population belonged to the Social Class 1 category (the highest social class group). Black males in the social class 1 category contributed the smallest percentage to the Black male population count.

Table 4.34. Black male population count by social class and age-groups (35+years), 2000 US Census, Florida population multiplied by 5 years

	Age-Group			
	35-44	**45-64**	**65+**	**Total**
Social Class 1	47,610	59,595	15,670	122,875 (7.94%)
Social Class 2	132,415	122,635	21,330	276,380 (17.85%)
Social Class 3	250,030	221,145	49,130	520,305 (33.61%)
Social Class 4	150,150	174,175	62,885	387,210 (25.01%)
Social Class 5	33,835	101,090	106,260	241,185 (15.58%)
Total	614,040	678,640	255,275	1,547,955 (100%)

Thirty-two percent of the total population contributed by Black females belonged to Social Class 3 category (Table 4.35). The second highest percentage of the population count is contributed by Black females in Social Class 4 category (23.88%). Only 9.79% of Black female population belonged to the Social Class 1 category (the highest social class group). The social class 1 category contributed the least to the population count.

Table 4.35. Black female population count by social class and age-group (35+years), 2000 US Census, Florida population multiplied by 5 years

	Age-Group			
	35-44	**45-64**	**65+**	**Total**
Social Class 1	72,980	84,355	27,370	184,705 (9.79%)
Social Class 2	199,785	160,285	30,630	390,700 (20.72%)
Social Class 3	252,615	262,505	86,355	601,475 (31.90%)
Social Class 4	144,395	193,710	112,155	450,260 (23.88%)
Social Class 5	29,920	87,345	141,330	258,595 (13.71%)
Total	699,695	788,200	397,840	1,885,735 (100%)

Almost thirty-one percent of the total population contributed by NH-White males belonged to Social Class 3 category (Table 4.36). The second highest percentage of person years is contributed by NH-White males in Social Class 2 category at 28.03%. This is in contrast to Black males, with Social Class 4 as the second highest percent of the population contributed. 27.48% of NH-White male population belonged to the Social

Table 4.36. Non-Hispanic White male population count by social class and age-group (35+years), 2000 US Census, Florida population multiplied by 5 years

	Age-Group			
	35-44	**45-64**	**65+**	**Total**
Social Class 1	867,335	1,635,445	1,156,910	3,659,690 (27.48%)
Social Class 2	1,030,275	1,613,055	1,089,480	3,732,810 (28.03%)
Social Class 3	1,149,720	1,531,070	1,425,735	4,106,525 (30.84%)
Social Class 4	316,010	395,015	593,085	1,304,110 (9.79%)
Social Class 5	53,255	145,495	314,180	512,930 (3.85%)
Total	3,416,595	5,320,080	4,579,390	13,316,065 (100%)

Class 1 category. This is almost 3.5 times the percentage of that for Black males. The social class category with the least amount of population contributed was the social class

5 category (the lowest social class group).

Thirty-eight percent of the population count was contributed by NH-White females belonged to Social Class 3 category (Table 4.37). The second highest percentage of the population is contributed by NH-White females in Social Class 2 category at 29.09%. This is in contrast to Black females, with Social Class 4 as the second highest percent of person years contributed. 19.37% of NH-White female person years belonged to the Social Class 1 category. This is 2.0 times higher than the percentage of that for Black females. The social class category with the least amount of the population contributed was the social class 5 category (the lowest social class group).

Table 4.37. Non-Hispanic White female population count by social class and age-group (35+years), 2000 US Census, Florida population multiplied by 5 years

	Age-Group			
	35-44	**45-64**	**65+**	**Total**
Social Class 1	840,695	1,296,335	793,860	2,930,890 (19.37%)
Social Class 2	1,190,510	1,859,595	1,353,105	4,403,210 (29.09%)
Social Class 3	1,082,905	2,094,790	2,570,290	5,747,985 (37.98%)
Social Class 4	224,380	453,500	849,705	1,527,585 (10.09%)
Social Class 5	36,105	105,220	383,530	524,855 (3.47%)
Total	3,374,595	5,809,440	5,950,490	15,134,525 (100%)

Overall, Social Class 3 residents contributed the majority of the population to this study (34.42%). Social Class 5 residents contributed 4.82% of the total study population. Black residents were more likely then NH-Whites to have less than a high school education. Consequently, NH-Whites were more likely then Black residents to continue their education beyond high school.

Total Census Tract Stroke Deaths by Race and Sex

A total of 42,810 stroke deaths were documented for all Florida residents 35 years of age and older within the 1998-2002 study time period. Death records which did not

include educational attainment information were not included in this count. NH-White females and males accounted for the highest percentage of stroke deaths in the study, with approximately 51.81% and 36.29% respectively (Table 4.38). Black males and females accounted for 4.91% and 6.98% of the stroke deaths respectively. The 65 years and up age group contributed 89.73% of the total number of stroke deaths in the study. As expected, the youngest age group contributed the least percentage of the stroke deaths. The 45-64 year age group accounted for the remaining 8.87% of the stroke deaths. Within each of the race-sex groups the occurrence of stroke deaths increased with age.

Table 4.38. Percent Stroke Deaths for Race-Sex Groups By Age-Group, Florida 1998-2002

	35-44	45-64	65_up	Total
Black Males	4.61	30.94	64.45	4.91 (100)
Black Females	4.55	18.03	77.42	6.98 (100)
NH-White Males	1.31	9.11	89.58	36.29 (100)
NH-White Females	0.73	5.37	93.90	51.80 (100)
Total	1.40	8.87	89.73	100

The highest percentage of stroke deaths was among residents of the Social Class 3 category at 44.22% (Table 4.39). Social Class 4 deaths made up the smallest percentage of total stroke deaths (10.53%). The remaining three social class groups each contributed around 15% of the total stroke deaths.

Table 4.39. Percent Stroke Deaths by Social Class Group, Florida 1998-2002

	SC1	SC2	SC3	SC4	SC5	Total
Total	**15.45%**	**14.87%**	**44.22%**	**10.53%**	**14.93%**	**100%**

Black males and females contributed the least percentage of deaths to the study, 4.91 and 6.98% respectively (Table 4.40). Over half of the study deaths were contributed by NH White females while NH White males made up 36.29 percent of the study deaths. NH Whites consistently contributed the highest percentage of deaths by social class. NH

White females generally contributed the highest percentage of deaths with the exception of Social Class 1, where NH White males made up 52.66% of the SC1 deaths. Black males consistently contributed the least percentage of deaths for each of the social class groups.

Table 4.40. Percent Stroke Deaths for Race-Sex Groups by Social Class Groups, Florida 1998-2002.

	SC1	SC2	SC3	SC4	SC5	Total
Black Males N=2104	1.82	2.29	3.81	8.63	11.37	4.91
Black Females N=2990	3.93	4.24	4.75	11.76	16.13	6.98
NH-White Males N=15,536	52.66	39.48	33.26	31.74	28.35	36.29
NH-White Females N=22,180	41.58	53.98	58.18	47.87	44.14	51.81
Total	**100**	**100**	**100**	**100**	**100**	**100**

Seen in Table 4.41, is the number of census tracts for which educational attainment data is available for specific race-sex-age groups. In no instances did the US Census report social class information for the 12 race-sex-age groups for all 3138 census tracts. In particular, educational attainment data for Black Floridians is reported for only a small number of the census tracts. For Black males and females, the smallest number of census tracts with educational attainment information is for the 65 years and older age group. The exception occurs for educational attainment group 5, where 35-44 year old Black males and females have data reported for the least number of census tracts.

For Non-Hispanic White males, the smallest number of census tracts with educational attainment information is for the 35-44 years and older age group (with the exception of social class 2). For Non-Hispanic White females, across all age-groups, population counts for those in social class groups 4 and 5 were the least reported. Overall, less than one-third of the census tracts have complete data for reporting

educational attainment information for Black Floridians. The opposite is true for NH-Whites. In most social class categories, a larger number of the census tracts have reported data for NH-White males and females. The exception is the information

Table 4.41. Number of census tracts (by race, sex, age-group) for which educational attainment data were reported, Florida, 2000 US Census, Summary File 4

		College Degree and Beyond Social Class Group 1	Some College / Associates Degree Social Class Group 2	High School Graduate Social Class Group 3	9-11 years of education Social Class Group 4	Less than 9 years of education Social Class Group 5
Black Males	35-44	465	751	1055	707	393
	45-64	565	738	1029	770	706
	65+	247	323	604	560	696
Black Females	35-44	562	793	1057	735	380
	45-64	581	750	1052	776	668
	65+	308	401	721	653	746
Non-Hispanic White Males	35-44	1909	2113	2628	1600	763
	45-64	1998	2129	2688	1718	1363
	65+	1941	2076	2643	1758	1753
Non-Hispanic White Females	35-44	1917	2104	2625	1508	601
	45-64	1984	2130	2708	1745	1218
	65+	1895	2093	2703	1809	1851

available for NH-White males and females for Social Class 5 (lowest social class group). An average of 26% of the census tracts have data for NH-Whites 35-44 years of age, nearly 53% of the census tracts have data for NH-Whites 45-64 years of age and around 75% of the census tracts have data for NH-Whites 65 years of age and above.

Stroke Death Rates and Outcome Scores: Census Tract Level

All Florida Census Tracts

Stroke death rates and outcomes scores at the census tract level were calculated

for all Florida census tracts (Table 4.42). Each of the average rates and disparity scores are weighted by the total census tract population. Upon examination of the race-sex specific age adjusted rates, the lowest rates for all race-sex groups are observed for Social Class 2. For Black residents, the highest rates occur within Social Class 3, however, the highest rates for Non Hispanic White residents occur in the lowest social class group (Social Class 5). Black residents have higher stroke death rates than NH-White residents for each of the social class categories. For both males and females, the largest racial difference in rates occurs in Social Class 3. Racial disparity scores (ratios, difference and percent difference scores) for males are greatest for the Social Class 3 residents. For females, the highest ratio and percent difference score occurs for the Social Class 2 category; however, the highest difference score occurs for the Social Class 3 category for females.

