A MICRO AND MACRO ANALYSIS OF U.S. ADULT MORTALITY

A Dissertation

by

CHRISTI NICOLE RUSSELL

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree
of

DOCTOR OF PHILOSOPHY

May 2010

Major Subject: Sociology

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ABSTRACT

A Micro and Macro Analysis of U.S. Adult Mortality

(May 2010)

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This study utilized individual and county-level analysis to examine the impact of demographic and distal factors on individual level mortality as well as spatial and county specific attributes on county death rates. The data used in the individual level analyses come from the linked NHANES NDI data set and the county level data come from multiple sources including spatially referenced sources. Findings suggest that having private or Federal insurance attenuates the mortality discrepancy between racial groups at the individual level and that spatial factors have an impact on county death rates that are not explained by county attributes. These analyses present questions about using common indicators to measure mortality at different levels suggesting that the variables used to understand mortality on the individual level are different from those that should be used to understand mortality at the county level.

DEDICATION

This work is dedicated to the person who told me over and over and over and over again how important it was to finish—Bill. I am also dedicating this to Bill because I never want to hear "Trust me, I'm a doctor" again. And because he is the most wonderful partner a person could have.

This dissertation is also dedicated to anyone who bothers to read it. Thanks!

ACKNOWLEDGEMENTS

I would like to thank my committee members for helping me develop and express my ideas. Dr. Poston was incredibly encouraging and supportive throughout the research and writing process. He has been key to my development as a demographer and competent scholar. I am also very grateful for the directions and feedback given to me in a kind honest manner by Dr. Wunneburger, Dr. Fossett, and Dr. Saenz.

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CHAPTER I

INTRODUCTION

Human longevity has undergone spectacular changes in the past 100 years. Figure 1 shows that while the death rate has decreased throughout the 20th century, the average expectation of life at birth has increased from around 47 years in 1900 to around 77 years in 2000, and by 2008 was nearing 78 years (NCHS 2007; Wilmoth 1998). This dramatic gain or more than 30 years in average life expectancy is mainly due to the virtual elimination of infectious disease as a major cause of death in the U.S., which, has served to greatly increase the probability of survival, especially in the first five years of life (Bell, Wade and Gross 1992). This early life survival has provided large increases in life expectancy over the past hundred years.

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This dissertation follows the style of *Demography.*

Figure 1: Life Expectancy at Birth (upper panel) and Crude Death Rate (lower panel): United States, 1900-2000
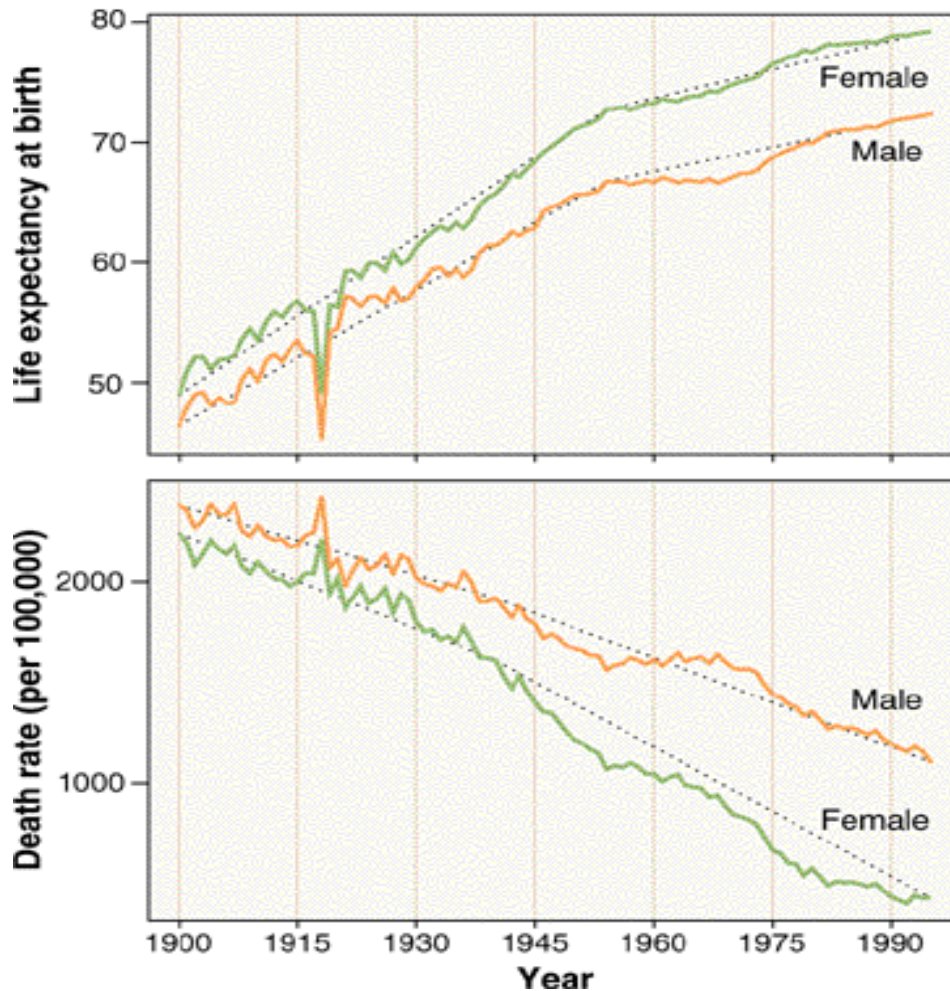
Source: (Wilmoth 1998)

As the probability of surviving to old age increased so has average life expectancy. The increase in gains in life expectancy at the older ages, however, is much more modest than the increases at the younger ages. Figure 2 shows the percent distribution of the American population by age throughout the twentieth century (Hobbs and Stoops

2002). The percentage of people reaching ages 65 and above has tripled over the past century, increasing from 4.1 percent in 1900 to 12.1 percent in 2000)(Hobbs and Stoops 2002). There have also been increases in the percentages in the 25-44 and 45-64 age groups, but these are not as great as that in the 65+ age category.

Life expectancy is not greatly increased by gains in the probability of survival at the older ages because, according to the fatalist school of thought, there are not many gains left on which to capitalize. At the older ages people are close to death so their survival gains will translate into minimal life expectancy gains. This minimal gain in life expectancy in the older ages, despite a decreasing mortality rate, is known as the Taeuber Paradox, named after the famous demographer, Conrad Taeuber who was one of the first to point out this phenomenon. The Taeuber Paradox states that the eradication of one cause of death will not eradicate death or significantly improve life expectancy; instead it will only give the survivors the opportunity to die of something else (Keyfitz 1977). In the 1970's Nathan Keyfitz (1977) showed that eradicating cancer as a cause of death would only improve life expectancy by 2.6 years because those who "survived" cancer would likely die of heart disease or some other chronic

degenerative disease as they became more susceptible to these chronic diseases with increasing age.

Figure 2: Percent Distribution of the Total Population by Age: United States, 1900 to 2000

	1900	1910	1920	1930	1940	1950	1960	1970	1980	1990	2000	
65+	4.1	4.3	4.7	5.4	6.8	8.1	9.2	9.9	11.3	12.6	12.4	65+
45-64	13.7	14.6	16.1	17.5	19.8	20.3	20.1	20.6	19.6	18.6	22.0	45-64
25-44	28.1	29.2	29.6	29.5	30.1	30.0	26.2	23.6	27.7	32.5	30.2	25-44
15-24	19.6	19.7	17.7	18.3	18.2	14.7	13.4	17.4	18.8	14.8	13.9	15-24
<15	34.5	32.1	31.8	29.4	25.0	26.9	31.1	28.5	22.6	21.5	21.4	<15
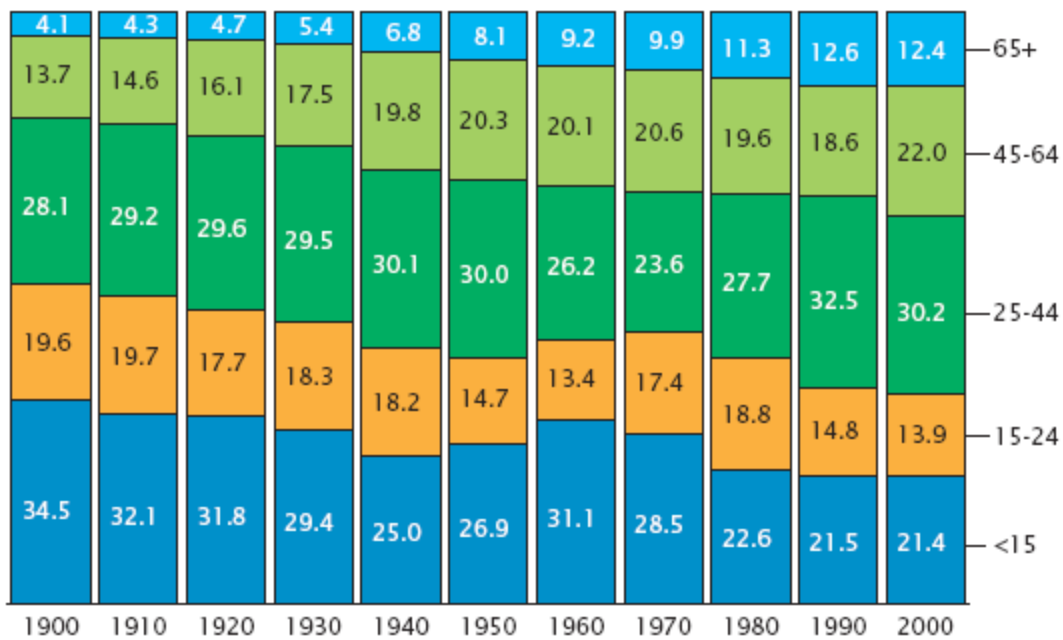
Source:(Hobbs and Stoops 2002)

The population wide access to early life expectancy gains is well established in U.S. society, but what is not as straight forward is who has access to longevity gains at the older ages. The curve of mortality increases at age 65, though the rate of mortality is not a straight line (Carey et al. 1992). It is during these later years where there is a discrepancy between extra years of longevity in a population and who has access to these gains and why. Mortality ensures that there is not a limitless life gain

available to mankind. This unknown biological limit is not
a necessarily a precise number of years that a person can
live, i.e., what is more commonly referred to as a person's
"time." Humans are capable of behaviors and actions that
can increase longevity and not only reach but surpass any
so-called biological limit to life; they are capable of
generating longevity or extra years of life.

The demographic literature has shown that "the force
of death is not the same for everyone" (Rogers, Hummer and
Nam 2000). Death is more likely to occur when people
engage in certain kinds of damaging behaviors, such as
smoking and excessive drinking, as well as if they perceive
themselves to be in poor health or have a lower social
status. On the other hand death is less likely to occur
for the insured, the well educated, and those who live in
the western portion of the U.S. (Rogers, Hummer and Nam
2000).

Human behavior can impact longevity but there are also
social factors that over which individuals have little
control, but will still exert influence on individual
longevity. The structure of U.S. society must be taken
into account when considering life expectancy gains for the
population. American society is stratified by race and
class (Saenz and Morales 2005, Sakamoto and Powers 1995).

All too often the poor and minority populations are denied access to the protection American society gives to its majority members. Accordingly, the poor and minority populations of America are more vulnerable to mortality and do not have as much access to longevity protections.

The poor population of the U.S. does have access to government funded health care that may provide access to longevity protections. Medicaid is the government health insurance for the poor and the uninsured. Medicare is the government insurance program for those over 65; part A is hospital insurance that most people do not pay for (people who work pay Medicare taxes throughout there working life), and part B is medical insurance for which most people pay a monthly premium. Medicaid and Medicare grant people access to moderate amounts of health care. However, Medicaid does not provide access to much preventative care, with the exception of some immunizations and prenatal care. According to the Department of Health and Human Services Medicare does provide a limited amount of preventative care including a onetime physical exam, cholesterol screening once every 5 years, various cancer screenings for four types of cancer every one or two years depending on the type of cancer, seasonal influenza immunizations, and diabetes screening as deemed necessary by a physician.

The limited access to preventative health care provided by government insurance may be the main reason behind the increased mortality of people who only have government health insurance compared to those with private insurance (Sorlie et al 1994). Persons with private insurance have been shown to live longer than those who rely solely on government insurance, but the effects of health insurance are not straightforward. The World Health Organization (WHO 2000) has released a report evaluating the health systems of the world and found that the U.S. spent more of its GDP on health expenditures than any other country but failed to be the healthiest country in the world or have the most equitable distribution of good health among the population. Of the money spent on health expenditure in the U.S., forty-four percent is from public sources and the remaining fifty-six percent comes from private sources (WHO 2000). Despite the fact that the U.S. spent the most money in the world on health services and over half of the money came from private sources (forty percent of which was from private health insurance), the people of the U.S. are not the longest lived people in the world (WHO 2000). This suggests that the relationship between health care and longevity is anything but simple.