Table 4.42. Weighted average stroke death rates and disparity scores by social class group, Florida 1998-2002, (Number of Census tracts)

	SC 1 (2111)	SC 2 (2230)	SC 3 (2892)	SC 4 (2000)	SC 5 (2224)
Black Male Age Adjusted Rate	711.40	524.19	1465.96	888.07	1292.87
Black Female Age Adjusted Rate	966.32	809.89	1396.70	874.14	1329.47
NH White Male Age Adjusted Rate	208.61	174.82	369.07	405.84	1030.17
NH White Female Age Adjusted Rate	284.15	190.23	297.41	448.74	1151.97
Male Black-White Ratio	6.75	9.90	14.83	11.30	14.40
Female Black-White Ratio	8.54	18.43	15.21	13.91	10.38
Male Black-White Difference	502.79	349.36	1096.91	482.23	262.69
Female Black-White Difference	682.17	619.65	1099.28	425.41	177.50
Male Black-White Percent Difference	575.35	890.37	1382.77	1030.17	1340.24
Female Black-White Percent Difference	753.99	1742.62	1421.50	1291.57	938.33

Restricted Census Tracts

Only those census tracts that had nonzero values (i.e., population counts not equal to zero) for each of the 12 race-sex-age-groups were retained in subsequent analyses.

Utilization of this inclusion/exclusion criterion resulted in the retention of only a portion of the census tracts. Please refer back to Table 4.41 for the exact number of census tracts for which educational attainment (by race-sex-age-group) data was available.

Because of the small number of census tracts with available data for each of the race-sex social class groups, calculation of an accurate population (denominator data) count for the social class groups at the census tract level was not possible. Without the denominator counts, the calculation of age-adjusted stroke death rates for the 20 race-sex social class groups was not possible. This limitation of the data prevented any further examination of the research question regarding the influence of social class on the magnitude of racial disparity in .stroke mortality at the census tract level.

Although reliable age adjusted stroke death rates could not be calculated at the census tract level, the data were sufficient for the calculation of rates for each of the social class groups, by race and sex, for the state of Florida as a whole. Three age-group specific rates were calculated (Tables 4.43, 4.44, 4.45), as well as, age-adjusted rates (Table 4.46). For those in the 35-44 year age-group, Black males and females experienced higher deaths rates than their non-Hispanic White counterparts in each of the social class groups. The largest racial disparity is seen in the Social Class 1 category. Within the Social Class 1 category, Black males experienced 7.1 times the stroke death rate of NH White males and Black females had a rate 5.7 times that of NH White females. The rates for Blacks and Whites are the most similar within the Social Class 4 category. Black females have rates ranging from 3.8 to 4.5 times higher than NH White females for the remainder of the social class categories. Black males have rates from 1.6 to 2.8 times that of NH White males for the remainder of the social class categories.

Table 4.43. Race-Sex Specific Stroke Death Rates (per 100,000): 35-44 Years of Age, Florida 1998-2002

	Black Males	Black Females	NH White Males	NH White Females
Social Class 1	14.70	15.07	2.07	2.62
Social Class 2	12.08	15.52	4.27	3.44
Social Class 3	20.40	24.54	8.87	6.46
Social Class 4	11.32	17.31	10.44	12.48
Social Class 5	17.73	23.39	11.26	5.54

For those in the 45-64 year age-group, the largest racial disparity is seen in the Social Class 1 category (Table 4.44). Within the Social Class 1 category, Black males experienced 4.4 times the stroke death rate of NH White males and Black females had a rate 5.9 times that of NH White females. The rates for Blacks and Whites are the most similar within the Social Class 5 category, with NH white females experiencing a slightly higher rate than Black females. Black males and females have rates between 2 and 4 times that of NH White males and females for the remainder of the social class categories.

Table 4.44. Race-Sex Specific Stroke Death Rates (per 100,000): 45-64 Years of Age, Florida 1998-2002

	Black Males	Black Females	NH White Males	NH White Females
Social Class 1	67.12	69.94	15.22	11.88
Social Class 2	53.00	49.29	18.35	13.77
Social Class 3	132.49	84.95	40.95	27.21
Social Class 4	85.55	58.85	38.23	28.44
Social Class 5	102.88	73.27	63.92	77.93

For those in the 65 years and up age-group, the largest racial disparities are seen in social class group 3 for males and group 2 for females (Table 4.45). In social class group 3, Black males have rates 2.0 times higher than NH White males. In social class group 2, Black females have rates 2.2 times that of NH White females. The rates for Blacks and Whites are the most similar within the Social Class 5 category, with NH white

females experiencing a slightly higher rate than Black females. Black females have rates ranging from 1.5 to 2.1 times higher than NH White females for the remainder of the social class categories. Black males have rates ranging from 1.5 to 1.7 times that of NH White males for the remainder of the social class categories.

Table 4.45. Race-Sex Specific Stroke Death Rates (per 100,000): 65-up Years of Age, Florida 1998-2002

	Black Males	Black Females	NH White Males	NH White Females
Social Class 1	472.24	694.19	277.98	324.24
Social Class 2	304.73	522.36	199.45	231.98
Social Class 3	767.35	711.02	390.53	403.65
Social Class 4	354.62	348.62	210.26	235.49
Social Class 5	580.65	679.26	545.23	713.63

When the age-adjusted rates are examined, Black males and females experienced higher deaths rates in social class groups 1 thru 4 when compared to the rates for Non-Hispanic White residents (Table 4.46). For social class group 5, Non-White Hispanic females experienced the highest stroke mortality rate at 210.53 per 100,000. For males, the largest racial disparity in stroke mortality exists within the Social Class 3 category. Within the Social Class 3 category, Black males experienced 2.17 times the stroke death rate of NH White males. For females, the largest racial disparity in stroke mortality exists within the Social Class 1 and 2 categories. Black females had a rate 2.35 and 2.34 times that of NH White females for social class categories 1 and 2, respectively. The rates for Black and Non-Hispanic Whites females are the most similar within the Social Class 5 category, with NH white females experiencing a slightly higher rate than Black females. The rates for Black and Non-Hispanic Whites males are also the most similar within the Social Class 5 category, with Black males experiencing a slightly higher rate than Non-Hispanic White males (1.17 times higher).

Table 4.46. State level age-adjusted (35+ years) stroke death rates and disparity scores by social class group, Florida 1998-2002

	SC 1	SC 2	SC 3	SC 4	SC 5
Black Male Age Adjusted Rate	149.60	101.57	252.10	127.64	192.64
Black Female Age Adjusted Rate	201.00	149.94	211.93	111.53	199.05
NH White Male Age Adjusted Rate	75.43	58.23	116.36	71.47	165.01
NH White Female Age Adjusted Rate	85.49	63.96	112.85	74.08	210.53
Male Black-White Ratio	1.98	1.74	2.16	1.78	1.16
Female Black-White Ratio	2.35	2.34	1.87	1.50	0.94
Male Black-White Difference	74.17	43.34	135.74	56.17	27.63
Female Black-White Difference	115.51	85.98	99.08	37.45	-11.48
Male Black-White Percent Difference	98.32	74.42	116.65	78.59	16.74
Female Black-White Percent Difference	135.11	134.42	87.79	50.55	-5.45

Summary of Findings

The investigation into the potential influence of social class on the magnitude of Black-White disparities in stroke mortality was precluded by lack of data. Reliable age adjusted stroke death rates could not be calculated at the census tract level. However, the calculation of rates for each of the social class groups, by race and sex, for the State of Florida as a whole was possible. As expected, stroke mortality rates increased with age for each of the race-sex groups. In each of the three age-group categories, Black males and females consistently experienced higher stroke mortality rates across each of the social class groups. The exceptions were instances in which 45-64 year old and 65+ year old NH-White females in Social Class 5 experienced slightly higher stroke mortality rates than Black females. Most decedents in this social class group experienced the least favorable stroke death rates. Of particular note is the observation that Black and Non Hispanic White residents experience similar rates only when examining the social class 5 category.

The study hypothesis stated that Black-White disparities in stroke mortality would

be greatest for those in the lowest social class group (social class group 5). The results did not support the study hypothesis; instead, stroke mortality rates were lowest for those in social class group 5 (Table 4.47). The lowest disparity scores occurred for those in social class group 5 for both males and females for each of the three disparity scores. For each of the three disparity score outcomes, male disparities are highest for high school graduates (social class three) and female disparities are highest for the college educated. A test for trend in the disparity scores across social class groups was completed. There were no statistically significant trends in any of the disparity scores across social class groups as measured by the Mantel-Haenszel Chi Square test for trend (Table 4.47).

Table 4.47. State level Black-White disparity scores by social class group, Florida 1998-2002

	SC 1	SC 2	SC 3	SC 4	SC 5	Trend Probability
Male Ratio	1.98	1.74	2.16	1.78	1.16	0.1798
Male Difference	74.17	43.34	135.74	56.17	27.63	0.5442
Male Percent Difference	98.32	74.42	116.65	78.59	16.74	0.1816
Female Ratio	2.35	2.34	1.87	1.50	0.94	0.0528
Female Difference	115.51	85.98	99.08	37.45	-11.48	0.0651
Female Percent Difference	135.11	134.42	87.79	50.55	-5.45	0.0530

Part IV: Research Question 3

Question: Is there effect modification by social class of the ASPoH and Black-White disparities in stroke mortality relationship?

Hypothesis: ASPoH will have a greater impact of Black-White disparities in stroke mortality for the lower social class groups.

Descriptive Statistics

Table 4.48. Florida population and stroke death counts by social class category

	Social Class Category				
Ages 35+ years	**SC1(high)**	**SC2**	**SC3**	**SC4**	**SC5(low)**
Black Males					
Deaths	121	146	721	389	727
Population	123,940	277,137	521,111	387,912	242,467
Black Females					
Deaths	260	270	899	530	1031
Population	185,597	391,327	602,144	450,834	259,875
Non-Hispanic White Males					
Deaths	3483	2513	6297	1431	1812
Population	3,659,902	3,732,923	4,106,766	1,304,615	515,279
Non-Hispanic White Females					
Deaths	2750	3436	11,015	2158	2821
Population	2,931,156	4,403,315	5,748,146	1,528,106	527,428

Table 4.48 shows that the majority of the stroke deaths and population counts, for all race-sex groups, are concentrated within social class groups 3 and 5. The Black male and female populations were lowest in the Social Class 1 (highest) category. In contrast, for NH-White males and females, the populations were lowest in the Social Class 5 category.