Complicating the relationship between American's and their longevity is the vastness of America. Not only do people exist in a social context but they also exist in a geographic context. In previous demographic research it was thought that regional differences in mortality and longevity patterns were due to the attributes of the population living in the region. It may be that the population does not deserve sole credit for the variation since inequality in the population does not always translate into mortality inequality in the region or to the degree that is present in the population (Lynch 2004).

In this dissertation I will be examining the variation in longevity after the curve of mortality has begun to rise. I hope to contribute to the overall body of literature on the state of longevity in the U.S. In my dissertation I will analyze several factors that have been shown to impact longevity, namely, inequality, health insurance and geographical context. In considering these factors, I will put together a cohesive picture of longevity in contemporary America. Individual and aggregate analyses will be utilized in order to consider these factors in the appropriate context.

The next chapter of this dissertation reviews and discusses the current literature dealing with inequality,

health insurance and geographical context and their relationships and effects on human longevity.

CHAPTER II

LITERATURE REVIEW

In this chapter I will review theory and literature dealing with adult mortality and longevity as well as the relationship between spatial attributes and mortality. Mortality will be put in a historical context and current discrepancies between individual factors as well as spatial factors and their impact on life span will be evaluated in order to orient the reader to the state of mortality today.

Longevity Advances

Of all the achievements of the modern world, the increase in the human lifespan is one of the most remarkable. Since John Graunt constructed the first life table in the 1660s to describe the rate of death by age, the increases in longevity have been nothing short of incredible. Modernity has brought to the majority of the population the opportunity to survive to old age, a feat that was unheard of during Graunt's time (Glass 1955, Kung et al. 2005). Graunt's simple table showed that only three percent of 17th century England could be expected to live to age 66. In the U.S. today over 83 percent of the population can be expected to live to age 65 (Glass 1955, Kung et al. 2005).

Looking at a 400 year time frame shows the remarkable

improvements in the probability of survival to old age. However, viewing the past one hundred years shows the incredible advances of modernity in a starker light. By comparing three individuals born over an 80 year period (starting in 1929 and ending in 2004), it becomes evident how longevity has changed and which age groups are most advantaged by these changes.

My Grandmother, Estella, was born in 1928 and died when she was 78 years old. When Estella was born, U.S. life tables showed that, on average, she could be expected to live to age 62, a figure she surpassed by 16 years (Hill 1936). When I was born in 1978, U.S. life tables showed that I could expect, on average, to reach age 77 and Estella (then 49) could expect, on average, another 31.6 years (Rice 1978). My niece Maddie was born in 2004; U.S. life tables showed that she could be expected to live until the age of 80, Estella (then 73) could expect to live another 14 years and I (then 26) could expect to live another 55 years.

It is in the later years when Americans fail to reach their life-expectancy potential. As my grandmother aged, her life expectancy increased past her life expectancy at birth and though she realized her life expectancy at birth she failed to realize her life expectancy at her older

ages. When Estella was 49 she was expected to live until

age 80, when she was 73 she was expected to live until age

87. Estella died at age 78 missing her life expectancy at

age 49 by two years and her life expectancy at 73 by 9

years.

Table 1 contains data showing the average life

expectancy increases for key dates in mine, Maddie's and my

Grandmother's life times. I have shown the average life

expectancy at birth, at age 30, and at age 65; it is this

later age when old age starts and when the curve of

mortality becomes steeper. The dramatic differences in

life expectancy at birth from 1928 to 1978 and from 1928 to

2004 (15.1 and 18.1 years) reflect a time of change in the

U.S. that saw the dramatic increase of survival at the

younger ages (Bell, Wade and Gross 1992). Compare the

gains in life expectancy at birth gains between 1928 to

2004 with those between 1978 to 2004 (18.1 years versus 3

years).

The gains at age 30 (9.6 years from 1928 to 2004 and

2.3 years from 1978 to 2004) reflect the near eradication

of infectious disease as the leading cause of death in the

U.S. in the first half of the twentieth century. The life

expectancy gains at age 65 are much less dramatic than at

birth, or at age 30. From 1928 to 1978 there was a 5.6

year gain, and a further increase of 1.6 years by 2004.

These diminished gains reflect the abbreviated potential

life expectancy left to the population as it reaches its

maximum life span.

Table 1: Average Life Expectancy for Total U.S. Population

	1929	1978	2004
e0	54.7	73.3	77.4
e30	35.5	46.6	49.8
e65	11.9	16.3	18.4

Source: Arias 2007, Rice 1978, Hill 1936

The assumption that there is a maximum life span is

not universally recognized in the demographic literature.

Mortality and longevity scholars who work under the premise

of a longevity maximum belong to the fatalist school of

mortality research (Carnes, Olshansky and Grahn 1996). The

fatalist argument extends from the often repeated

observation that the limits apparent in the biological

attributes of the human body (e.g., flexibility, height,

organ function) may be extended to the human organism as a

whole thereby arguing that there is a biological limit to

human longevity (Fries 2005). It is but a small leap from

accepting the limits of human ability to extending the

argument and accepting limited human longevity. Senescence

is a powerful argument against immortality.

On the opposite end of the spectrum is the belief that the life-span is relatively indeterminate (Carey and Vaupel 2005). This is the more optimistic school or perspective. The evidence that mortality and longevity scholars use to support the idea of an indeterminate life-span is prominent in today's demographic literature. The deceleration of the mortality rate at old ages along with the tendency towards a rectangularization of the survival curve, and the increasing decline of old age mortality, all offer powerful support for the belief that humans are not bound by finite years of life (Yashin et al. 2001). If persons were to eat, exercise and produce less free radicals, they could potentially live to an indeterminate age.

Another area of research that lends support to the pro-longevist (or optimistic) model of life-span is experimental research performed on different species. Model system research done on yeast, drosophila and worms usually shows positive longevity outcomes when free radicals are reduced and food and environment are manipulated (Carey 2003). By showing that it is possible to increase the life-span through environmental and behavioral manipulation of less complicated species, the pro-longevist school extends their findings to the

possibility of increasing life-span in humans through similar manipulations.

The debate between the pro-longevist and fatalist schools will likely continue beyond the near future. Fatalists argue that the pro-longevist model trades longevity for hardiness. Subscribers of the pro-longevist school point out that the fatalists are not able to accurately report what the maximum life-span is.

My research is strongly in the fatalist camp. Despite the huge gains in life-expectancy over the past 100 years, it is also still evident that every life table since their inception shows a continuously diminishing number of people surviving at each successive year and not a single one has ever survived past 122-123 years (Olshansky and Carnes 2001).

Inequality in Longevity

Figures 3 and 4 demonstrate the inequality in longevity advances over the past eighty years. The figures illustrate the differences in life expectancy at every age for white and nonwhite males and females; these two figures are a graphic representation of life expectancy as reported in U.S. life tables (Arias 2007, Rice 1978, Hill 1936).

These two graphs serve to unmask important demographic generalizations in life expectancy. Life

expectancy is impacted by race and by sex and further

impacted by the social and economic characteristics that

are, to a degree, influenced by race and sex (Rogers,

Hummer and Nam 2000). At every age a white woman is

expected to live longer than a white man, and a nonwhite

woman is expected to live longer than a nonwhite man.

Until age 70 nonwhites are not expected to live as long as

whites.

Figure 3: Female Life Expectancy

Female Life Expectancy 1927-2003
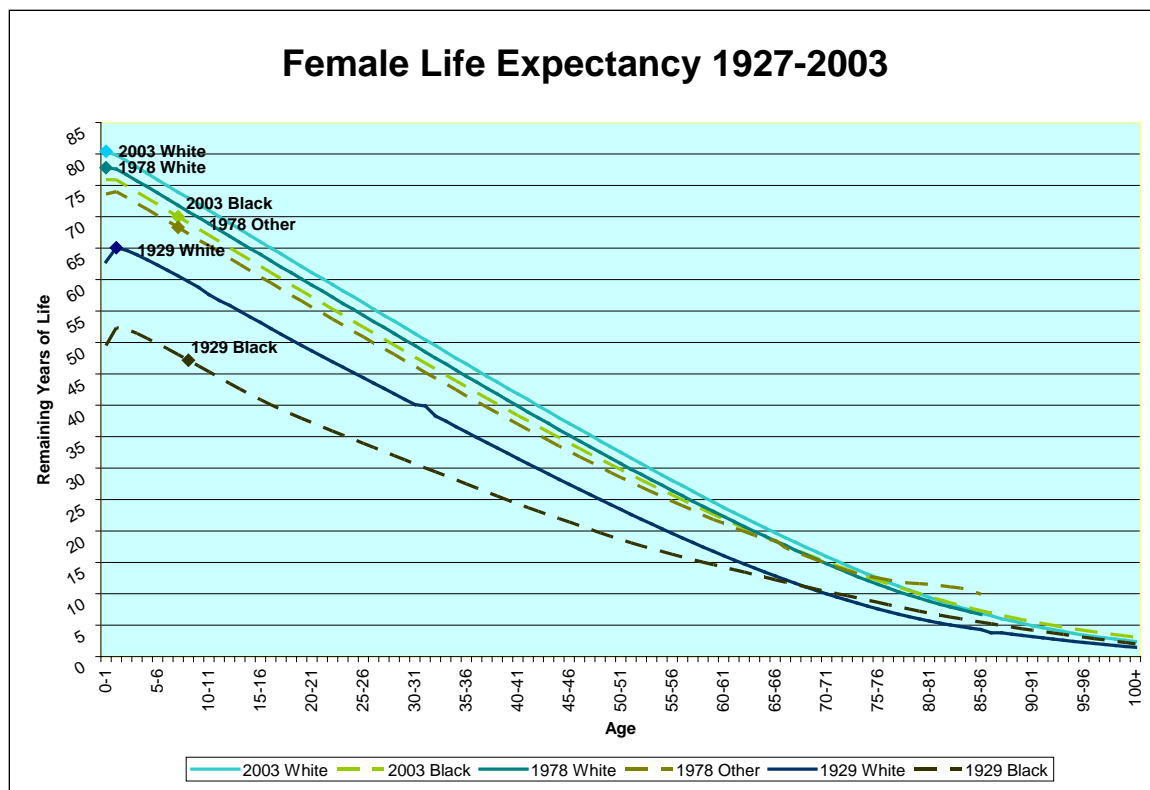
Source: Arias 2007, Rice 1978, Hill 1936

Figure 4: Male Life Expectancy

Male Life Expectancy 1927-2003
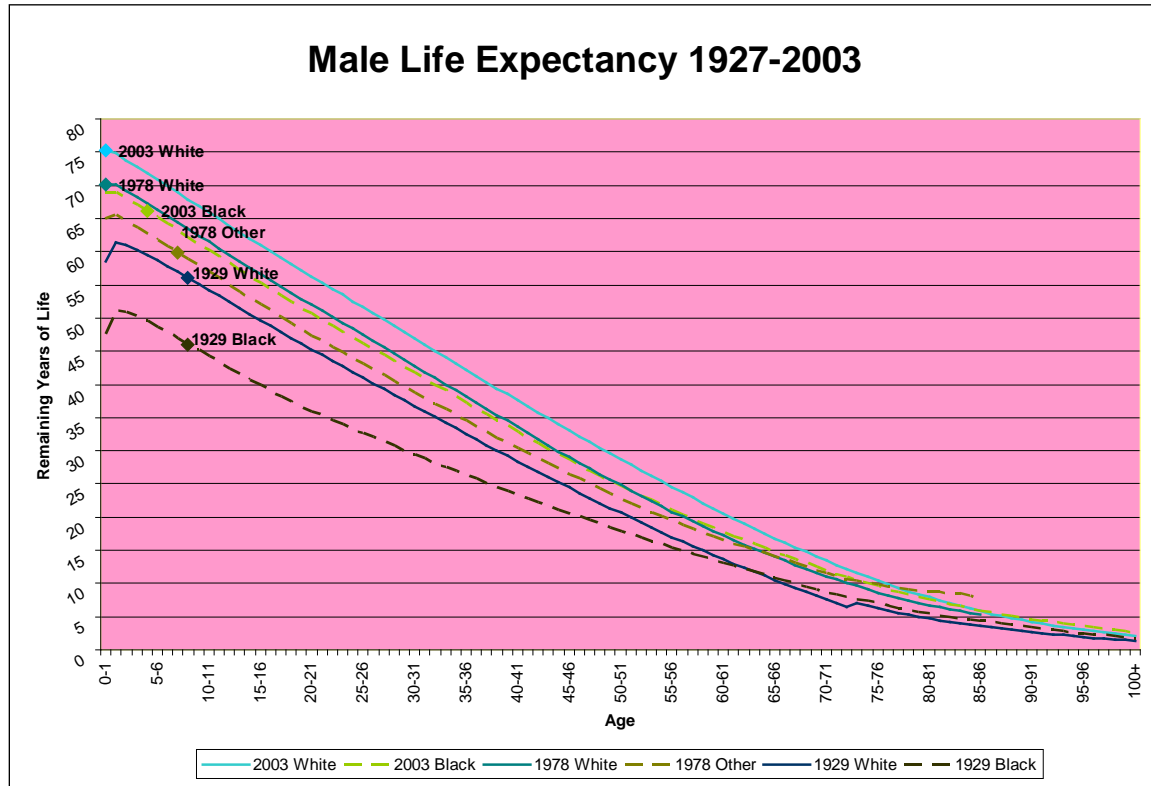
Source: Arias 2007, Rice 1978, Hill 1936

It is important to be mindful of the demographic, distal and proximate factors that affect longevity (Rogers et al. 2005) and thus realize that the structure of society has far reaching impacts on an individual's life expectancy. There are demographic or ascribed factors that impact longevity, over which individuals have little or no control. These include sex, race and ethnicity, and age. There is little the individual can do to attenuate their influence on their longevity.

In America, women are expected to live five years longer than men (PRB 2005). Rogers, Hummer and Nam (2000) showed that women maintain a longevity advantage even after controlling for detrimental behaviors and economic factors. They hence concluded that there is a type of biological or economic factor connected with being female that provides a buffer against mortality.

The effects of race and ethnicity on mortality are not as obvious and as pervasive as those of sex. Those who classify themselves as "not white" can be expected to have abbreviated years of life compared to whites (Rogers et al. 2005). This relationship seems to be linked to the unequal social and economic status of non-whites. This can be inferred by the evidence that race and ethnicity do not impact mortality in every culture, though the relationship between mortality and social class does translate across cultures (Marmot, Kogevinas and Elston 1987).

The effect of social class is a complicated one in the study of mortality and longevity. It is a distal cause, influenced by demographic factors, and has an enormous impact on people's lives. A person's social class determines one's socioeconomic status and"individuals at the lowest socioeconomic levels are several more times as likely to die as individuals at the highest socioeconomic

levels" (Hummer, Rogers and Eberstein 1998:553).
Socioeconomic status also has an effect on other factors
that influence mortality, such as the level of achieved
education, the way the person interprets and treats
disease, lifestyle behaviors, place of residence, and, most
importantly, whether a person has health insurance, and if
yes, the type.

Rogers, Hummer and Nam (2000) have conducted a
comprehensive study of adult mortality and show clearly the
important effects of factors such as education, income and
employment status on mortality. Those with less than 12
years of education were 60 percent more likely to die than
those with more than 17 years of education; indeed, with
every reduction of every level of educational attainment,
there was an increase in the hazard of dying (Rogers,
Hummer and Nam 2000). The same may be said for income.
People at the highest income level were 101 percent less
likely to die than those at the lowest income level (Rogers
et al. 2005).

Health Insurance and Mortality

Medical advances, along with general health and
sanitation, over the past 100 years have seen the virtual
eradication of small pox, scarlet fever and bubonic plague.
Medicine is also responsible for making numerous diseases

and disorders non-lethal that would have resulted in death

sentences in the 1800's (White and Preston 1996). White

and Preston (1996) conducted a clever thought experiment

and were able to show what the population of the U.S. would

be if death rates were frozen at 20 year intervals from

1900 to 2000 (see Figure 5). The chart shows dramatic

population gains up until the 1960's, the period when

communicable diseases were being eradicated as the major

cause of death. The next era in mortality from the 1960's

to 2000 show a much less dramatic population gain. This is

due in large part to the fact that the medical advances

that were occurring to treat the new leading cause of

mortality, i.e., chronic degenerative disease, were much

more specific in nature and tended to benefit a fewer

number of people.   From 1960 on, medical gains and amazing

medical technology has become available and has allowed the

medical profession to perform surgeries and interventions

that give very few people access to increased longevity.

This specific advancement has been translated into much

slower population growth (White and Preston 1996).

Figure 5: Population Estimates Based on Historical Death Rates

Growth in specific medical gains that benefit few
members of society may be thought of as unequal access to
longevity. The access is limited in two ways. The first
is that these specific medical gains may not apply to every
person; every specific disease may not be treatable by
these methods. The second is that all people who would
benefit from these specific medical gains may not have
equal access to them. This could be due to an inability to
pay for costly medical procedures, limited spatial access,
and/or lack of knowledge about the procedures (Bell, Wade
and Gross 1992; White and Preston 1996).

Health insurance has an obvious link to longevity protection because one's possession of insurance increases one's access to medical treatment and medical prevention of disease. Having access to life saving emergency treatments will often mean the difference between certain death and added life. Besides the immediate need for emergency treatment, health insurance also provides access for preventive medicine and the treatment of acute and chronic disease (De Flora et al. 1996). As communicable disease has waned as the leading cause of death, chronic degenerative disease has risen to replace it. The impact of chronic degenerative diseases on health care is substantial. Unlike communicable disease, a simple one time treatment and round of medication is unlikely to cure a chronic degenerative disease. The diagnosis of chronic degenerative disease usually means continuous medical care to "treat," not to "cure" the disease.

As the structure of disease has changed in America, so too has health insurance. The business of health insurance has numerous goals, one of which is cost effectiveness (Fox 2005). During an age of chronic degenerative disease, cost effectiveness in the form of higher deductibles, a set price list for services, and reduced coverage, have come to be characteristics of the prevalent health care business

model (Fox 2005). This business model of health care has the, perhaps unintentional, consequence of increasing the cost of coverage. Under these circumstances health care becomes unaffordable to many children and adults, leaving those who cannot afford coverage and are in poor health in a situation that will almost certainly reduce their life-span.

Along these lines, Ayanian and colleagues have noted the following:

> Uninsured adults generally encounter greater barriers to preventive services and treatment of chronic illnesses than to acute care. They are more likely than insured adults to report poor health status, delay seeking medical care, and forgo necessary care for potentially serious symptoms. Uninsured adults receive fewer screening services for cancer and cardiovascular risk factors, present with later-stage diagnoses of cancer, and experience more avoidable hospitalizations. They also face an increased risk of death, particularly when hospitalized(Ayanian et al. 2000)

Of course, health care coverage and access to increased longevity are not free. McWilliams and colleagues (2004) have shown a significantly greater rate

of mortality among the near elderly (age 55-64) without health insurance, compared to those with health insurance, in a study that followed participants over eight years. In the McWilliams study, evidence points to an excess of 105,000 deaths that were attributable to lack of insurance (McWilliams et al 2004).

Most Americans who are very poor and display a medical need are eligible for Medicaid, though Medicaid does not cover preventive health care for most eligible people. Medicare is available universally to those over the age of 65 (Rogers, Hummer and Nam 2000). Private health insurance can be bought directly or may be provided partially or fully by an employer. Though it seems logical to assume that any health insurance is better than no health insurance, this is not always the case. Bixby, Dow and Lacle (2005) followed a cohort of 876 people age 60 and over (in Costa Rica) from 1984 to 2000 in order to measure the effect of health insurance on their mortality risks. The results of the Costa Rican study show a highest survival rate for the uninsured followed closely by the group who had been insured by an employer, and the lowest survival rate for the poor who were granted insurance by the government (Bixby, Dow and Lacle 2005).

A similar study was conducted in the U.S. Sorlie and associates (1994) utilized a data set that followed U.S. participants age 25 to 64 years of age for eight years. They did not include people of age 65 and over owing to the difficulty in ascertaining the impact of being elderly and uninsured in the U.S. because of the pervasiveness of Medicare after age 65). Their findings were similar to those in the Costa Rican study in that persons who had government insurance (Medicare or Medicaid) had a higher rate of death than persons with private employer insurance. Though the Costa Rican and American government health insurance plans are not the same, these two studies give support to the idea that the quality of government health insurance is important when considering its impact on life span; it has not been shown to be as effective as private insurance in increasing longevity.

Geography and Mortality

Another factor that deserves recognition in analyses of factors contributing to longevity is the person's or population's geographic context. Regional variation in U.S. mortality has been demonstrated consistently with the South having higher age-adjusted mortality rates and the West the lowest rates (Zopf 1992; Lanska and Kryscio 1994). The above and other studies have considered these regional

mortality differences to be a function of social inequality, and not geography. Though this research is certainly valuable, it does seem to discount place. It must be considered that geography per se is also a social context; most people have the free will to choose where they live, but at the same time the context/location in which an individual or population lives will also determine (to an extent) their life chances. When we add to these considerations Tobler's first law of geography that "everything is related to everything else but near things are more related than distant things (Waldo 2004)," the relationship between longevity and geography becomes multifaceted. Teasing out the effect of geography on life span is a multifaceted issue that deserves special consideration.

Mitchell, Dorling and Shaw (2002) have attempted to address the relationship between geography and mortality in Great Britain. Previous studies conducted in Britain had shown that the geographic areas with the highest level of disparity in socioeconomic status also had the poorest mortality outcomes (Ben-Shlomo, White and Marmot 1996; Wilkinson 1992). Mitchell, Dorling and Shaw (2002) incorporated descriptive variables about the population (age, sex, social class and employment status) in order to

distinguish between the compositional and contextual effects of geography. They found that after controlling for the descriptive variables, geographical context maintained an influence in distinguishing between excess mortality and lower than expected mortality. This study gives evidence to support the assumption that geography does indeed have an impact on longevity that is not associated with the characteristics of the population (Mitchell, Dorling and Shaw 2002). Additional research has also shown that risk factors and individual/population characteristics alone do not account for all the geographic variation in mortality rates(Balarajan and McDowall 1988).

Other mortality scholars have isolated in their research certain aspects of geography as they relates to mortality. The impact of pollution is often considered when considering the relationship between place and mortality. Both Cox proportional hazard modeling and spatial modeling have been used to show a significant relationship between fine particulate and sulfur oxide-related pollution and lung cancer and cardiopulmonary death (Pope et al. 2002). There is also evidence that the relationship between mortality and air pollution may be cumulative in that cause-specific mortality at the older ages showed a significant relationship with air pollution,

whereas a similar relationship was not found at the younger ages did not (Gouveia and Fletcher 2000). Hospital utilization has also been studied in analyses of the relationship between geography and mortality. In the U.S. positive spatial relationships have been shown to exist between stroke fatality and geographic specific patterns of hospital utilization (Lanska and Kryscio 1994). Research examining geography and access to adequate hospital services has showed that there is a significantly higher rate of mortality for patients in rural hospitals compared to those in urban areas; this suggests that lack of access may be responsible in part for the higher mortality rates in rural areas (Hand, Klemka-Walden and Inczauskis 1996).

This chapter has reviewed some of the main literature dealing with longevity and mortality. The literature dealing with longevity is vast. In this dissertation I will focused on four major factors that influence longevity: longevity advances, inequality, health insurance, and geography. Studies reviewed in this chapter have shown that these factors influence longevity in different ways, some providing longevity advantages and others providing longevity disadvantages.

The next chapter of this dissertation will focus on data, methods and hypotheses. I first look at the data

sets I will use in my analyses. Next, I discuss and review the methods to be used. I then conclude the next chapter with a statement of the hypotheses that will be tested in the subsequent chapters.

CHAPTER III

DATA, METHODS, AND HYPOTHESES

In this chapter I provide a description of the demographic and spatial data sets I will use in this dissertation. All of the variables to be used in the dissertation analyses are defined in this chapter. I also include brief explanations and rationales for both the individual ordinary least squares (OLS) regression models and the aggregate Moran's I and resulting logistic regressions. This chapter concludes with a specification of my hypotheses.

The Demographic Data

I have the exciting opportunity to take advantage of the commitment of the National Center for Health Statistics (NCHS) to "maximize the scientific value of population-based surveys" (NCHS 2007). As stated by the NCHS:

NCHS is currently linking various NCHS surveys with death certificate records from the National Death Index (NDI). Linkage of the NCHS survey participants with the NDI provides the opportunity to conduct a vast array of outcome studies designed to investigate the association of a wide variety of health factors with mortality.