Summary statistics for the study outcome variables are presented in Table 4.49. These statistics demonstrate that there are differences between Black and White stroke mortality rates. The median statistic for each of the outcome variables reflects the evenness of the race specific rates occurring in at least 50% of the census tracts retained in the study analyses. The minimum scores represent those instances in which the Black age-adjusted stroke death rate was at or near zero and the White age-adjusted stroke death

Table 4.49. Effect Modification: Summary Statistics for Black-White stroke mortality disparity measures

	Mean	Med	Std Dev	Min	Max
Male Black-White Ratio[1]	9.57	0	65.27	0	1809.48
Female Black-White Ratio[2]	9.19	0	52.55	0	860.00
Male Black-White Difference[3]	701.25	-51.09	7473.58	-75476.40	67202.81
Female Black-White Difference[3]	506.77	-68.12	7157.29	-67815.90	43472.14
Male Percent Difference[1]	857.55	-100.00	6527.10	-100.00	180847.70
Female Percent Difference[2]	819.16	-100.00	5255.60	-100.00	85900.00
[1]: N=2411 census tracts, [2]: N=2898 census tracts, [3]: N=4133 census tracts					

rate is either similar to or much larger than the Black stroke death rate. The maximum scores are representative of those instances in which the Black stroke death rates are much larger than the stroke death rate for NH-Whites.

Regression Analyses

Simple linear regression was used to test the model: Disparity Score → ASPoH. Separate regression analyses were run for each of the 5 social class groups. For example, for Social Class Group 1 only, a regression analyses was run to test how well the ASPoH variables could predict the Male Black-White Ratio score. Next, the same analysis was performed for Social Class Group 2, only. These analyses were then completed separately for each of the three remaining Social Class groups. This technique was continued for each of the remaining Black-White disparity scores. As a consequence of using this methodology, statistically significant differences (of the disparity and ASPoH relationship) between social class groups cannot be determined.

When testing the predictability of each of the disparity scores by the ASPoH variables, for each social class group, the following models were found to be statistically significant ($p<.05$).

1. Social Class 3: Female Black-White Disparity Difference = $ß_0$ + $ß_1$Prin1 + $ß_2$Prin2 + $ß_3$Prin3 + $ß_4$Prin4 + ε

2. Social Class 4: Male Black-White Disparity Difference = $ß_0$ + $ß_1$Prin1 + $ß_2$Prin2 + $ß_3$Prin3 + $ß_4$Prin4 + ε

3. Social Class 4: Female Black-White Disparity Difference = $ß_0$ + $ß_1$Prin1 + $ß_2$Prin2 + $ß_3$Prin3 + $ß_4$Prin4 + ε

Predictability of ASPoH-1 across Social Class Groups

Male Ratio: ASPoH-1

There is no obvious trend in parameter estimates from the highest social class group to the lowest social class group (Table 4.50). With every one unit increase in ASPoH-1, the male Black-White ratio decreases 0.8432 for social class group 1 and decreases 0.0093 for social class group 2. The ratio decreases 1.3773 and 0.3984 for Social Class 3 and 4, respectively. For social class 5 residents, the ratio increases 1.8890 with every one unit increase in ASPoH-1. In the current and remaining regression models, results obtained when utilizing the Male Percent Difference Score were numerically identical to those obtained with the use of the Male Ratio Score.

Table 4.50. Individual regression models which measured effect modification by social class of the association between the male Black-White stroke mortality ratio and the 'Area Social Predictors of Health-1' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr> | t |**
Social Class 1 (2111)	-0.8432	1.1873	-0.71	0.4780
Social Class 2 (2230)	-0.0093	1.3287	-0.01	0.9944
Social Class 3 (2892)	-1.3773	2.3829	-0.58	0.5634
Social Class 4 (2000)	-0.3984	3.0992	-0.13	0.8978
Social Class 5 (2224)	1.8890	1.3740	1.37	0.1699

Female Ratio: ASPoH-1

There is no obvious trend in parameter estimates from the highest social class

group to the lowest social class group (Table 4.51). With every unit increase in ASPoH-1, the female Black-White ratio decreases 1.1784 and 1.7813 for social class groups 1 and 5 respectively; however, the ratio decreases only slightly for social class groups 2 and 4 (0.2557 and 0.2911 respectively). The ratio increases 2.3202 for Social Class group 3. In the current and remaining regression models, results obtained when utilizing the Female Percent Difference Score were numerically identical to those obtained with the use of the Female Ratio Score.

Table 4.51. Individual regression models which measured effect modification by social class of the association between the female Black-White stroke mortality ratio and the 'Area Social Predictors of Health-1' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	-1.1784	1.2754	-0.92	0.3560
Social Class 2 (2230)	-0.2557	2.1217	-0.12	0.9041
Social Class 3 (2892)	2.3202	1.1821	1.96	0.0500
Social Class 4 (2000)	-0.2911	2.0011	-0.15	0.8844
Social Class 5 (2224)	-1.7813	1.2689	-1.40	0.1609

Male Difference: ASPoH-1

There is no trend in parameter estimates from the highest social class group to the lowest social class group (Table 4.52). With every one unit increase in ASPoH-1, the male Black-White difference score decreases 218.1073 and 411.8448 (statistically significant, $p<.05$) for social class groups 2 and 4. The difference score increases 336.9195, 204.4327 and 40.6721 for Social Class 1, 3 and 5, respectively.

Table 4.52. Individual regression models which measured effect modification by social class of the association between the male Black-White stroke mortality difference score and the 'Area Social Predictors of Health-1' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	336.9195	194.0280	1.73	0.0832
Social Class 2 (2230)	-218.1073	139.1213	-1.57	0.1174
Social Class 3 (2892)	204.4327	176.0836	1.16	0.2459
Social Class 4 (2000)	-411.8448	179.8761	-2.29	0.0223
Social Class 5 (2224)	40.6721	200.8182	0.20	0.8395

Female Difference: ASPoH-1

There is no trend in parameter estimates from the highest social class group to the lowest social class group (Table 4.53). With every one unit increase in ASPoH-1, the female Black-White difference score decreases for social class groups 1 and 5. The difference score increases for social class groups 2, 3 and 4. The increase in the Female Difference score of 427.4808 per unit increase in the ASPoH-1 variable for social class 3 is statistically significant ($p<.05$).

Table 4.53. Individual regression models which measured effect modification by social class of the association between the female Black-White stroke mortality difference score and the 'Area Social Predictors of Health-1' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	-95.3442	189.8774	-0.50	0.6157
Social Class 2 (2230)	27.0368	141.9722	0.19	0.8490
Social Class 3 (2892)	427.4808	126.9901	3.37	0.0378
Social Class 4 (2000)	58.0998	182.2580	0.32	0.7500
Social Class 5 (2224)	-193.1107	220.3813	-0.88	0.3811

Predictability of ASPoH-2 across Social Class Groups

Male Ratio: ASPoH-2

With every one unit increase in ASPoH-2, the male Black-White ratio increases 1.0612, 0.0173 and 4.3451 for social class groups 1, 3 and 5 (Table 4.54). The ratio decreases 0.7950 and 5.2862 for Social Class 2 and 4, respectively.

Table 4.54. Individual regression models which measured effect modification by social class of the association between the male Black-White stroke mortality ratio and the 'Area Social Predictors of Health-2' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	1.0612	2.4132	0.44	0.6603
Social Class 2 (2230)	-0.7950	2.7043	-0.29	0.7686
Social Class 3 (2892)	0.0173	4.9602	0.00	0.9972
Social Class 4 (2000)	-5.2862	6.2953	-0.84	0.4016
Social Class 5 (2224)	4.3451	3.1765	1.37	0.1721

Female Ratio: ASPoH-2

With every one unit increase in ASPoH-2 the female Black-White ratio increases 2.4913 for social class group 3 (Table 4.55). The ratio decreases with each unit increase of the ASPoH-2 variable for the remaining social class groups. The largest decrease in the female Black-White ratio (2.1904) occurs for social class group 1.

Table 4.55. Individual regression models which measured effect modification by social class of the association between the female Black-White stroke mortality ratio and the 'Area Social Predictors of Health-2' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	-2.1904	2.5398	-0.86	0.3889
Social Class 2 (2230)	-1.9846	4.2438	-0.47	0.6402
Social Class 3 (2892)	2.4913	2.4428	1.02	0.3081
Social Class 4 (2000)	-0.1706	4.1399	-0.04	0.9671
Social Class 5 (2224)	-0.7137	2.7448	-0.26	0.7949

Male Difference: ASPoH-2

There is no trend in parameter estimates from the highest social class group to the lowest social class group (Table 4.56). The difference score increases with an increase in the ASPoH-2 variable for social class groups 1, 3 and 5. The largest increase in the male Black-White difference score occurs for social class group 3, with an increase of 729.5325 points. However, for social class groups 2 and 4, the male Black-White difference score decreases, 192.7746 and 908.5961 (statistically significant, $p<.05$) respectively, with every one unit increase in ASPoH-2.

Table 4.56. Individual regression models which measured effect modification by social class of the association between the male Black-White stroke mortality difference score and the 'Area Social Predictors of Health-2' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	384.3310	402.1029	0.96	0.3395
Social Class 2 (2230)	-192.7746	291.1515	-0.66	0.5081
Social Class 3 (2892)	729.5325	379.6207	1.92	0.0549
Social Class 4 (2000)	-908.5961	373.4455	-2.43	0.0152
Social Class 5 (2224)	284.0109	435.1022	0.65	0.5141

Female Difference: ASPoH-2

There is no trend in parameter estimates from the highest social class group to the lowest social class group (Table 4.57). The female Black-White difference score decreases with an increase in ASPoH-2 for social class groups 2, 4 and 5. The largest decrease in the female difference score occurs for social class group 4. For social class group 4, the female difference score decreases 769.2337 (statistically significant, $p<.05$) with every one unit increase in the ASPoH-2 variable. For social class groups 1 and 3,

Table 4.57. Individual regression models which measured effect modification by social class of the association between the female Black-White stroke mortality difference score and the 'Area Social Predictors of Health-2' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	135.8651	393.5013	0.35	0.7300
Social Class 2 (2230)	-102.1741	297.1179	-0.34	0.7310
Social Class 3 (2892)	569.2816	273.7794	2.08	0.0378
Social Class 4 (2000)	-769.2337	378.3905	-2.03	0.0424
Social Class 5 (2224)	-245.4847	477.4884	-0.51	0.6073

the female Black-White difference score increases 135.8651 and 569.2816, respectively, with every one unit increase in ASPoH-2.

Predictability of ASPoH-3 across Social Class Groups

Male Ratio: ASPoH-3

With every one unit increase in ASPoH-3, the male Black-White ratio increases 1.7117 and 1.5273 for social class groups 1 and 2, respectively (Table 4.58). The ratio decreases for social class groups 3, 4 and 5, with the largest decreases occurring for groups 3 and 4. The male Black-White ratio score decreases 7.3710 and 6.1254 points for social class groups 3 and 4. Although the t-score for social class 3 is significant, $p<.05$, the overall model was not significant and further interpretation of this outcome is not permitted.

Table 4.58. Individual regression models which measured effect modification by social class of the association between the male Black-White stroke mortality ratio and the 'Area Social Predictors of Health-3' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	1.7117	1.7508	0.98	0.3288
Social Class 2 (2230)	1.5273	1.9791	0.77	0.4407
Social Class 3 (2892)	-7.3710	3.4348	-2.15	0.0322
Social Class 4 (2000)	-6.1254	4.5224	-1.35	0.1765
Social Class 5 (2224)	-0.9849	2.0684	-0.48	0.6342

Female Ratio: ASPoH-3

The female Black-White ratio decreases with every one unit increase in the ASPoH-3 score for social class groups 1, 2 and 5 (Table 4.59). The largest decrease, 4.6915, occurs for social class groups 2. The female Black-White ratio increases with every one unit increase in the ASPoH-3 score for social class groups 3 and 4. The largest increase in the female ratio score (3.6796) occurs for social class groups 2.

Table 4.59. Individual regression models which measured effect modification by social class of the association between the female Black-White stroke mortality ratio and the 'Area Social Predictors of Health-3' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	-0.8769	1.8034	-0.49	0.6271
Social Class 2 (2230)	-4.6915	2.9803	-1.57	0.1161
Social Class 3 (2892)	3.6796	1.6747	2.20	0.0283
Social Class 4 (2000)	0.9512	3.0337	0.31	0.7540
Social Class 5 (2224)	-1.1614	1.8615	-0.62	0.5329

Male Difference: ASPoH-3

The difference score decreases with an increase in ASPoH-3 for all social class groups, with the exception of social class 3 (Table 4.60). The largest decreases in the male Black-White difference score occur for social class groups 4 and 5 with decreases of 742.6002 (statistically significant, p,.05) and 622.2808, respectively. For social class group 3, the male Black-White difference score increases 132.1856 with every one unit

increase in ASPoH-3.

Table 4.60. Individual regression models which measured effect modification by social class of the association between the male Black-White stroke mortality difference score and the 'Area Social Predictors of Health-3' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	-200.4952	280.8351	-0.71	0.4755
Social Class 2 (2230)	-97.9421	203.2443	-0.48	0.6300
Social Class 3 (2892)	132.1856	252.1665	0.52	0.6003
Social Class 4 (2000)	-742.6002	253.1480	-2.93	0.0035
Social Class 5 (2224)	-622.2808	290.8366	-2.14	0.0327

Female Difference: ASPoH-3

The female Black-White difference score increases with an increase in ASPoH-3 for social class groups 1 and 2(Table 4.61). For social class groups 1 and 3, the female Black-White difference score increases 195.8663 and 431.6962 with every one unit increase in the ASPoH-3 score. The difference score decreases with an increase in ASPoH-3 for social class groups 2, 4 and 5. The difference score decreases 603.1654 for the lowest social class group. The increase in the Female Difference score of 431.6962 per unit increase in the ASPoH-3 variable for social class 3 is statistically significant ($p<.05$).

Table 4.61. Individual regression models which measured effect modification by social class of the association between the female Black-White stroke mortality difference score and the 'Area Social Predictors of Health-3' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	195.8663	274.8275	0.71	0.4763
Social Class 2 (2230)	-334.6438	207.4093	-1.61	0.1071
Social Class 3 (2892)	431.6962	181.8604	2.37	0.0178
Social Class 4 (2000)	-429.0295	256.5000	-1.67	0.0948
Social Class 5 (2224)	-603.1654	319.1690	-1.89	0.0591

Predictability of ASPoH-4 across Social Class Groups

Male Ratio: ASPoH-4

With the exception of social class 4 results, the male Black-White ratio decreases

with every one unit increase in the ASPoH-4 score (Table 4.62). The largest decrease is seen for social class 2 with a 3.8573 decrease in the ratio score with one unit increase in the ASPoH-4 variable. For those in social class 4, the male ratio increases 13.3944 points with every one unit increase in ASPoH-4.

Table 4.62. Individual regression models which measured effect modification by social class of the association between the male Black-White stroke mortality ratio and the 'Area Social Predictors of Health-4' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	-0.4280	2.6894	-0.16	0.8736
Social Class 2 (2230)	-3.8573	2.8996	-1.33	0.1841
Social Class 3 (2892)	-0.3378	4.8869	-0.07	0.9449
Social Class 4 (2000)	13.3944	7.8845	1.70	0.0902
Social Class 5 (2224)	-0.5510	2.9039	-0.19	0.8496

Female Ratio: ASPoH-4

There is no obvious trend in parameter estimates from the highest social class group to the lowest social class group (Table 4.63). For social class groups 1, 4 and 5, there is a decrease in the female Black-White ratio score with every one unit increase in ASPoH-4. The largest decreases are seen for those residents without a high school diploma. The female Black-White ratio increases for social class groups 2 and 3, with the largest increase of 3.0487 observed for those with some college education (social class group 2).

Table 4.63. Individual regression models which measured effect modification by social class of the association between the female Black-White stroke mortality ratio and the 'Area Social Predictors of Health-4' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	-1.8392	2.7920	-0.66	0.5104
Social Class 2 (2230)	3.0487	4.2881	0.71	0.4774
Social Class 3 (2892)	1.1207	2.0374	0.55	0.5824
Social Class 4 (2000)	-3.6121	4.4296	-0.82	0.4152
Social Class 5 (2224)	-3.2624	2.9685	-1.10	0.2722

Male Difference: ASPoH-4

The male Black-White difference score increases 43.5065 and 193.6020 points with an increase in the ASPoH-4 score for social class groups 3 and 4 (Table 4.64). However, for social class groups 1, 2 and 5, the male Black-White difference score decreases from 133.0851 (for social class 1) to 691.3068 (for social class 2) points with an increase in the ASPoH-4 score.

Table 4.64. Individual regression models which measured effect modification by social class of the association between the male Black-White stroke mortality difference score and the 'Area Social Predictors of Health-4' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	-133.0851	389.2332	-0.34	0.7325
Social Class 2 (2230)	-691.3068	293.0601	-2.36	0.0186
Social Class 3 (2892)	43.5065	299.7640	0.15	0.8846
Social Class 4 (2000)	193.6020	364.3303	0.53	0.5953
Social Class 5 (2224)	-141.5969	383.0479	-0.37	0.7117

Female Difference: ASPoH-4

The female Black-White difference score increases 96.5101 (for social class 5) to 630.5164 (for social class 1) points with increases in the ASPoH-4 score for social class groups 1, 3 and 5 (Table 4.65). Alternately, there is a 205.8951 point decrease in the difference score with an increase in the ASPoH-4 score for social class group 2 and a 368.0623 point decrease for social class group 4.

Table 4.65. Individual regression models which measured effect modification by social class of the association between the female Black-White stroke mortality difference score and the 'Area Social Predictors of Health-4' variable.

(# of census tracts)	**Parameter Estimate**	**Std Error**	**t-value**	**Pr > |t|**
Social Class 1 (2111)	630.5164	380.9069	1.66	0.0983
Social Class 2 (2230)	-205.8951	299.0664	-0.69	0.4914
Social Class 3 (2892)	382.3863	216.1874	1.77	0.0772
Social Class 4 (2000)	-368.0623	369.1547	-1.00	0.3191
Social Class 5 (2224)	96.5101	420.3632	0.23	0.8185

Summary of Findings

The potential for effect modification by social class of the association between Black-White disparities in stroke mortality and ASPoH variables was investigated. Study findings were dependent upon the particular Black-White disparity score and the ASPoH variable under investigation. Patterns of association across social class groups, or the lack thereof, were not consistent across Black-White disparity measures. The study hypothesis stated that the ASPoH measures would have the greatest impact on those residents in the lowest social class category, i.e. SC5 (less than 9 years of education). An increase in the disparity score would suggest greater differences in stroke mortality rates between Blacks and non-Hispanic Whites, therefore supporting the hypothesis. This hypothesized effect was identified in two instances (Table 4.66). When utilizing the ASPoH-1 and ASPoH-2 variables to estimate effects on the Male Black-White Ratio disparity score, the disparity score increased the greatest amount for those residents with less than 9 years of education. None of the remaining regression models supported the study hypothesis.