In this dissertation I use the Third National Health and Nutrition Examination Survey (NHANES III) which was administered between 1988 and 1994 and which was based on a complex multi stage sample plan. The NHANES III dataset has three parts: a survey administered by a trained interviewer; an examination by a licensed medical doctor; and a blood and urine lab analysis. The NHANES III dataset is particularly suited to longevity studies in that the survey questions cover demographic, health service utilization, and a number of behavioral variables. Tobacco use is one of the most important behavioral variables in predicting longevity, making access to tobacco use, when considering mortality, vital (Mokdad et al. 2000).

After the completion the NHANES III survey instrument, participants over the age of 17 whom then died were matched with the National Death Index (NDI) up through the date of December 31, 2000. Based on seven key identifiers from the NHANES III dataset, participants were linked to the NDI files using probabilistic matching (NCHS 2007). The resulting file contained 20,024 study participants over the age of 17 whose mortality status, as of 12-31-2000, has been established, that is, whether or not they died. I restrict my sample to the participants aged 25 or more who experienced death; my subjects thus are likely to have

completed schooling and have begun career employment. Such a restriction to the over 25 population leaves a sample size of 2,566 dead persons.

Definition of the Individual Level Variables

The demographic analysis utilizes a single dependent variable, age at death. This is a calculated variable. Age at death was found by adding the respondents' age (in months) at the date of the initial interview with the months that then elapsed from the date of interview until the date the respondent died. Date of death is provided by the NCHS through a probabilistic matching between the NHANES III and the NDI (NCHS 2007). The NCHS utilizes strict criteria in which seven parameters must be met in order to positively link a respondent to a death certificate, indicating a high level of technical accuracy in the date of death (NCHS 2007).

I utilize these linked data to examine the effects on age at the date of death of ascribed characteristics, social and economic characteristics, behavioral and health factors, and the presence of insurance on longevity. The presence of life insurance is my key independent variable. I direct the reader to the following list for a detailed specification of the independent variables to be used in the demographic analysis.

1. Female

This is a dummy variable coded 1 if the respondent is female and 0 if the respondent is male.

2. White

This is a dummy variable coded 1 if the respondent self-identified as White and 0 if the respondent categorized himself/herself as any other race or ethnicity. When breaking out the data by race and ethnicity it was my original intention to have the category "other" contain respondents who did not fit in White, Black, Mexican-American, or Hispanic categories. This other category would only have contained eleven respondents and because of this small population I have grouped "other" in with the Whites.

3. Black

This is a dummy variable coded 1 if the respondent self-identified as Black and 0 if the respondent categorized herself/himself as any other race or ethnicity.

4. Mexican-American

This is a dummy variable coded 1 if the respondent self-identified as Mexican-American and 0 if the respondent categorized himself/herself as any other race or ethnicity.

5. Hispanic

This is a dummy variable coded 1 if the respondent
self-identified as Hispanic and 0 if the respondent
self-identified as any other race or ethnicity.

6. Medicare

This is a dummy variable coded 1 if the respondent was
enrolled in Medicare at time of interview and 0 if the
respondent was not enrolled in Medicare.

7. Medicaid

This is a dummy variable coded 1 if the respondent was
enrolled in Medicaid at the time of the interview and
0 if the respondent was not enrolled in Medicaid.

8. Private

This is a dummy variable coded 1 if the respondent was
enrolled in a private insurance plan at the time of
the interview and 0 if the respondent was not so
enrolled.

9. Married

This is a dummy variable coded 1 if the respondent
self-identified as married, or living as married, and
0 if not.

10. Education

This is a categorical variable that ranges from 0 to
17. The categories correspond to the highest grade

the respondent completed. The 0 category is assigned
to persons who either never attended school or only
completed kindergarten.

11. Poverty

This is a ratio of the respondents reported annual
income to the 2000 poverty threshold.

12. Self Health

This is a self-reported categorical variable coded 5
for excellent heath, 4 for very good health, 3 for
good health, 2 for fair health and 1 for poor health
and 0 for very poor health.

13. Tobacco

This is a dummy variable coded 1 if the respondent was
currently using tobacco, namely, cigarettes, cigars,
pipe, chewing tobacco or snuff, and 0 if not.

Models 1 through 10

I will be estimating 10 ordinary least squares
regression models with age at death as the dependent
variable and the above ascribed, social and economic,
behavioral and health and insurance characteristic as the
independent variables. Ordinary least squares regression
is appropriate for this analysis for a number of reasons.
First, the dependent variable is continuous and

(theoretically) unbounded and there are no perfect or high levels, of collinearity between the independent variables.

I use nested models to show whether or not having insurance attenuates the effects of the ascribed, social and economic and behavioral characteristics. So I will be estimating several models, and these will not be hierarchical, that is, each model may or may not contain variables in the previous models. The first model will be comprised of age at death as the dependent variable, and ascribed characteristics as the independent variables. In the second model I add insurance to glean its impact on age at death. The third model will add social and economic variables to the model without including insurance while the fourth model will include ascribed characteristics, social and economic variables and insurance. The fifth model will include ascribed characteristics and health variables in the model, while I add insurance in the sixth model. The seventh model includes ascribed characteristics and behavioral variables while the eighth model adds insurance to these variables. The ninth model will include all variables with the exception of insurance. The final model, or complete model, contains all the independent variables to test the combined impact of ascribed, social and economic, behavioral and health variables on age at

death and to measure how insurance affects the outcome, as well as the impacts of the other independent variables on age at death.

County Level Data

In analyses of this dissertation that will have spatial components, I will use multiple data sets that are geographically referenced. The first geographically referenced data set was developed by the U.S. Census Bureau. The Census Bureau provides cartographic boundary files for the U.S. in their TIGER/Line Files. The TIGER/Line file I use contains the boundary data for all U.S. counties.

Another set of spatially referenced data came from the Columbia University's Socioeconomic Data and Application Center (SEDAC). It is the Gridded Population of the World, version 3(GPWv3) dataset. The GPWv3 dataset has population density per square kilometer for most countries in the world, including the U.S.A. The GPWv3 population numbers are based on United Nations population figures. I utilize the 2000 GPWv3 data for the United States at the county level. More current population data are available, but I decided to use the 2000 data to be consistent with the individual level data. In this way it may be possible to

build a picture of mortality at the individual and county level for the year 2000.

 A third data set is from the CDC Wonder online data base that has extensive county level mortality data as well as demographic characteristics by county. I am able to gather age-specific mortality rates for each U.S. county as well as cause-specific mortality rates for each U.S. county. I utilize these data for the year 2000.

 I gather Age-Specific mortality rates for three age groups, 65-74, 75-84, 85 and above. I do this in order to be consistent with my earlier reasoning positing that by analyzing age-specific mortality rates (ASMRs) for those over 65, I anticipate a more meaningful analysis because of the steep increase in the curve of mortality after the age of 65. I break the ASMRs into three groups for the same reason, that is, I expect the force of mortality to increase at each age group as the curve of mortality becomes steeper. Thus, restricting the ASMRs into smaller over 65 categories should allow for the measurement of more homogenous age groups with respect to mortality behavior.

 The final data set to be used in my spatial analyses is drawn from the U.S. Department of Health and Human Services (HHS) website (www.hhs.gov/). The HHS website contains the Centers for Medicare and Medicaid Services

(CMS) records, both current and historical. I am able to find and utilize the total per capita (by county) fee for service Medicare expenditure from the CMS page of the HHS site. I have decided to use the Medicare fee for service numbers as indicators of insurance expenditures in the county for several reasons. The first is that there are no trustworthy private insurance expenditure data available by county. Private insurance is also able to charge different rates based on the economic conditions of the area of service, and no such variation occurs in Medicare fee for service expenditures. In 2000, public monies accounted for 45.2percent of the medical expenditures (CMS 2002). Of these public expenditures 32.9percent of the monies were paid by Medicare or Medicaid programs (CMS 2002).

The spatial analysis is a multi-step process that involves creating data layers and formatting data so that they are useable in ArcGIS mapping software. I will utilize the centroid shape file of the GPWv3 data that represents the population density of the U.S. I will then join the GPWv3 layer with the Census boundary layer and, in effect, superimpose the official U.S. government recognized county boundary areas over a grid of the U.S. that contains density information. I do this in order to create mean population density center points for each U.S. county in

order to estimate global and local autocorrelation. It is important to use actual population density center points for each specific county in order to avoid arbitrary geographic points in the calculation of spatial statistics. After defining the mean population density center for each U.S. county, I will join the county specific Medicare expenditure attribute data to the county density layer in order to create a master layer. This master layer will contain the ASDRs and the CSDRs which will be used as dependent variables in the measurement of autocorrelation between the counties.

I will then estimate a series of Moran's I statistics for each of the three ASDR and three CSDR layers using inverse distance squared weighting to accommodate the expected pattern of relationship between density points. Inverse distance weighting is particularly beneficial in this instance because it places more weight on the nearest counties and less weight on the relationships of counties with other counties as the distance between the counties increases. The ASDRs and CSDRs of nearby counties should have a greater relationship than the relationship between ASDRs and CSDRs of counties that are farther apart. If a global spatial relationship is displayed in the U.S. (measured by a significant Moran's I coefficient), the

analysis will be extended and a Getis-Ord G* will be estimated using inverse distance squared weighting in order to display patterns of clustering or dispersion of ASDRs and CSDRs.

The Getis-Ord G* statistic will be used in a regression model with ASDRs and CSDRs as the dependent variables and county attributes as the independent variables. The resulting coefficients will be standardized to determine that portion of the explained variation in county level ASDRs and CSDRs that can be explained by spatial factors (Weeks et al. 2000).

Definition of Spatial Variables

The spatial analysis utilizes multiple dependent variables, three ASDRs and three CSDRs. Each of these dependent variables is a rate based on mortality at the county level for the year 2000. The six dependent variables are as follows:

1-3. 65-74 ASDR, 75-84 ASDR and 85+ ASDR

The age-specific rate is found by dividing the number of people who died in the age groups 65-74, 75-84 and 85 and older, by the population of that age of the county in which they were a resident during their life.

4-6. Neoplasm CSDR, Stroke CSDR, Ischemic Heart Disease CSDR

Each particular cause-specific death rate is found by
using the cause of death data on the death certificate
for those over 65 as the numerator, and dividing the
number of cause-specific deaths for either cancer, stroke
or heart disease by the population of the county in which
the decedent was a resident.

I utilize these spatial data to look at the relationship
of county death rates on their surrounding counties. In
order to determine whether the mortality behavior of one
county is related to its neighbors I utilize a set of
spatial data that will remain the same for every spatial
model. The spatial data are the mean population center of
each particular county that is based on the geographic
point of the county at which the population is evenly
distributed around.

Models 11 through 16

I will estimate a Moran's I to determine whether or
not there is clustering, dispersion, or no relationship
between the dependent variable (65-74 ASDR, 75-84 ASDR, 85+
ASDR, Neoplasm CSDR, Stroke CSDR and Ischemic Heart Disease
CSDR) across the counties of the U.S.

If the Moran's I results in absolute values of 2.0 or
more, I will interpret this to indicate that there is a
significant amount of dispersion or clustering of

ASDRs/CSDRs across the U.S. and it is appropriate to test for local levels of autocorrelation between the counties. In order to test for clustering or dispersion of the ASDRs/CSDRs between the counties, I will estimate a Getis Ord G using the six ASDR/CSDR dependent variables and the county population density. The resulting G statistic is based on a Z score and there is a score returned for each county in the analysis. If the Z score is greater than an absolute value of 1.96 then the county's mortality behavior is significantly related to the mortality behavior of the surrounding counties.

I will then estimate six OLS models with the three ASDRs and three CSDRs as the dependent variables. The main independent variable will be the Z score taken from the Getis Ord G analysis. Other independent county level variables will be the per capita Medicaid expenditure by county and the mean age of the county in the year 2000. The resulting OLS coefficients will be standardized to determine how much effect the spatial relationships between counties account for the variation in mortality.

Hypotheses

I will test a number of hypotheses based on my extensive review of the relevant literatures. The tests of my hypothesis will be broken into three parts; Models 1-10, spatial models 11-22 and models 23-28.

For models 1-10:

1. There will be a significant relationship between age of death and ascribed, SES, behavioral and health variables.

2. This significant relationship will be impacted positively by the presence of private insurance.

For models 11-16:

3. There will be significant amounts of clustering and dispersion of ASDRs and CSDRs across the U.S.

4. There will be significant amounts of clustering and dispersion of ASDRs and CSDRs between counties.

5. There will be a significant amount of variation in mortality between the counties of the U.S. that is accounted for by spatial variation.

In this chapter of my dissertation I discussed the data and methods to be used in my analyses, and I also outlined the corresponding hypotheses that I will be testing. The next chapter of this dissertation will provide a more in

depth look at the data and whether or not they are

appropriate for the analyses proposed.

CHAPTER IV

DESCRIPTION OF THE DATA

A major question I will be addressing in my
dissertation is the following: what are the main factors
impacting mortality and mortality rates in the year 2000?
To address this question, I will use four separate data
sets. They are in two main sections; individual level data
and county level data. These data represent the
information that will be informing two very distinct
portions of the above question, having to do with persons
and having to do with counties. Before endeavoring to
answer the different sides of the above question with my
statistical analyses, I examine in this chapter the
descriptive information about each data set. The
descriptive data are shown in Table 2.

Table 2: Descriptive Statistics: 2,566 Individuals

Variable	Mean	Std. Dev.	Min	Max
Age at Death (Months)	700.92	230.37	309	1219
Female	.53	.50	0	1
White	.47	.50	0	1
Mexican-American	.24	.43	0	1
Black	.28	.43	0	1
Hispanic	.01	.14	0	1
Married	.63	.48	0	1
Education	10.67	4.17	0	17
Poverty	2.49	1.81	0	9.98
Self-health	2.07	1.90	0	5
Tobacco	.30	.46	0	1
Medicare	.33	.47	0	1
Medicaid	.10	.31	0	1
Private	.67	.47	0	1

As indicated in Table 2 there are 2,566 respondents in the individual level data set. The average age at death is 700.92 months or about 58.41 years of age. The youngest respondent to die was age 25 while the oldest was 101 years old. The sample is 53 percent female. The most common self-reported racial category is White (47 percent) followed by Black (28 percent) and Mexican American (24 percent). The majority of the sample was married at the time of the interview (63 percent). The mean number of years of education achieved by the sample was 10.67 years. Respondents were, on average, living at 2.49 times above the poverty threshold. On a six point scale (ranging from

zero to five) the mean score for self reported health was
2.07 indicating that, on average, people were more inclined
to report their health being closer to poor than to
excellent. At the time of the interview 30 percent of the
sample was consistently using a tobacco product. The
majority of the sample had private medical insurance (67
percent), while 33 percent had Medicare and 10 percent had
Medicaid (recall that these categories are not mutually
exclusive of each other).

Table 3: Descriptive Statistics: 2,534 Counties

Variable	Mean	Std. Dev.	Min	Max
65-74 ASDR	1.91	0.57	0.28	4.89
75-84 ASDR	2.98	0.87	0.61	8.04
85+ ASDR	2.97	1.16	0.55	10.75
Neoplasm DR	1.18	0.20	0.49	2.75
Stroke DR	4.86	1.59	1.11	15.44
Heart Disease DR	3.13	1.00	0.49	7.77
Medicare Expenditure per capita	3,016.14	874.95	151.81	12291.33
Median Age of County	37.05	3.72	20.70	54.30
Population of County	108,842	322,127	2,308	9,519,338

Table 3 presents descriptive statistics for the
various county level variables. There are 2,534 counties
in this data set which is more than 600 counties less than
total number of counties in the U.S. This discrepancy is

present for two reasons; the first is that my aggregate analysis is restricted to the continental U.S. The second reason is an artifact of the population size of the county along with the number of deaths in 2000. The Center for Disease Control (CDC) does not release age-specific or cause-specific death rates when the occurrence of the event is deemed as too rare in the county (because of the counties small population) and that the reporting of the rate would be a violation of anonymity. Accordingly, my aggregate sample contains 2,534 counties.

The mean Age-Specific Death Rate (ASRD) is the lowest for the 65-74 age group (1.91), followed by the 85 and older age group (2.97), with the highest ASDR in the 75-84 group (2.98). The highest 65 to 74 ASDR is found in Norton County, VA (4.89), the highest 75 to 84 ASDR is in Galax County, VA (8.04) and the highest 85+ ASDR is in Dewey County, OK (10.75). The lowest ASDRs range from 0.28 (for the 65-74 age group) in Douglas County, CO to 1.16 (for the 85 and older group) in Liberty County, GA.

The mean Cause-Specific Death Rate (CSDR) is highest for the stroke group (4.86), with a lower CSDR for the heart disease group (3.13), and the lowest for the neoplasm group (1.18). It is striking to me that there were no zero CSDRs in any counties in 2000, giving evidence of the

pervasiveness of the three leading causes of death. The

stroke group had a high CSDR of 15.44 in Somervell County,

TX and a low of 1.11 in Richmond County, NY. The heart

disease group had a low of 0.49 in Douglas County, CO and a

high of 7.77 in Emporia County, GA. The neoplasm group

ranged from a low value of 0.49 in La Paz County, AZ to a

high of 2.75 in Madison County, MI.

The independent variables I will use to analyze the

county-level mortality rates also show interesting

variation. The mean per capita Medicare expenditure across

all the counties is $3,016.14. The smallest Medicare

expenditure for the year 2000 was in Mathews County, VA

(zero dollars), with $12,291.33 being the largest

expenditure in Swisher County, TX. The range between the

minimum and maximum Medicare expenditure is extreme and can

be attributed (somewhat) to the small populations of

Swisher and Mathews County, although the maximum value may

be considered inflated because of the high dollar

expenditure by the Medicaid program for the treatment of

renal disease in Swisher County. The mean age of the

counties in the year 2000 is 37.05, with Madison County, ID

as the youngest county (20.7), and Charlotte County, FL

being the oldest county (54.3).

The standard deviations for the individual-level and county-level variables shown in Tables 1 and 2 are all of a reasonable value and are suitable for my regression modeling. I also wish to ascertain whether the independent variables I will use in my models do not have problematically high relationships with each other. One way I address this issue is by examining the pairs of zero-order correlations. Table 4 shows the correlations for the micro-level independent variables.

Table 4 shows that most of the independent variables are only slightly correlated with one another (see Table 4). Education and poverty have a strong relationship as does White and Mexican-American and White and Black, but even these are not so high that the variables cannot be used in the same regression equation.

Table 4: Correlation Matrix: Individual Level Independent Variables

	White	Mexican-American	Black	Hispanic	Female	Married	Education	Poverty	Self-Health	Tobacco	Medicare	Medicaid
Mexican-American	-0.54											
Black	-0.54	-0.32										
Hispanic	-0.14	-0.08	-0.09									
Female	0.04	-0.04	0.01	0.00								
Married	0.05	0.12	-0.17	-0.01	-0.18							
Education	0.27	-0.36	0.04	0.00	0.05	0.03						
Poverty	0.33	-0.22	-0.14	-0.03	-0.03	0.17	0.50					
Self Health	0.12	-0.13	-0.01	0.02	-0.01	0.03	0.23	0.21				
Tobacco	-0.03	-0.08	0.12	-0.04	-0.16	-0.06	-0.07	-0.13	-0.07			
Medicare	0.26	-0.15	-0.13	-0.06	-0.03	-0.13	-0.16	-0.06	-0.10	-0.10		
Medicaid	-0.16	0.02	0.14	0.04	0.09	-0.24	-0.17	-0.28	-0.07	0.04	0.10	
Private	0.29	-0.27	-0.04	-0.06	0.02	0.14	0.39	0.47	0.14	-0.09	-0.03	-0.35

Second, I also examined the tolerances of each

independent variable. My key substantive variables all have

respectable tolerance values of greater than .6.

I next look at the zero-order correlations of my three

county-level independent variables. They are shown in Table

5.

**Table 5: Correlation Matrix, County Level Independent
Variables**

	Median Age	Population Size
Medicare Expenditure per capita	-0.09	0.15
Median Age		-0.16

The three county level independent variables all have

low correlations with each other; their use in the same

regression equations will not be problematic.

The next chapter of this dissertation contains the

individual level analysis. I will present results from an

estimation of ten OLS models in order to test my individual

level hypothesis.

CHAPTER V

MICRO-LEVEL ANALYSES OF FACTORS INFLUENCING AGE AT DEATH

In this chapter of my dissertation, I provide the results of my hypothesis tests with respect to the prediction of age at death. Many different models are estimated for different groups of subjects. Their results provide evidence with regard to whether or not my various hypotheses were supported.

Descriptive Results

Before presenting the results of my regression analyses, I first present descriptive results of my dependent variable, age at death, cross-tabulated by race and ethnicity and gender.

Table 6 presents data on mean age at death for the various races, ethnicities and for males and females. I have provided the weighted mean values of age at death for the entire racial and ethnic group as well as for the female specific and male specific mean ages. In my sample, White females outlive White males by thirty years. Black females outlive males by sixteen years; the same is true for the Hispanic group. However, Mexican-American women and men have the smallest longevity gap of this sample with women enjoying a four year advantage. For the overall

sample, women outlive men by twenty years. Though it is
expected that women will live longer than men, this sample
of decedents far exceeds the female five year life
expectancy advantage commonly reported in the literature
from data on period life tables.

Table 6: Mean Age at Death by Race-Ethnicity and Gender: 2,566 Individuals

	Males	Females	Total
White	36.25	66	43.67
Black	44.83	60.42	53.00
Mexican-American	40.42	44.58	40.75
Hispanic	54.17	70.74	60.92
Males			37.00
Females			64.83
Sample			58.41

This sample also appears to be somewhat unique along
racial lines. As I addressed in the literature review
portion of this dissertation, life table data on life
expectancy show that minority racial groups do not live as
long as the White majority. There is evidence of Hispanics
and Mexican-Americans living as long as Whites, and this is
known as the Hispanic paradox. But there is little evidence
of the minority groups having an advantage over Whites. An
exception to this is the so-called the Black White
mortality cross-over that occurs among the very old,

usually after age 80, when minority age-specific death rates become less than those of the majority. However, the cross-over would not be expected to be manifested in my sample of decedents because it includes people 25 and older; in fact, the mean age at death of my sample is under sixty years.

However, when I examine the racial and gender breakdown of average age at death for my sample of decedents, certain anomalies of these data become apparent. White males have the youngest mean age of death in the sample at just over 36 years; this does not reflect the national death behavior in the year 2000 when the average life expectancy for White males was 75 (Kung et al. 2005). Black, Mexican-American and Hispanic males' average age at death in my sample data is low compared to the national average in 2000. We must remember that my data are restricted to persons of age 25 and over, and the life expectancy data used above for comparisons are based on life table age-specific death rate data for the entire population.

Nevertheless, given the somewhat anomalous nature of my sample data, it will be interesting to examine the impacts the various independent variables, including the race/ethnic and gender variables, on age at death.

I turn next to the multiple regression analyses. These results will be presented in two sections. In the first section I present the results from multiple regressions predicting age at death that were estimated for the entire sample. In the second section I discuss the results from regression equations that were estimated for each of the race-ethnic-specific sub-groups.

Multiple Regression Results: Equations for the Full Sample

Model 1 and all the models estimated here have as the dependent variable, age at death; the independent variables in Model 1 are the ascribed independent variables of female, Black, Mexican-American and Hispanic, with White used as the reference variable. Race is positively associated with age at death (see table 7). Mexican-Americans, on average, have a 19.75 months longevity advantage over Whites, while Blacks have moiré than a 21.4 month advantage. Hispanics, i.e., non-Mexican Hispanics, have a huge advantage, but the number of cases in my sample is very small. Model 1 also shows the very significant female advantage over males of over 330 months.

The second model is identical to model 1 with the exception of the inclusion of the insurance variables of Medicare, Medicaid and private insurance. Model 2 shows that having insurance is significantly associated with age

of death (see table 7). Having Medicaid adds, on average

152 months of life, Medicare adds 329 months and having

private insurance adds 52 months. Adding insurance to the

models serves to reduce considerably the longevity

advantage of females by almost 13 years. Adding insurance

variables to the model results in the Black advantage over

Whites to become not statistically significant. But the

Hispanic advantage now becomes significant, and the Mexican

group retains its advantage.