Table 4.66. Summary results for regression models which measured effect modification by social class of the association between the disparity in stroke mortality measures and the 'Area Social Predictors of Health' variables

Predictor Variable	Black-White Stroke Mortality Difference Score	Social Class Group with the greatest increase in the Disparity Measure
ASPoH-1	Male Ratio	SC5
	Female Ratio	SC3
	Male Difference	SC1
	Female Difference	SC3
ASPoH-2	Male Ratio	SC5
	Female Ratio	SC3
	Male Difference	SC1
	Female Difference	SC3
ASPoH-3	Male Ratio	SC1
	Female Ratio	SC3
	Male Difference	SC3
	Female Difference	SC3
ASPoH-4	Male Ratio	SC4
	Female Ratio	SC2
	Male Difference	SC4
	Female Difference	SC1

Chapter Five

Discussion

Introduction

Many questions remain regarding the determinants of racial disparities in stroke mortality. The influence of individual characteristics and neighborhood economic structure on health has been the focus of several studies over the past two decades. Education, income or occupation (or a combination of two or more of these measures) are typically used as measures of individual social class or socioeconomic status, while an area-based socioeconomic indicator (composed of various area/neighborhood level economic and social measures obtained from census data) often represents the economic structure. Findings from this type of research frequently support the hypothesis that living in economically deprived areas and being a member of a lower SES group are both associated with an increased prevalence of negative health outcomes.[114]

Given these research findings, there exists an opportunity to examine whether these area characteristics may affect race groups differentially, possibly leading to disparities in health outcomes, specifically stroke mortality. This study was an attempt to further our understanding of Black-White disparities in stroke mortality by looking beyond racial differences in individual level factors commonly associated with these disparities. This study departs from this extensively investigated path, and instead focuses on social and economic aspects of the community as contributing factors in these

disparities. To attempt to understand the basis of race-based stroke mortality patterns, this study examined variations in Black-White disparities in stroke mortality at the census tract level as a function of area characteristics. Racial inequalities in stroke mortality represent a major challenge for which effective action must focus on the social and economic environment. [115]

'Are there contextual social and economic area characteristics related to Black-White disparities in stroke mortality independently of and/or in conjunction with individual-level variables?' was the primary research question investigated in this study. In the effort to address this issue, a progression through the following questions was required: (1) Are Black-White disparities in stroke mortality elevated in those areas with lower amounts of social and economic resources (represented by the Area Social Predictors of Health variables)? (2) Are higher levels of Black-White disparities in stroke mortality associated with low levels of social class? (3) Is there effect modification by social class of the ASPoH measure and Black-White disparities in stroke mortality relationship? In response to these research questions, the study hypotheses were: (1) Black-White disparities in stroke mortality will be greatest at lower levels of ASPoH, (2) Black-White disparities in stroke mortality will be greatest for those in the lowest social class group, and (3) ASPoH will have a greater impact on Black-White disparities in stroke mortality for the lower social class groups.

Major Findings

Research Question One

Specifically, this study examined the effect of area social predictors of health (ASPoH) on Black-White disparities in stroke mortality rates for Florida residents

between 35 and 74 years of age within the 1998-2002 five-year time period. Four measures of area social predictors were developed through the use of principal components analyses (accounting for a total of 76.29% of the variance in the data). Principal component one (ASPoH-1) was representative of the general economic status of the census tract (median household income and percent of households within the census tract that were above the poverty rate). Principal component two (ASPoH-2) was representative of the percent of occupied homes and the percent of the population employed fulltime. Principal component 3 (ASPoH-3) was representative of area affluence (Median home value and percent of home ownership). Principal component 4 (ASPoH-4) was representative of opportunities afforded by educational resources (Percent of census tract residents who are employed and percent of census tract residents 25 years and older who are high school graduates).

Predictability of ASPoH Variables

Multiple linear regression models were used to test the predictability of the gender specific racial disparity scores by the ASPoH measures (4 principal components) at the census tract level. The regression model predicting the Female Ratio score was the only statistically significant model in these analyses. Because there were census tracts with populations too small to have any expected stroke deaths, analyses were performed only on those census tracts with non-zero age-adjusted rates for each of the race-sex groups. This restriction results in the inclusion of only 363 census tracts in the analyses for males and 323 census tracts for females.

Multiple linear regression models were then used to test the predictability of age adjusted stroke death rates separately for Black males and females by the ASPoH

measures for these select census tracts. ASPoH-1 was a statistically significant predictor of the Black male and female age adjusted stroke mortality rates. With a one point increase in the ASPoH-1 score, the rates increased 83.87 per 100,000 person years for males and 77.42 per 100,000 person years for females. These results are in conflict with results obtained when examining the more inclusive group of Florida census tracts (2199 census tracts), given that only a subset of the original census tracts are included in these analyses. It is also possible that this subset of census tracts are more homogeneous than the originating data set which could possible result in the attenuation of any association that may be seen the area measure and the Black male and female stroke mortality rates.

These higher Black male and female stroke mortality rates in more affluent areas may indicate that the ASPoH measures are not actually capturing levels of "area affluence." The probability must be considered that the Black males and females residing in these census tracts are not as likely to have incomes in the higher brackets as White residents. If these assertions are true, higher rates of adverse health outcomes may be expected.[84,85,86] These finding also may be indicative of Black males and females residing in more affluent neighborhoods yet they are not able to take advantage of the resources and services available within the community area. Perhaps those Black decedents, who resided in more affluent neighborhoods, nevertheless had relatively lower incomes when compared to the White residents in these areas. These findings could be reflective of the literature that describes more adverse health outcomes for societies in which there is great income inequality.[116] Research of metropolitan areas suggests that in addition to the absolute amount of income, relative disparity of income distribution within a population is also important for health. Findings show that areas with high income inequality had

significantly greater age-adjusted total mortality than those with low inequality.[117]

The household economic measure (ASPoH-1) was also a statistically significant predictor of the Non Hispanic-White male and female age adjusted stroke mortality rates. Areas with a larger proportion of residents in higher income levels and a small proportion below the poverty level experience significantly lower stroke death rates for both males and females. With a one point increase in the ASPoH-1 score, the age adjusted stroke death rates decreased 23.65 and 26.32 per 100,000 for males and females respectively. In contrast, higher NH-White female stroke mortality rates were associated with areas having more expensive homes and a larger percentage of home ownership (ASPoH-3). The Non Hispanic-White female age adjusted rate increased 13.50 per 100,000 with a one unit increase in the ASPoH-3 score.

Both household economic measures (ASPoH-1) and occupied homes and employment measures (ASPoH-2) were statistically significant predictors of the Black-White male ratio score and the Black-White female ratio score. More favorable measures of the ASPoH-2 variable were associated with lower disparity scores. ASPoH-1 was a statistically significant predictor of the Black-White male difference score and the female difference score. Increased household economic measures were predictive of increased Black-White difference scores.

ASPoH-1 and ASPoH-2 were statistically significant predictors of the Black-White female percent difference score and the male percent difference score. Increases in these measures were associated with increases in the percent difference scores. When this disparity score is utilized, more disadvantaged areas, as measured by the ASPoH-2 variable, are more likely to experience racial disparities of a greater magnitude than more

affluent areas. More affluent areas, as measured by the ASPoH-1 variable, are more likely to experience racial disparities of a greater magnitude than less affluent areas. This finding is in opposition to the study hypothesis. Once again, these finding may be due to the heterogeneity of various characteristics within census tracts. For example, it may be possible that those census tracts which fall within the "more affluent" category are comprised of families with vast differences in income. Possibly, these census tracts have household incomes from both extremes, with the majority of incomes in the higher brackets. Given the evidence presented regarding health disparities in areas of high income inequality, we would expect these results if the majority of the census tracts included in the analyses were of such economic circumstances.

Inequalities in area resource are accompanied by differences in life conditions which may adversely influence health.[98] These health inequalities result from the differential accumulation of exposures and experiences among those residing in different neighborhood environments. The effect of inequality on health reflects a combination of negative exposures and lack of resources accessible by individuals. Consequently, this lack of individual resources influences services and investments made available for these individuals. More equitable distribution of public and private resources is likely to have the greatest impact on reducing Black-White health disparities.

Research Question Two

The investigation into the potential influence of social class on the magnitude of Black-White disparities in stroke mortality was precluded many times by lack of available data. Therefore, a cautionary approach must be taken in the interpretation of these results. Due to privacy issues, the release of educational attainment data (at the

census tract level) for particular race-sex-age groups was limited. This resulted in multiple census tracts with no educational attainment data reported for the majority of the study groups. Therefore, the accurate calculation of population (denominator) counts for many of the race-sex specific social class groups was not possible. Overall, less than one-third of the census tracts have complete data for reporting educational attainment information for Black Floridians. The opposite is true for Non-Hispanic Whites. In most social class categories, a larger majority of the census tracts have reported data for Non-Hispanic White males and females. This limitation prevented any further examination of the census tract level influence of social class on the magnitude of racial disparity in stroke mortality.

Reliable age adjusted stroke death rates could not be calculated at the census tract level. However, the calculation of rates for each of the social class groups, by race and sex, for the State of Florida as a whole was possible. As expected, stroke mortality rates increased with age for each of the race-sex groups. In each of the three age-group categories, Black males and females consistently experienced higher stroke mortality rates across each of the social class groups. The exceptions were instances in which 45+ year old Non Hispanic white females in Social Class 5 experienced slightly higher stroke mortality rates than Black females. Most decedents in this social class group experienced the highest stroke death rates.

Social and economic disadvantage is associated with poor health and with increased exposure to risk factors for adverse health outcomes.[118] It is well known that a number of factors affect a person's health status, including income, occupation, education, environment, and access to services. It has been further established that an

additional factor, race, also has an impact. The Black-White health disparity may be a function of the overrepresentation of Black Americans in lower socioeconomic groups. This fact makes it difficult to ascertain whether health differentials between Black and White Americans will remain when income is held constant.

Research Question 3

Given the above mentioned limitations, a cautious investigation of effect modification by social class category was completed. Separate simple linear regression analyses (testing the association between area predictors and racial disparities in stroke mortality) were run for each of the 5 social class groups. The existence of effect modification by social class of the association between Area predictors and Black-White disparities in stroke mortality was dependent upon the particular disparity score and the Area predictor under investigation.

Using multiple linear regression to measure effect modification by social class of the association between the disparity in stroke mortality measures and 'Area Social Predictors of Health' variables, three of the sixteen regression models were found to be statistically significant ($p<.05$). For social class group 3, the ASPoH variables were found to be significant predictors of the female Black-White difference score. For social class group 4, the ASPoH variables were found to be significant predictors of the female and male Black-White difference score.