Table 7: Individual Level Regression Results Predicting Age at Death, Models 1 and 2: 2,566 Individuals

	Model 1			Model 2		
	Coef.	Std. Er.	P>|t|	Coef	Std. Error	P>|t|
Mexican-American	19.75	6.48	0.00	48.71	5.19	0.00
Black	21.42	9.10	0.02	10.33	9.01	0.25
Hispanic	208.16	2.69	0.00	257.17	5.85	0.00
Female	331.80	7.02	0.00	176.92	14.09	0.00
Medicaid				152.47	15.03	0.00
Medicare				329.47	15.05	0.00
Private				51.66	6.44	0.00
Constant	441.84	2.69	0.00	392.83	5.85	0.00
R-squared	0.59			0.74		

Model 3 adds the achieved variables of marriage,

education and poverty to the demographic variables used in

Model 1 (see table 8). Model 3 shows that the Mexican-

American, Black and Hispanic groups retain their longevity

advantage over Whites. Females continue to have an

advantage over males. Surprisingly marriage is a
significant but negative predictor of age at death with a
longevity disadvantage of just under four years for married
versus not married respondents. Education is also a
significant negative predictor of age at death with an
average 3.6 month disadvantage for every extra year of
education earned. Poverty is a significant indicator of
mortality. Every one point increase in the rate a person
is above the poverty threshold increases their life by 20
months, on average.

   Model 4 builds on the demographic and achieved
variables in model three by adding insurance variables to
the model. Race and ethnicity continue to be significant
positive predictors of age at death (see table 8). The
advantage of the Mexican-American and Hispanic groups
increases with the addition of insurance variables to the
model, while the Black advantage decreases. Marriage and
education continue to have a significant negative
association with age at death. While the longevity
disadvantage associated with being married decreases by
around 26 months, the longevity advantage of earning an
extra year of education increases by almost 6 months.
Having insurance would serve to increase a person's

longevity by 226 months for Medicaid, 313 months for

Medicare and 123 months for private insurance.

Table 8: Individual Level Regression Results Predicting Age at Death, Models 3 and 4: 2,566 Individuals

	Model 3			Model 4		
	Coef.	Std. Er.	P>|t|	Coef	Std. Error	P>|t|
Mexican-American	51.20	8.68	0.00	59.89	6.74	0.00
Black	48.82	8.62	0.00	41.13	8.91	0.00
Hispanic	279.29	6.68	0.00	374.03	17.25	0.00
Female	339.16	7.66	0.00	182.84	14.99	0.00
Married	-45.03	7.56	0.00	-19.20	5.40	0.00
Education	-3.61	0.51	0.00	-9.38	1.01	0.00
Poverty	20.29	1.50	0.00	8.51	1.18	0.00
Medicaid				226.40	18.44	0.00
Medicare				313.31	16.42	0.00
Private				123.50	14.70	0.00
Constant	445.21	6.30	0.00	425.56	4.03	0.00
R-squared	0.62			0.77		

Model 5 includes the ascribed variables of female,

Mexican-American, Black and Hispanic, along with a measure

of perceived health (see Table 9). The variable self-

health is a progressive scale with five being excellent

health and one being poor health. Race and ethnicity

continue to be significant positive predictors of months of

life (see Table 9). Females continue to have a significant

longevity advantage over males. For every point increase

on the one (poor health) to five (excellent health) scale a

person's longevity increases, on average, by 37 months.

Model 6 contains the variables in Model 5, but adds

the health insurance variables of Medicare, Medicaid and

private insurance (see table 9). Race and ethnicity

continue to be positive significant predictors of

longevity. The advantage associated with being female

decreases by about 12 years. Self reported health remains a

significant predictor, though its impact is increased by

the presence of insurance. The longevity advantage of

self-reported health increases by 5.5 months when the

insurance variables are added to the model. Insurance

continues to be a significant predictor of age at death.

Medicaid provides a 105 month advantage, Medicare a 331

month advantage, and private insurance a 17 month

advantage.

Table 9: Individual Level Regression Results Predicting Age at Death, Models 5 and 6: 2,566 Individuals

	Model 5			Model 6		
	Coef.	Std. Er.	P>|t|	Coef	Std. Error	P>|t|
Mexican-American	57.43	9.31	0.00	78.29	6.53	0.00
Black	59.54	11.05	0.00	42.32	8.83	0.00
Hispanic	233.64	5.19	0.00	256.66	5.84	0.00
Female	329.48	7.00	0.00	183.79	15.90	0.00
Self Health	37.15	5.84	0.00	42.67	5.42	0.00
Medicaid				105.40	19.36	0.00
Medicare				330.78	16.08	0.00
Private				16.74	7.62	0.03
Constant	304.91	22.13	0.00	265.33	17.35	0.00
R-squared	0.60			0.76		

Model 7 contains the ascribed variables, female, Mexican-American, Black, Hispanic, as well as whether or not the respondent used any form of tobacco in the month preceding the interview (See Table 10).   Tobacco use is shown to be significantly and negatively related to age at death and abbreviates life for tobacco users by just under six years.

Model 8 contains the variables of Model 7, but adds the insurance variables. (see Table 10). The insurance variables result in an increase of the Mexican-American and Hispanic advantage and a decrease in the Black and female advantage. The addition of insurance variables also diminishes considerably the significant longevity disadvantage associated with being a tobacco user. And Medicare, Medicaid and private insurance continue to show a significant relationship with lifespan.

Table 10:   Individual Level Regression Results Predicting Age at Death, Models 7 and 8: 2,566 Individuals

	Model 7			Model 8		
	Coef.	Std. Er.	P>|t|	Coef	Std. Error	P>|t|
Mexican-American	16.85	7.03	0.02	47.74	5.30	0.00
Black	19.92	8.98	0.03	16.12	8.89	0.07
Hispanic	198.03	3.03	0.00	250.62	5.30	0.00
Female	356.92	8.72	0.00	178.55	14.12	0.00
Tobacco	-57.36	7.63	0.00	-32.63	6.87	0.00
Medicaid				176.20	16.82	0.00
Medicare				328.02	15.11	0.00
Private				50.13	6.22	0.00
Constant	451.97	3.03	0.00	399.38	5.30	0.00
R-squared	0.60			0.76		

Model 9 is a compilation of all the previous models, and I refer to it as the "complete" model. It includes the independent variables of female, Mexican-American, Black, Hispanic, married, education, poverty, self-health and tobacco. (See Table 11). The race-ethnic variables continue to show a lifespan advantage, as does the female variable. The poverty variable performs as expected: for every one point above the poverty threshold, a person can expect to live, on average, 19 months more. Self-health has a similar relationship with longevity in this model as it had in Model 5 and serves to increase age at death by over four years for every increase in perceived health (from one to five).   Tobacco use in the previous month is not a significant predictor of age at death in this model.

Model 10 is the final model and includes all of the variables from model nine along with the health insurance variables. Adding the insurance variables has a similar outcome as the addition of these variables had in Model 2, 4, 6 and 8. The Hispanic and Mexican-American advantage increases, while the Black and female advantage decreases. The poverty variable continues to have the same kind of effect. Insurance continues to have a significant positive impact on age at death. Medicare provides a 319 month advantage, Medicaid a 182 month advantage, and private insurance an 84 month advantage.

Table 11: Individual Level Regression Results Predicting Age at Death, Models 9 and 10: 2,566 Individuals

	Model 9			Model 10		
	Coef.	Std. Er.	P>|t|	Coef	Std. Error	P>|t|
Mexican-American	82.58	11.63	0.00	87.52	8.75	0.00
Black	96.30	11.69	0.00	76.79	9.42	0.00
Hispanic	312.27	15.59	0.00	371.34	23.92	0.00
Female	349.85	14.25	0.00	191.57	16.91	0.00
Married	-19.03	15.72	0.23	-0.53	13.18	0.97
Education	-7.06	0.69	0.00	-10.29	1.07	0.00
Poverty	19.06	3.92	0.00	9.68	3.11	0.00
Self Health	51.90	8.42	0.00	47.68	6.67	0.00
Tobacco	-4.68	19.26	0.81	5.11	17.85	0.78
Medicaid				182.46	21.90	0.00
Medicare				318.91	17.66	0.00
Private				83.53	16.19	0.00
Constant	283.83	26.96	0.00	278.71	19.67	0.00
R-squared	0.64			0.79		

The above analyses now enable me to address whether or not my main hypotheses were supported in the individual level regression models using data for the complete sample.

There was a consistent positive advantage in longevity for Blacks, Mexican-Americans and non-Mexican Hispanics, compared to Whites. Sex was a significant predictor with females having a consistent longevity advantage over males. Surprisingly, marriage and education were significantly, though negatively, related with longevity, although marriage was not always a significant predictor. Tobacco use had significant negative effect on longevity, although this variable lost its significance in the complete model. The three insurance variables consistently were related with additional months of life.

The addition of the insurance variables consistently served to decrease the magnitude of the effects of the independent variables of female, Black, marriage, and poverty, and increased the effects of the independent variables of Mexican-American, Hispanic, and education. Adding the insurance variables resulted in the poverty variable having inconsistent impacts on age at death. My individual level hypotheses received mixed support by this analysis. But the more important result is my evidence that the magnitudes of the effects of various demographic

and behavioral variables are often attenuated when the Medicaid, Medicare and private insurance variables are added to the equations.

But given that in all ten models there were large and usually significant coefficients for the race-ethnic variables, this would indicate that the dynamics of longevity are likely not the same in each group. Thus in the next section of this chapter, I re-estimate many of the above models, but do so for each of the three main race-ethnic groups.

Multiple Regression Results: Race-specific Equations

I begin this section by first estimating race-ethnic-specific equations for Whites, Blacks and Mexican Americans using only one independent variable, sex. The results in

Table 12 shows that the female variable is a significant predictor of age at death for Whites and Blacks, but not for Mexican Americans. Adding the insurance variables to these race-specific models (Table 13) serves to decrease the size of the female advantage.

Table 12:   Individual Level Regression Results Predicting Age at Death: Model 1, Race-Ethnic Subgroups

	White and Other			Mexican-American			Black		
	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|
Female	356.67	8.11	0.00	50.80	23.16	0.06	186.90	14.31	0.00
Constant	435.71	2.67	0.00	485.01	18.91	0.00	538.90	4.09	0.00
R-squared	0.64			0.02			0.22		
Observations	1,212			621			733		

Table 13:   Individual Level Regression Results Predicting Age at Death: Model 2, Race-Ethnic Subgroups

	White and Other			Mexican-American			Black		
	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|
Female	143.69	17.32	0.00	9.75	23.15	0.67	182.07	26.43	0.00
Medicaid	139.71	18.26	0.00	51.69	46.38	0.27	-35.09	37.91	0.36
Medicare	466.02	16.64	0.00	413.88	37.16	0.00	52.77	26.68	0.05
Private	-16.36	6.12	0.01	36.66	5.29	0.00	265.35	9.62	0.00
Constant	440.71	5.44	0.00	462.14	3.05	0.00	357.42	8.31	0.00
R-squared	0.90			0.59			0.46		
Observations	1,212			621			733		

The effect of marriage on longevity is not consistent for all races (Table 14). Marriage is not a significant predictor among Whites, it is a positive significant predictor among Mexican-Americans, and is a negative and significant predictor among blacks. Education is only significant in the White model and has a negative association as was seen in the earlier models. Being above the poverty line is a significant positive predictor for all three races although the magnitude of the benefit varies widely.

Table 15 adds the insurance variables to those in the previous model. Adding insurance results in marriage becoming a significant positive predictor for Whites, and

it increases the advantage for Mexican-Americans and
decreases the disadvantage for blacks. The insurance
variables also results in education no longer being
significantly associated with age at death.

Table 14: Individual Level Regression Results Predicting Age at Death: Model 3, Race-Ethnic Subgroups

	White and Other			Mexican-American			Black		
	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|
Female	378.46	9.72	0.00	37.93	29.56	0.20	227.37	14.73	0.00
Married	-6.35	9.29	0.49	48.52	9.36	0.00	-208.49	14.83	0.00
Education	-4.60	0.48	0.00	-3.72	3.65	0.31	-2.88	7.31	0.69
Poverty	16.44	1.58	0.00	30.19	11.12	0.01	131.59	11.12	0.00
Constant	439.87	5.88	0.00	434.79	32.94	0.00	445.71	68.43	0.00
R-squared	0.67			0.13			0.67		
Observations	1,212			621			733		

Table 15: Individual Level Regression Results Predicting Age at Death: Model 4, Race-Ethnic Subgroups

	White and Other			Mexican-American			Black		
	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|
Female	151.70	18.19	0.00	-1.11	24.14	0.96	69.00	35.97	0.06
Married	44.29	5.26	0.00	62.84	11.01	0.00	-130.94	43.25	0.00
Education	-1.25	1.60	0.44	1.78	4.09	0.66	0.51	6.41	0.94
Poverty	1.05	1.06	0.32	21.27	16.61	0.20	161.39	28.73	0.00
Medicaid	150.79	27.62	0.00	91.73	52.48	0.08	88.50	44.55	0.05
Medicare	479.14	18.15	0.00	395.25	45.92	0.00	216.81	40.23	0.00
Private	-30.49	25.14	0.23	-4.72	19.10	0.81	98.02	44.79	0.03
Constant	436.51	3.57	0.00	387.95	27.04	0.00	262.00	70.90	0.00
R-squared	0.91			0.52			0.74		
Observations	1,212			621			733		

Increasingly positive views of health are positively
associated with longevity for all three racial groups
though the advantage is greatest for blacks followed by
Mexican-Americans and then Whites (Table 16). Adding the

insurance variables to this model results in numerous and

various changes (Table 17).