When examining the Black-White Male Ratio disparity outcome, ASPoH-1 was not shown to be a statistically significant predictor for any of the Social Class groups. However, the parameter estimates increased the most for the lowest social class group, indicating greater Black-White differences in stroke mortality rates for with less than nine

years of education. The ratio scores were similarly impacted for those in social class groups 2 and 4. There is an increase in the Black-White male ratio score from the highest social class group to the lowest social class group, indicating differential effects based on social class groups. A similar effect on the male Black-White ratio scored occurred when measuring the effect of the ASPoH-2 variable. The greatest difference in stroke mortality rates between Blacks and Whites occurred for those in social class 5. For the remaining ASPoH predictors, there is a difference in effect on the outcome across social class groups, indicating that the effect of the ASPoH predictors on the disparity score is social class dependent.

Within social class group three, the four ASPoH variables were found to be significant predictors of the female Black-White difference score. ASPoH-1, ASPoH-2 and ASPoH-3 were each individually significant predictors of the female difference score. Increases in each of the ASPoH scores resulted in significant increases in the difference scores. If measures of the ASPoH-1 and ASPoH-3 variables accurately capture economic advantage and disadvantage, the results suggest that social class group 3 residents residing in more economically advantaged areas have greater female Black-White differences in stroke mortality rates.

Within social class group four, the four ASPoH variables were found to be significant predictors of the male and female Black-White difference score. ASPoH-1, ASPoH-2 and ASPoH-3 were each individually significant predictors of the female difference score, while only ASPoH-2 was an individually significant predictor of the male difference score. Increases in each of the ASPoH scores resulted in significant decreases in the difference scores. Results suggest that social class group 4 residents

residing in more economically advantaged areas, as measured by ASPoH-1 and ASPoH-3, have lesser male and female Black-White differences in stroke mortality rates.

This study proposed a relationship between an 'area social and economic measure' and Black-White disparities in stroke mortality after adjustment for social class. Findings from this study are ambiguous, at best. There are no patterns in the results from which one may infer the slightest associations. Each area measure had its strongest impact on differing social class group. Again, these results are most likely due to the aforementioned issue of the lack of availability of educational attainment data. Another possibility may be that the proposed association (between the area measures and Black-White disparity in stroke mortality) may be, instead, mediated and/or attenuated through a third unmeasured aspect of the environment related to both race and social class. Disparities in stroke mortality may be reflective of inequities in the distribution of community resources. Possible area level risk factors for stroke mortality include reduced access to specialized medical care facilities and physicians in areas of lower socioeconomic status.[13] Additionally, stroke patients who reside in less affluent areas may not receive emergency treatment in a similarly efficient manner as those who reside in more affluent areas. This increased time to care may be due to the quality of the roads, the accessibility of the stroke patient's residential address by emergency care workers, as well as the number of emergency medical transport providers in the area.

Strengths and Limitations

A major strength is that I have been able to examine socioeconomic status at a smaller geographic unit than is typically investigated. A composite of a multitude of census tracts level variables was used in order to calculate the area score instead of

simply using median income and poverty levels.

Study results suggest that the new composite measure was meaningful. The study was able to show significant associations between the composite score and a selection of the Black-White disparity scores. Possible improvements that could be made in the development of the composite score would include the addition of other census tract level datasets representative of unmeasured dimensions of the social and economic environment. These additions would hopefully produce a measure with a more complete representation of social and economic resources available to census tract residents.

The use of multiple disparity measures is a strength of the study design. Both absolute (difference score) and relative (ratio and percent difference scores) Black-White disparity scores were utilized in this study. An absolute measure of disparity is a simple arithmetic difference between a group rate and a specified reference point. A relative measure of disparity expresses the difference between rates in terms of the chosen reference point. The percentage difference expresses the simple difference from the reference point as a percentage of the reference point. While their formulae are unique, absolute and relative measures of disparity calculated from the same reference point should lead to the same conclusion (i.e., have the same direction) about disparities between groups. The use of both types of disparity measures in this study allows for a check of the consistency in the implications of the disparity measures. A particular problem, and limitation, is that results from performing a sequence of analytical comparisons on these disparity scores is that the more comparisons conducted, the more type I errors we will make when the null hypothesis is true. The type I familywise error rate considers the possibility that one or more type I errors are made in the group of

comparisons.

There are possible limitations to study validity with the use of census-based characteristics of residential areas in the study of health differentials. The 2000 census data were used to calculate Area predictor scores for those residents who died from stroke from 1998-2002. Given that the census is taken once per decade, the appropriateness of appending census data that are at least one decade old to records to proxy current socioeconomic characteristics may be in question.[119] Therefore, in some instances, the ASPoH measure was calculated from data only relevant after the resident had died. The potential effect on study validity is limited for this particular study given that the year 2000 census data is used in order to approximate 'socioeconomic status' for those residents who died from stroke within the 1998-2002 time period. Since the census data were collected within the boundaries of the study time frame, confidence is high in the comparability of the data to the actual residential social and economic situation of the Florida stroke decedents. Additionally, the effect of this potential bias may be limited due to the findings that socioeconomic characteristics of neighborhoods generally do not change significantly over such short time periods.[119]

Determination of the appropriate level of aggregation of the census data in relation to study outcome particulars was a challenge. This study used data aggregated at the census tract level, which typically contains 5000 residents. The census tract level was the smallest level of information available for the stroke decedents included in this data. Data aggregated at a smaller more homogeneous geographic level, the census block for example, would have been preferable and possibly more informative, but was not available for this study.

As each person will only die once, if there are mutually exclusive causes of death, the causes of death compete with each other in the same subject. Competing causes of death may influence any research on either subject, resulting in the competing death bias. This study does not attempt to compare different disease mortality rates to one another. Additionally, it is not possible to estimate a difference between Black and White residents in the rates of competing causes of death without death certificate information on the 'contributing causes.' There is also no reason to doubt the accuracy of the recording of stroke as the cause of death. Furthermore, there is deficient reason to suppose that the underlying cause of death for Black and White residents would have been incorrectly categorized at differing rates.

Selection bias due to missing data may have occurred in this study. When there are a large number of variables, the regression procedure excludes an entire observation if it is missing a value for any of the variables (listwise deletion). This may result in exclusion of a considerable percentage of observations and induce selection bias. In this particular study, missing data may be distributed differentially between Black and White residents and may generate spurious associations. In this particular study, it would be more likely that population counts for Blacks are affected more than population counts for Whites, particularly for Black men in the 35-45 year age-group. The enumeration of this particular demographic group has been shown to be complicated.[120]

Missing educational attainment population counts at the census tract level posed a challenge. Missing educational attainment data was more prevalent for Black residents in the higher social class categories. In these instances, no analyses were possible do to the lack of available data. These instances were more likely to occur in census tracts with

small Black populations. Also, given that there are fewer Blacks within the higher social class groups in general, this lack of reporting was likely to occur within most census tracts and may not be specific to small population census tracts.

Another possible limitation is the question of appropriateness in using the boundaries of a census tract as a proxy to the boundaries within which resources are available. Those residents with more resources will be able to avail themselves of additional resources outside of these boundaries. The social structure may be more extensive for the more affluent. The ability to travel and work outside of one's immediate residential space may not be captured by this resource availability measure.

Aggregate level analyses are often criticized for being subject to the ecological fallacy. Consideration should be given to the possibility that analyses at the individual level may be inappropriate when seeking to determine aggregate level social and economic correlates of health and illness.[118] This study was correlational, and has the expected challenges of nonrandomized studies. These limitations include selection biases and confounding by uncontrolled variables. In this instance, individuals within census tracts could not be assigned into socioeconomic groups, and, therefore, randomization was not possible. In addition, the calculated area resource availability measure is only a proxy for level of area economic and social wellbeing. However, the association between the calculated measure and racial stroke mortality rates is similar to findings from a multitude of studies using SES measures such as employment, income and education.

An area SES score derived from census data is currently the only available data recorded and stored on a regular basis. Utilization of this type of data relies on the assumption that area of residence may provide additional information on social position

connoting an aspect of status that is not captured by individual SES measures. Possible correlation between individual study variables composing the ASPoH score must be taken into consideration. Also, the association between area SES and disparities in stroke mortality may be affected if census tract aggregates differ greatly in their socioeconomic heterogeneity. Areas are not internally homogeneous, and census tracts containing a mixture of deprived and less deprived households will have a middle ranking. The scores from census tracts with small populations (or rural areas) are more susceptible to small variations.

There is potential problem with the analyses of the restricted subset of census tracts for research question one. Restricting the analyses to those census tracts where neither the Black stroke mortality rate nor the NH-White stroke mortality rate was equal to zero possibly resulted in the exclusion of those census tracts with either very large NH-White populations or very large Black populations. Excluding those census tracts with large NH-White populations possibly resulted in the exclusion of the most affluent census tracts, whereas the exclusion of those census tracts with predominately Black populations likely resulted in the exclusion of the poorest census tracts. Consequently, the range of economic levels of the census tracts included in the restricted analyses was limited.

A potential problem with utilizing educational attainment data obtained from death certificates is the possibility that family members may report a higher level of educational attainment on the death certificate than actually achieved. Also, economic conditions make it extremely difficult, and, therefore, less likely for poor people to live in affluent areas, resulting in a small number of poor people residing in these areas. There is also the expectation that very few rich people reside in disadvantaged areas. These

situations potentially limit the study's ability to detect a potential interaction between individual SES and ASPoH on racial disparities in stroke mortality.

This study proposed that area socioeconomic structure contributes to and/or limits life choices ultimately leading to poor health outcomes. Because this study utilized ecologic data, we must take into consideration the possibility that poor health led people to move to more-deprived areas. Economic conditions influence residence in affluent and poor areas. Those who reside in poorer neighborhoods tend to have poorer health, an effect that is exacerbated in Blacks.