Table 16: Individual Level Regression Results Predicting Age at Death: Model 5, Race-Ethnic Subgroups

	White and Other			Mexican-American			Black		
	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|
Female	350.89	8.71	0.00	38.18	28.70	0.18	320.28	69.13	0.00
Self Health	18.30	8.31	0.03	37.09	6.21	0.00	72.75	30.66	0.02
Constant	369.43	30.51	0.00	386.77	15.64	0.00	273.41	112.23	0.02
R-squared	0.64			0.12			0.29		
Observations	1,212			621			733		

Table 17: Individual Level Regression Results Predicting Age at Death: Model 6, Race-Ethnic Subgroups

	White and Other			Mexican-American			Black		
	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|
Female	140.55	17.98	0.00	-1.41	23.16	0.95	112.26	63.34	0.08
Self Health	18.17	6.25	0.00	42.19	9.58	0.00	-46.85	29.33	0.11
Medicaid	125.88	19.47	0.00	37.55	48.14	0.44	-15.75	44.60	0.72
Medicare	464.09	16.81	0.00	411.59	39.72	0.00	29.96	17.83	0.09
Private	-29.47	7.40	0.00	-13.60	13.86	0.33	301.19	23.67	0.00
Constant	385.94	19.75	0.00	373.72	18.91	0.00	503.99	92.41	0.00
R-squared	0.90			0.52			0.61		
Observations	1,212			621			733		

The regression results in Tables 18 and 19 illustrate

the impact of how the use of tobacco products impacts age

at death. Whites who had used tobacco in the past month

are at a four years longevity disadvantage, Blacks at a 20

year disadvantage and, surprisingly, Mexican-American

tobacco users live, on average, ten years longer than

Mexican-Americans who were not tobacco users. The addition

of the insurance variables has an inconsistent effect on

the effect of tobacco use.

Table 18: Individual Level Regression Results Predicting Age at Death: Model 7, Race-Ethnic Subgroups

	White and Other			Mexican-American			Black		
	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|
Female	382.69	10.50	0.00	58.51	25.68	0.02	199.37	14.28	0.00
Tobacco	-52.05	9.21	0.00	111.04	9.40	0.00	-240.75	13.35	0.00
Constant	444.11	2.94	0.00	4ob66.30	3.18	0.00	623.69	6.89	0.00
R-squared	0.65			0.21			0.56		
Observations	1,212			621			733		

Table 19: Individual Level Regression Results Predicting Age at Death: Model 8, Race-Ethnic Subgroups

	White and Other			Mexican-American			Black		
	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|
Female	139.12	17.55	0.00	19.18	21.76	0.38	174.71	26.33	0.00
Tobacco	31.06	7.54	0.00	98.53	8.69	0.00	-115.13	29.25	0.00
Medicaid	115.45	19.90	0.00	41.14	45.68	0.37	-13.45	42.48	0.75
Medicare	473.26	17.16	0.00	404.46	35.11	0.00	59.45	26.69	0.03
Private	-20.07	6.57	0.00	11.64	5.95	0.05	166.92	29.56	0.00
Constant	438.72	5.15	0.00	457.29	3.01	0.00	464.64	29.86	0.00
R-squared	0.91			0.60			0.61		
Observations	1,212			621			733		

Table 20 presents the results of the complete model.

Marriage and tobacco lose their impact on age at death for

Whites in the complete model, income and self health are no

longer significant predictors for Mexican-Americans, and

education and gender lose their impact for the black group.

The other independent variables behave as they did in the

earlier models. Adding insurance to the complete model

(Table 21) results in the attenuation of the female

advantage in the White and Mexican-American groups,

marriage becomes more beneficial, the impact of education

is lessened, and higher earnings give less access to

additional months of life among Whites and Mexican-

Americans. With insurance in the model a positive view of

health becomes more beneficial.   Tobacco is still

volatile, with insurance in the model, with no consistent

pattern among the races.

**Table 20:   Individual Level Regression Results Predicting
Age at Death: Model 9, Race-Ethnic Subgroups**

	White and Other			Mexican-American			Black		
	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|
Female	371.11	19.77	0.00	60.75	29.31	0.04	75.46	45.38	0.10
Married	2.87	20.15	0.89	26.09	13.30	0.05	-191.34	39.39	0.00
Education	-7.48	0.84	0.00	-7.92	3.93	0.04	-4.75	5.72	0.41
Poverty	17.29	5.32	0.00	11.84	14.63	0.42	123.19	17.25	0.00
Self Health	43.74	11.22	0.00	-2.50	12.19	0.84	-85.48	23.76	0.00
Tobacco	3.00	26.89	0.91	116.98	25.58	0.00	-120.74	32.06	0.00
Constant	306.15	35.65	0.00	498.07	41.27	0.00	75.46	45.38	0.00
R-squared	0.69			0.27			0.73		
Observations	1,212			621			733		

**Table 21:   Individual Level Regression Results Predicting
Age at Death: Model 10, Race-Ethnic Subgroups**

	White and Other			Mexican-American			Black		
	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|	Coef.	Std. Er.	P>|t|
Female	138.62	19.27	0.00	18.96	22.86	0.41	-2.27	45.91	0.96
Married	29.17	14.08	0.04	43.69	10.01	0.00	-129.87	47.78	0.01
Education	-3.06	1.70	0.07	-3.17	4.53	0.48	-1.10	5.57	0.84
Poverty	8.90	3.40	0.01	7.47	12.53	0.55	152.87	24.72	0.00
Self Health	28.94	7.58	0.00	8.76	13.73	0.52	-52.35	24.74	0.04
Tobacco	44.84	19.97	0.03	92.21	14.47	0.00	-125.57	33.16	0.00
Medicaid	118.41	29.84	0.00	49.76	51.48	0.33	120.51	52.51	0.02
Medicare	473.49	18.12	0.00	385.34	38.62	0.00	181.72	35.47	0.00
Private	-44.13	27.15	0.10	-20.62	23.69	0.39	33.79	45.36	0.46
Constant	340.88	22.83	0.00	430.57	29.19	0.00	571.94	112.55	0.00
R-squared	0.91			0.62			0.77		
Observations	1,212			621			733		

By breaking out my regression analysis by race and ethnicity, I have shown a different view of mortality than was possible when I controlled for race. Marriage does not have the same impact for all three races. The magnitude of the advantage provided by higher earnings is vastly different across races, and tobacco use has a consistently negative impact only on the black sample. In the search for equality in mortality it is important to remember that equality is a goal and that special efforts must be made in identifying the discrepancy between races as is evidenced by this analysis.

The behavior of race throughout the analyses presented in this chapter has not been consistent with the prevailing literature. Given that the non-White longevity advantage is so different than what would be expected in the population at large it is worth considering why these data behaved this way. Consider the cross-sectional nature of these data: the interview subjects were matched to the death records four years after the final interview was conducted. Many of the participants were young and had not died four years after the interview. The mean age of the White male decedents is just under 34 years leading me to the tentative conclusion that the decedents were atypical of the population at large and that these data likely

captured non-traditional deaths. Were these same data to be matched to death records 50 years from now I am fairly confident that they would mimic national mortality behavior.

Because these data are so atypical of what the current literature indicates I attempted several different analyses schemes. First I attempted hazard modeling. The data behaved in even more unpredictable ways when estimated with hazard models and that scheme was abandoned. Next I tried restricting the sample to the 65 and over decedents. The over 65 group resulted in a small sample size which allowed a great deal of statistical noise into the analyses—the sample was too small to use multivariate analyses. It was at this point I utilized the 25 and over decedents for the analyses and broke those analyses out by racial group to further deal with the unusual findings.

In retrospect, were I to be on the front side of this analyses I would have included the living in the sample. With the living in the sample I could estimate a logistic model using age as a control and look at what variables were significant predictors of dying. After determining what increased a participants likelihood of dying I would have dropped the living from the sample and estimated the race-specific models (as I did at the end of the Chapter V

analyses) to further analyze mortality differential between racial groups and determine if insurance translated into mortality gains.

The next chapter of my dissertation contains the county level analyses. I will utilize county level data to test my county level mortality hypotheses.

CHAPTER VI

MACRO-LEVEL ANALYSES OF FACTORS INFLUENCING COUNTY-LEVEL

DEATH RATES

In this chapter of my dissertation, I turn to an aggregate-level analysis of mortality. Here I estimate a number of models examining mortality among the counties of the U.S. These tests will provide evidence enabling me to evaluate once again my hypotheses that focus on the impacts of insurance on mortality. But rather than examining them at the micro-level, as I did in the last chapter, here is examine them among counties. Numerous spatial and traditional demographic techniques are used here to estimate these models among U.S. counties.

Context of Mortality

In my review of the current literature earlier in this dissertation I noted that the context an individual lives in will affect his/her mortality; quite literally, this context can be thought of as the individual's geographic location. One way to examine this context is through hierarchical modeling in which the micro-level outcome of death is allowed to be influenced by both micro-level and macro-level factors. But this was not an option with the data I used in the micro-level chapter of this dissertation. Given that I do not have access to the

individuals' specific geographic locations I must find
another way to study the relationship mortality has with
geographic context. One solution is to study the mortality
patterns of the context itself. Put another way, by
studying the death rates of counties of the U.S., I will be
able to form a more precise picture of American mortality.

Furthermore by separating the micro and macro level
analyses, I will be able to take advantage of the
opportunity to determine if the mortality of a place is
different than the mortality of an individual. Does a
place have its own mortality—is there something about a
county that has a unique impact on age at death that cannot
be addressed by looking at aggregated individual data? Of
course, the county and individual level results will not be
directly comparable, but each level-specific conclusion can
help inform the interpretation of the larger conclusions to
be drawn from this study.

Global Autocorrelation Results

Before conducting the county level regression
analyses, I must first determine whether U.S. county death
rates are related to each other. I will present these
results first.

When trying to discern if there is a relationship
between counties, it is first necessary to determine if

there is a relationship across counties. This may be accomplished by estimating a Moran's I coefficient for each of the six specific death rates used in this analysis. Models 11 through 16 are global spatial autocorrelation models testing for spatial relationships between the death rates across the continental U.S. Moran's I is used to detect departures from spatial randomness.

The Moran's I results indicate that there is a significant amount of spatial autocorrelation of Age-Specific Death Rates (ASDRs) and Cause-Specific Death Rates (CSRDs) across the counties of the U.S. that is present in each of three ASDR groups and the three CSDR groups (Table 22). The 65-74 ASDR's, the 75-84 ASDRs, and the 85+ ASDRs are all significantly clustered across U.S. counties; the significance levels are p<.01. The same relationship is present with the neoplasm, heart disease and stroke CSDRs. All of them are significant at the p<.01 level), indicating significant amounts of clustering across the counties of the continental U.S.

None of the magnitudes of the Moran's I coefficients, however, are particularly high. Usually, absolute values of 1.00 indicate strong levels of dispersion or clustering. Were the Moran's I analyses to be the final tests for spatial relationships, this might prove to be a problem for

this analysis. As it is, the Moran's analyses are only

meant to inform the presence of spatial relationships; the

magnitude of the coefficients are not a point of concern,

but rather, are evidence that some amount of variation in

the death rates can be attributed to spatial predictors.

Table 22: Model 11-16-Global Autocorrelation for Age-Specific and Disease-Specific Death Rates: 2,534 Counties

Model	Death rate	Moran's I	Z	P
11	65-74	0.14	45.46	.0000
12	75-84	0.12	36.79	.0000
13	85+	0.26	82.23	.0000
14	Neoplasm	0.10	36.45	.0000
15	Heart Disease	0.15	47.27	.0000
16	Stroke	0.12	36.85	.0000

County Specific Autocorrelation

Having determined that there is a significant amount

of relationship between death rates across the counties, I

now present a local measure of spatial autocorrelation to

determine the amount of relationship that the death rates

of individual counties share with the death rates of other

counties.