Consistency with the Literature

More affluent areas (as measured by the ASPoH-2 variable) were associated with smaller Black-White disparity scores. Consistent with the literature, within each racial group, residents in low SES areas experienced increased stroke mortality rates.[12] Results demonstrated higher stroke mortality rates for disadvantaged areas[94] and higher rates for Black residents compared to Non-Hispanic White residents, a findings also consistent with the reported literature. As seen in previous research findings, Black males experienced the highest stroke mortality rates, followed by Black females, White males and females, respectively.[9] Additionally, Black stroke decedents tended to be younger that White stroke decedents. Consistent with the literature, Black decedents also tended to have less education and were less likely to continue their education beyond high school and were also less likely to have ever been married.[97]

Inconsistencies of my study findings with the literature include findings that those Black residents who attained a high school degree have the highest stroke death rates compared to all other educational attainment groups. These study results were in

opposition to the proposed study hypothesis and with previous research findings.[11] The opposite occurred for NH-White residents where stroke mortality rates were highest for those in the lowest social class group (less than 9 years of education).

For a restricted subset of census tracts, more complete population count and stroke death count data were available. When only those census tracts in which there exists a non-zero age adjusted stroke death rate for both Black and White residents are examined, study results are less variable. The reasoning for the different pattern of results that is observed between Blacks and Whites could be due to the conjecture that Blacks living in the same census tract as Whites may not have access to the same resources as White residents. Additionally, results support the conjecture that Blacks and Whites may not actually share immediate environments within the same census tract.

How may the environments for Blacks differ from the environments of Whites? Studies have shown that hazardous material dumpsites are more likely to be located in Black neighborhoods.[121] Additionally, counties with a higher percentage of Black residents and high rates of income inequality tend to have a higher proportion of chemical intensive facilities located within county boundaries.[122] Black Americans are disproportionately likely to be exposed to air toxins[123] and to reside closer to the nearest industrial emission facility.[124] More than poverty, home ownership or land value, race was found to be a stronger predictor of hazardous facility placements.[123] Ramification of Blacks disproportionately residing nearer to hazardous and higher risk facilities include the burden of disproportionate health risks, possibly resulting in increased Black-White disparities in adverse health outcomes.

Calculating Area predictor scores for census tracts in which those resources for

Whites drive the magnitude of the composite scores may not at all be representative of the quantity of resources Black residents actually possess. If the salaries of Whites are vastly higher than that of Blacks residing in the same census tracts, the SES status of Black residents will have very little impact on the magnitude of that composite score. Black and White workers have different income potentials. White males are at least two times more likely than Black males to be employed in management, business or finance positions. White females are 1.5 times as likely as Black females to possess employment in these fields. Black males and females are 2 and 1.5 times more likely to be in service oriented jobs. Therefore, Black and White residents of the same area may have inequalities in income accompanied by many differences in conditions of life, both at the individual and population level, which may adversely influence health.[125]

Perhaps separate composite scores should be calculated for Blacks and Whites for each of the census tract and examined to determine if race specific composites are influentially comparable to the composite that is not race specific. If the composites are not comparable, it may be inappropriate to assume that individuals living within the same neighborhood have access to the same resources. For instance, study findings demonstrate less physical activity among low-income housing units.[126] These finding potentially result from the likelihood that these areas not supportive of physical activity for the purposes of exercising. If Blacks are more likely to live in low income areas, adverse health outcomes associated with physical inactivity may disproportionately affect the Black population. Differential rates of large food store chains by neighborhood characteristics, such as proportion of Black population, may also contribute to the racial disparities in adverse health outcomes. Predominantly White neighborhoods tend to have

more supermarkets per household compared to predominately Black neighborhoods.[127] Large food store chains are more likely to offer healthy foods at affordable costs than small food chains. Difficulty obtaining healthy foods due to the lack of available supermarkets may result in unhealthy dietary patterns which could lead to increased risk of disease.[128]

It is possible that equalization of financial access may not ensure receipt of equal quality treatment. Policies to address unfavorable social conditions impacting health are needed. Such policies could include reduction of income inequality through tax reform, improved housing, and expanded educational and employment opportunities for the poor.[129]

Understanding health from a social perspective is important if appropriate interventions and policies are to be developed to eliminate disparities. This study analyzed Black-White disparities in stroke mortality from a social perspective that supports the assumption that health disparity among Blacks is related to unequal access to community resources. The key to decreasing the disparity is the development and implementation of policies that ensure equal access and equal treatment.[130]

Public Health Implications

Study findings suggest that racial disparity scores are elevated in deprived areas, and in some instances, even more so for lower social class groups. This suggests that initiatives to lessen Black-White mortality inequalities will need to address an individual's social class situation, while taking into account the role of residential environment in exacerbating and possibly overshadowing the effect of personal poverty. Study results suggest a change in the scope of interventions from a biomedical individual

level to interventions focusing on social determinants of health. Progress must be made to address the disjunction between individual factors and social determinants that impact racial disparities in stroke mortality. Recognizing the importance of the distribution of resources (as a measure of deprivation and wealth) among social groups is crucial to explaining the distribution of disease in populations and planning effective health interventions.

Local-level health policies must be developed with the hope of improving social, economic, physical and environmental conditions in the community that affect reducing Black-White health disparities. Efforts must be made to insure that all community members not only have access to medical services, but are additionally in a position to take advantage of these health services. Local government health officials must communicate with community members with the hopes of identifying barriers to and facilitators of the reception of available medical services. Strategies must be developed to increase access to healthcare services.

Changes in the health care system must be implemented in order to reduce disparities in adverse health outcomes. Proposed examples of beneficial change in health care include health insurance coverage for all, and racial equality in the receipt of proper medical interventions. A health care system with adequate representation of African American health professionals may also provide a positive impetus for reduction of race based health disparities by providing a more culturally sensitive, and therefore more effective, health care system.[131]

Area specific local health education programs must be initiated. Health officials must direct education efforts to specific communities within levels of socioeconomic

and/or social class. Planning must promote the community's understanding of policies and activities that will improve the community's health. It is hoped that these steps will lead to a better understanding of local community health issues and how social, economic and environmental conditions affect these health issues. Proposed changes related to the health of communities include the promotion of violence free-neighborhoods conducive to exercise, the addition of nutritious food stores, equality in income, educational and career opportunities.[131] Finally, these findings suggest the importance of repeating these analyses at the population level in additional areas as complements to analyses of single areas.

Future Research

More research is needed to gain a better understanding of the mechanisms through which the economic structure of a community influence the patterns of health and disease within and between communities. A clearer understanding, and definition of, the community in which residents live and experience life is fundamental. This can only be accomplished through contact with individuals within a defined location, and, thereby, ascertaining the location and geographic extent of social and economic interactions. Data must be compiled concerning community availability of healthy and affordable food stuff, access to recreational facilities, awareness of community influences of health and the effectiveness of the communications of health related information at the local level. Identification of utilized community resources as well as an understanding of why other resources are underutilized is important. Community barriers to healthy lifestyle opportunities must be acknowledged as well as the promoters of healthy lifestyle opportunities. Future research should develop methods to identify appropriate

populations of study within the most advantageous graphic unit. Researchers must identify the smallest geographic unit in which this type of research can be accomplished and reliable data can be obtained. Finally, efforts should be made to share research findings with the community, governing bodies and policy makers.

Conclusion

Lower ASPoH scores were predictive of higher Black-White disparities in stroke mortality at the Florida census tract level. These study results add to established literature solidifying individual socioeconomic status as a strong predictor of stroke mortality. These study results are also a contribution to our knowledge of the history of Black-White disparity in stroke mortality rates. This disparity research can be extended with the addition of information that strengthens the relationship between SES and stroke mortality by adding in the effect of an area measure of SES, and the influence that this measure has on the differences in stroke mortality rates between Black and White residents. With this study we are able to begin exploring census tract level influences of the actual Black-White disparity rate.

The literature suggests that SES does not fully account for the racial disparity in stroke mortality rates, and this study allows for the examination of group level influences of these disparities and attempts to find some type of policy resolution to these racial differences in rates. The interrelatedness of personal health behavior, social determinants, structural inequities, and institutionalized racism suggests that eliminating disparities will require large-scale, multidimensional, community-participatory interventions focused explicitly on health disparities for specific population groups, as well as on broader dimensions of social equality and economic justice.[132]

This study allows us to question the need for policy changes in resource availability and allocation at the census tract level that will make a difference. The primary purpose of this study was to examine the effect of area resource availability on Black-White disparities in stroke mortality. Results of this study support the conjecture that resource availability-related stroke mortality involves a complex combination of factors from a variety of avenues. This study may have only touched the surface of the influences that we should take into consideration when we attempt to measure the community resources that are needed to promote and maintain community health and reduce disparities in morbidity and mortality.

"…a fundamental social cause (of disease) involves resources like knowledge, money, power, prestige, and social connections that strongly influences people's ability to avoid risks and to minimize the consequences of disease once it occurs. Because of the very general utility of these social and economic resources, fundamental causes are linked to multiple disease outcomes through multiple risk-factor mechanisms…In a dynamic system, fundamental causes are likely to emerge. This is because the resources embodied in fundamental causes can be transported from one situation to another. Consequently, as health-related situations change, those with the most resources are best able to avoid diseases and their consequences. Thus, no matter what the profile of diseases and known risks happens to be at any given time, those who have greater access to important social and economic resources will be less afflicted by disease."[133]

Black-White disparities in stroke mortality present a major challenge for which effective action must focus on the social and economic environment. Analyses of individual risk may not provide useful information. Therefore, it is imperative that

researchers continue the search for modifiable aspects of the society for which changes in both policy and attitudes may be the key to unlocking the basis of the disparities in health outcomes that have existed since data such as these have been maintained.

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Appendices

Appendix A
Residential Address Geocoding Methods

Personal Communications with Bill Alfred, Florida Department of Health Tallahassee, FL
Date: August 8, 2005
Regarding: Geo-coding of death certificate residential addresses

- Florida Department of Health began geo-coding death certificates using deaths reported in 1995.
- Death certificates are moved into Access from Sequale Server
- Address correction software (Accumail) is then used to correct addresses to the postal address
- This process provides the Zip code + 4 digits, if possible
- Not all addresses can be corrected
- The Geo_result variable is an indication of how well the Accumail sort performed
- Addresses may be passed through Accumail again
- Accuracy for Accumail is 90-95%
- Addresses are then sent through Map Maker Plus, which provides latitude and longitude information
- From this information census tract information and geo_result information can be obtained
    - S5 as a georesult: Most accurate
    - Z5 as a georesult: coded to the zip+4; exact CT may or may not be good
- Data results from the Accumail sort is then run through the Map Marker Plus software in 3 to 4 batches. The difference between batched is the level of strictness utilized and the criteria is loosened for each successive batch.
- Usually take the results that get S5 as a geo_result
- This geo-coding is performed on a statewide basis
- Total death certificate records in which geo-coding was attempted
    - 1998: 157,172
    - 1999: 162,152
    - 2000: 162,840
    - 2001: 161,974
    - 2002: 163,024
- Accuracy in the geo-coding of death certificate residential addresses for 1998-2002
    - 1998: 93.7%
    - 1999: 93.0%
    - 2000: 93.3%
    - 2001: 87.0%
    - 2002: 94.1%

Appendix B
Definition of Study Variables

Exposures:

Black or African American. A person having origins in any of the Black racial groups of Africa. It includes people who indicate their race as ‘‘Black, African Am., or Negro,’’ or provide written entries such as African American, Afro-American, Kenyan, Nigerian, or Haitian.

White. A person having origins in any of the original peoples of Europe, the Middle East, or North Africa. It includes people who indicate their race as ‘‘White’’ or report entries such as Irish, German, Italian, Lebanese, Near Easterner, Arab, or Polish.

Area. For the purposes of this study, area is defined as a census tract

ASPoH. Socioeconomic conditions define the context within which the distributions of physiological and behavioral risk factors are determined. ASPoH describes features of social organization, structure, stratification, or environment, such as socioeconomic deprivation, economic inequality, resource availability, or opportunity structure. This ASPoH variable is a linear combination of the original census tract level variables subjected to principal component analysis.

ASPoH-1 is principal component number 1 (accounts for the highest percentage of variance within the census tract level variables) and thus is a linear combination of the original census tract level variables derived from principal component analysis.

ASPoH-2 is principal component number 2 (accounts for the second highest percentage of variance within the census tract level variables) and thus is a linear

combination of the original census tract level variables derived from principal component analysis.

ASPoH-3 is principal component number 3 (accounts for the third highest percentage of variance within the census tract level variables) and thus is a linear combination of the original census tract level variables derived from principal component analysis.

ASPoH-4 is principal component number 4 (accounts for the fourth highest percentage of variance within the census tract level variables) and thus is a linear combination of the original census tract level variables derived from principal component analysis.

Since ASPoH-1, ASPoH-2, ASPoH-3 and ASPoH-4 are derived from principal component analysis, they are, by definition, new independent variables.

Census Tract. Census tracts are small statistical subdivisions of a county designed to be relatively permanent. The goal is for census tracts, when originally designated, to have between 2,500 and 8,000 people and to be homogeneous with respect to population characteristics, economic status, and living conditions. Census tracts never cross county boundaries. Census tract size varies depending on the density of the population. They are designed to be fixed to allow comparisons over time but are occasionally split or combined to reflect significant changes in geography (such as the construction of an interstate) or population (rapid growth).

Dimensions of Social Determinants of Health

Economy Dimension

1. Poverty Rate. To determine a person's poverty status, one compares the

person's total family income with the poverty threshold appropriate for that person's family size and composition. If the total income of that person's family is less than the threshold appropriate for that family, then the person is considered poor, together with every member of his or her family. If a person is not living with anyone related by birth, marriage, or adoption, then the person's own income is compared with his or her poverty threshold. Poverty rate will be determined as a percentage of the total census tract population living in poverty.

2. Median Family Income. The median divides the income distribution into two equal parts: one-half of the cases falling below the median income and one-half above the median. For households and families, the median income is based on the distribution of the total number of households and families including those with no income. The median income for individuals is based on individuals 15 years old and over with income. Median income for households, families, and individuals is computed on the basis of a standard distribution. Median income is rounded to the nearest whole dollar. Median income figures are calculated using linear interpolation if the width of the interval containing the estimate is $2,500 or less. If the width of the interval containing the estimate is greater than $2,500, Pareto interpolation is used.

Employment Dimension

3. Percent Unemployed. All civilians 16 years old and over were classified as unemployed if they were neither ''at work'' nor ''with a job but not at work'' during the reference week, were looking for work during the last 4 weeks, and

were available to start a job. Also included as unemployed were civilians 16 years old and over who: did not work at all during the reference week, were on temporary layoff from a job, had been informed that they would be recalled to work within the next 6 months or had been given a date to return to work, and were available to return to work during the reference week, except for temporary illness.

4. Transportation system: This measure represents the percent of workers aged 16 years or older using various means of transportation (public versus private) to travel to work.

5. Full vs. part-time employment: This measure represents the percent of workers who work part-time compared to those workers who have full time employment.

Education Dimension.

6. Graduation rates: This measure includes the percent of population over 25 years of age without a high school degree

Housing Dimension

7. Median Rent. Median gross rent divides the gross rent distribution into two equal parts: one-half of the cases falling below the median gross rent and one-half above the median. Median gross rent is computed on the basis of a standard distribution

8. Median housing value (often utilized as a measure of wealth). (Median value of owner occupied housing units)

9. Vacancy rates: Percent of housing units vacant

10. Home Ownership: Percent of occupied housing units that are owner occupied

11. Overcrowded Housing: This value will be determined based on the mean number of persons per room

Social Class. For purposes of this study, social class will be based on educational attainment. Educational attainment was chosen as the measure of social class due to its availability on the death certificates and the belief that education is more reliable than the recorded occupation of the decedents. Decedent occupation may be considered not reliable because spouses sometimes overstate the occupation of their loved ones. Also, the categories may be over inclusive or not specific enough. For example, both a chemical engineer and an assembly-line engineer would be categorized as engineer, even though there are obvious differences in income and relative position within their respective occupations.

***Outcomes*:**

Stroke. For year 1998, stroke (cerebrovascular disease) is defined as code numbers 430 to 438 of the International Classification of Diseases (ICD), Ninth Revision. For years 1999-2002, codes I60 to I69 of the ICD Tenth Revision are used to denote death from stroke.

Age-Adjusted Stroke Mortality Rate. Age-adjusted rates are computed by the ***direct method*** by applying age-specific rates in a population of interest to a standardized age distribution (year 2000), in order to eliminate differences in observed rates that result from age differences in population composition (National Center for Health Statistics

http://www.cdc.gov/nchs/datawh/nchsdefs/ageadjustment.htm). Age-adjusted rates are calculated by the direct method as follows:

$$\sum_{i=1}^{n} r_i \times (p_i / P)$$

where r_i = age-specific rates for the population of interest

p_i = standard population in age group i

$P = \sum_{i=1}^{n} p_i$ for the age groups that comprise the age range of the rate being age adjusted

n = total number of age groups over the age range of the age-adjusted rate

Disparities. Health disparities are differences in the incidence, prevalence, mortality, and burden of diseases and other adverse health conditions that exist among specific population groups in the United States (NIH Definition).

Black-White Ratio Score: expressed as a quotient and interpreted as the relative magnitude of the Black stroke death rate compared to the Non-Hispanic White stroke death rate.

Black-White Difference Score: the absolute measure of disparity expressed simply as the arithmetic difference between the Black stroke death rate and the Non-Hispanic White stroke death rate (reference point).

Black-White Percent Difference Score: the difference between mortality rates (Black minus Non-Hispanic White) expressed as a percentage of the Non-Hispanic White death rate.

Appendix C:
Calculation Strategy for Principal Component Analyses Variables

1. Poverty rate: number of families within the census tract below the poverty level ÷ Total census tract families
2. Non-poverty rate: 100 minus poverty rate
3. Median Income: median family incomes for census tracts
4. Percent unemployed: (Males and Females in the labor force and the civilian unemployed) ÷ (Males and Females in the labor force (minus those in the armed forces)*100
5. Percent employment: 100 minus percent unemployed
6. Percent full-time employed: (Males and Females employed fulltime) ÷ (Total population 16 years and older)*100
7. Percent utilizing private transportation to work: (employed persons using private transportation to work) ÷ (employed persons using either private or public transportation to work)
8. Percent 25 years and older with High School education: (Male and Female high school graduates) ÷ (Total population 25 years and older)
9. Median rent: census tract median rent paid by renters
10. Median home value: Median value for owner-occupied housing units
11. Vacancy rate: number of vacant housing units ÷ total housing units in the census tract
12. Non-vacancy rate: 100 minus vacancy rate
13. Home ownership rate: number of owner occupied housing units ÷ number of

occupied housing units within the census tract

14. Overcrowded housing rates: (number of owner/renter occupied housing units with 2.01 or more occupants per room ÷ Total occupied housing units) *100
15. Non-Overcrowded housing rates: 100 minus overcrowded housing rates

Appendix D
Study Acronyms

1. ASPoH: Area Social Predictors of Health
2. BFAAdeathrate: Black female age-adjusted stroke death rate
3. BMAAdeathrate: Black male age-adjusted stroke death rate
4. CVD: cardiovascular disease
5. FPD: Female Percent Difference
6. MPD: Male Percent Difference
7. NH: Non -Hispanic
8. NHWFAAdeathrate: Non-Hispanic White female age-adjusted stroke death rate
9. NHWMAAdeathrate: Non-Hispanic White male age-adjusted stroke death rate
10. PCA: Principal Component Analysis
11. SES: Socioeconomic Status
12. SF3: Summary File 3
13. SF4: Summary File 4

About the Author

Tyra Dark obtained a Bachelors degree in Psychology from the University of Alabama, located in Tuscaloosa, Alabama. She received a Masters degree in Psychology from the University of South Florida. Her Masters work, within the Cognitive and Neural Sciences program, involved the study of drug effects and drug interactions. Prior to beginning the doctoral program in the Epidemiology and Biostatistics department, she conducted research of central nervous system responses to injury and worked as a Biological Scientist.