Table 23 shows the high and low values for the local

g-scores for the continental U.S. counties. A g-score is

the representation of the Getis-Ord statistic which

identifies spatial clusters based on weighted data points

that display values that are higher than expected. The

output for this test is a z-score that is often referred to as a g-score in the literature (ESRI 2009). The g-score interpretation is not very intuitive, although its meaning is quite easy to understand. The higher the score, the stronger the clustering relationship between counties; the lower the score, the stronger the dispersion is between counties; and values around zero indicate a lack of significant relationship between counties.

The age-specific death rate g-scores range from a low of -9.10 for the 75-84 ASDRs to a high of 14.17 for the 85+ ASDRs. The 65-74 ASDRs shows the greatest amount of dispersion in Jefferson County, CO, and the lowest amount in Mingo County, WV. The greatest amount of dispersion in the 75-84 ASDRs is found in Chaffe County, CO, and the largest amount of clustering is in McPherson County, KS. The 85+ ASDRs demonstrate the greatest amount of clustering in Smith County, KS and the least amount in Chaffe County, CO.

The cause-specific death rate g-scores display a similar range as the ASDRs, although the highs and lows cover a smaller range. The lowest value, -8.82, if for neoplasm CSDR, and the highest value, 7.99, is for heart disease CSDR. Gray County, KS has the largest amount of dispersion among the Neoplasm CSDRs, and James City County,

VA has the least. The greatest amount of clustering among

the heart disease CSDRs can be found in Carlisle County,

KY, and the greatest amount of dispersion can be found in

Chaffe County, CO. Kit Carson County, CO displays the

least amount of clustering among the stroke CSDRs and Vance

County, NC displays the most.

Table 23: Local Autocorrelation Descriptive Statistics: 2,534 Counties

Death Rate	Count	Mean	Std. Dev.	Minimum	Maximum
65-74	2,534	0.97	2.74	-7.01	7.67
75-84	2,534	0.42	2.45	-9.10	7.58
85+	2,534	-0.09	3.82	-7.24	14.17
Neoplasm	2,534	1.07	1.99	-8.82	5.20
Heart Disease	2,534	0.61	2.85	-8.67	7.99
Stroke	2,534	1.29	2.22	-7.75	6.00

Multiple Regression Results: Age-Specific Death Rates

Now that the local autocorrelation models have shown

that there is a significant amount of spatial correlation

between county death rates, I will now incorporate these

coefficients into OLS regressions models to determine

whether the spatial relationships between counties impact

the specific death rate of the county.

In all of the models presented in this section of the

chapter, the dependent variable is one of the six specific

death rates. The independent variables are mainly

consistent between the models. They are as follows: the median age of the county, the average Medicare expenditures of the county, the population of the county, and the g-scores for the specific death rate being modeled.

Model 17 is an OLS regression model with the 65-74 ASDR as the dependent variable (Table 24). All of the independent variables are significant predictors of county-level mortality as measured with the 65-74 ASDR. For every increase in the median age of the county the county's 65-74 ASDR increases by 0.07. The Medicare and population variables have significant impacts on the 65-74 ASDR although their coefficients are small (past the fourth decimal place). Medicare has a positive effect on the ASDR, while population has a negative effect. For every point increase in the association between the county's 65-74 ASDR with other counties the county's ASDR increases by 0.08. This abbreviated cache of independent variables accounts for 40% of the county level variation in 65-74 ASDR.

I have also presented the fully standardized betas to compare which predictors are having the greatest relative impacts on the death rate. For every one standard deviation increase in the Getis local autocorrelation score, there is an average of .38 standard deviation

increase in the 65-74 ASDR. Of the variables presented in

Model 17, the spatial relationship between counties has the

second largest impact on the 65-74 ASDR. The magnitude of

the spatial variable's impact on the ASDR indicates that a

county's relationship with other counties is a major

consideration in the study of aggregate mortality.

Table 24: Model 17 County Level Regression Predicting 65-74 ASDR: 2,534 Counties

	Coef.	Std. Error	P	Standardized Coef.
Median Age	0.07	0.00	0.00	0.47
Medicare Expenditure	0.00	0.00	0.00	0.06
Population	0.00	0.00	0.00	-0.08
G I Z-score	0.08	0.00	0.00	0.38
Constant	-0.94	0.09	0.00	
R-squared	0.44			

The dependent variable for Model 18 is the county

specific 75-84 ASDR. Unlike the 65-74 ASDR model, Medicare

expenditures in this model is not a significant predictor

of county death rate; however, the other independent

variables behave similarly (Table 25). An increase in

median age served to increase a county's 75-84 ASDR by

0.13, and an increase in the g-score increases the ASDR by

0.08. Again we see that the amount of spatial

autocorrelation present between the counties' ASDRs (75-84)

is a positive predictor of the ASDR.

The relative magnitude of the spatial variable among the 75-84 ASDR predictors is not as large it was for the younger group. Median age has the largest impact of all the variables in the model, accounting for an average .57 standard deviation change in the ASDR as median age increases.

Table 25: Model 18 County Level Regression Predicting 75-84 ASDR: 2,534 Counties

	Coef.	Std. Error	P	Standardized Coef.
Median Age	0.13	0.00	0.00	0.57
Medicare Expenditure	0.00	0.00	0.66	0.006
Population	0.00	0.00	0.00	-0.07
G I Z-score	0.08	0.01	0.00	0.23
Constant	-1.97	0.14	0.00	
R-squared	0.48			

Table 26 shows the OLS regression results with 85+ ASDR as the dependent variable. Again, all of the independent variables, save Medicare expenditures, are significant predictors of 85+ ASDR. A one unit increase in the median age of the county results in a 0.13 increase in the 85+ ASDR. And there is a similar positive significant relationship with the spatial autocorrelation g-index and the 85+ ASDRs, although the coefficient is larger at 0.15.

The standardized coefficients for the 85+ ASDR model differ somewhat from those in the earlier models. In the

mortality rates for this oldest age group, the spatial g-index relationship with the death rates has the greatest impact, followed by the median age of the county. This relationship points to the increasing importance of the relationship between counties and the mortality of the older age groups.

Table 26: Model 19 County Level Regression Predicting 85+ ASDR: 2,534 Counties

	Coef.	Std. Error	P	Standardized Coef.
Median Age	0.13	0.00	0.00	0.43
Medicare Expenditure	0.00	0.00	0.38	-0.01
Population	0.00	0.00	0.00	-0.06
G I Z-score	0.15	0.00	0.00	0.48
Constant	-1.92	0.18	0.00	
R-squared	0.53			

Models 17 through 19 have shown a rather consistent pattern of predictor effects across the three ASDRs under investigation. Median age of the county was a consistently positive predictor of the ASDR, and had the largest relative impact of all the predictors in the two younger ASDRs. Medicare expenditures was only a significant influence in the 65-74 ASDR, giving some evidence that expenditures do not impact death rates for the older groups. The spatial relationship between county ASDRs as reflected in the g-index was a significant predictor of

county ASDR for all age groups. When I examined standardized effects, this variable was seen to be one of the strongest effects on the ASDRs.

Multiple Regression Results: Cause-Specific Death Rates

I will now continue the OLS modeling of county death rates, but now will focus on mortality for the three most prominent causes of death in the U.S. today. The format of these CSDR models is the same as the format used above for the ASDR models, with the obvious exceptions that the dependent variable and the g-score reflect a specific CSDR.

Model 20 represents the OLS model with the neoplasm CSDR as the dependent variable (Table 27). Medicare expenditures, the population size of the county, and the spatial relationship g-index are all significant predictors of the neoplasm CSDR. The Medicare and population coefficients are very small, in the tens of thousandth range. The relationship between the g-index and neoplasm CSDR is positive and significant; for every point increase in the g-score, the neoplasm CSDR increases on average by 0.03.

Standardizing the coefficients shows that Medicare and population of the county do not have large relative impacts on the CSDR. The Getis score has the largest impact by quite a margin; with every one standard deviation increase

in the z-score, the Neoplasm CSDR increases, on average,

0.29 standard deviations.

Table 27: Model 20 County Level Regression Predicting Neoplasm CSDR: 2,534 Counties

	Coef.	Std. Error	P	Standardized Coef.
Median Age	0.00	0.00	0.59	0.01
Medicare Expenditure	0.00	0.00	0.05	0.04
Population	0.00	0.00	0.05	-0.04
G I Z-score	0.03	0.00	0.00	0.29
Constant	1.10	0.04	0.00	
R-squared	0.09			

The dependent variable for Model 21 is county stroke

CSDR (Table 28). The coefficients for the stroke CSDR

model behave very much like the coefficients in Model 20.

Medicare and population size have modest effects on the

CSDR, while, alternately, an increase in the z-score

accounts for, on average, a .15 increase in the stroke

CSDR.

The standardized coefficients are similar to those

calculated above for the neoplasm model, although there are

a few noteworthy differences. Medicare expenditures have a

negative effect on the stroke CSDR, whereas it had a

positive effect on the neoplasm CSDR. The magnitude of the

impact of population on the CSDR is more than double what

it was in the neoplasm CSDR. The spatial autocorrelation

statistic behaves in much the same way as it did in Model

20a although its magnitude is slightly less.

Table 28: Model 21 County Level Regression Predicting Stroke CSDR: 2,534 Counties

	Coef.	Std. Error	P	Standardized Coef.
Median Age	-0.02	0.01	0.07	-0.03
Medicare Expenditure	0.00	0.00	0.01	-0.05
Population	0.00	0.00	0.00	-0.10
G I Z-score	0.15	0.01	0.00	0.21
Constant	5.55	0.34	0.00	
R-squared	0.06			

Model 22 is the final model in the county-level

analyses; it predicts heart disease CSDR among the U.S.

counties. The effects of the independent variables

predicting heart disease CSDR are somewhat anomalous when

compared to the other CSDR models. The median age of the

county has a significant positive relationship with heart

disease CSDR, while it was not significant in the other

CSDR models (Table 29). Average Medicare expenditures were

a significant predictor in the other CSDR models, but it

does not have the same effect on the county heart disease

CSDR. Population size and the spatial relationship g-index

have similar effects in this CSDR model as was in the

previous CSDR models.

The standardized coefficients have more in common with the ASDR models than they do with the other CSDR models in this analysis. Median age has the greatest impact on the heart disease CSDR slope followed closely by the g-score index. Of the CSDR models, the standardized effect of the spatial relationship g-index variable is at its largest for the heart disease model.

Table 29: Model 22 County Level Regression Predicting Heart Disease CSDR: 2,534 Counties

	Coef.	Std. Error	P	Standardized Coef.
Median Age	0.12	0.00	0.00	0.44
Medicare Expenditure	0.00	0.00	0.13	0.02
Population	0.00	0.00	0.00	-0.06
G I Z-score	0.14	0.01	0.00	0.41
Constant	-1.38	0.16	0.00	
R-squared	0.45			

The population size of the county and the spatial relationship g-index variable were consistent predictors of the CSDRs in Models 20 through 22, with the spatial relationship having the largest impact on the CSDR in the neoplasm and stoke groups. Median age of county was only significantly associated with heart disease CSDR. Average Medicare expenditures were usually a significant predictor of the CSDRs, although the direction of the effect was not always the same.

Testing the Hypotheses

Having concluded the county level analyses, I will now address whether or not my earlier stated hypotheses are supported by the results of models 11-22.

There was a significant amount of autocorrelation present across the counties of the U.S for each of the ASDRs and CSDRs included in this study. The significant global autocorrelation statistics support my strategy that it would be appropriate to look for significant amounts of autocorrelation between the counties.

Estimating a local autocorrelation statistic for each of the contiguous U.S. counties showed that there was a significant amount of dispersion and clustering of ASDRs and CSDRs between the counties of the U.S. This was evidenced by the Getis z-scores that I then incorporated into the county level models estimated above.

The independent variables did not perform consistently in the three ASDR models though there were similarities. Average Medicaid expenditures was only a significant predictor of the 65-74 ASDR. The population size of the county was significant in all three models, although its effects were very small. Median age proved to be a constant positive predictor of the ASDRs and usually had the largest standardized effect of the independent

variables. More importantly the spatial measure g-index used to define the relationship between the ASDRs of the counties was significant in all the ASDR models and had one of the largest standardized effects.

The three CSDR models shared few similarities with the ASDR models, and there were limited consistent findings between the CSDR models. As was shown in the results for the ASDR models, population size of the county was a very small but significant predictor, and its standardized effect was negative. Medicare expenditures was important in predicting the stroke and neoplasm CSDRs. Median age of the county only significantly predicted the heart disease CSDR. A finding that was fairly consistent among all the CSDR and ASDR models is the impact that the spatial relationship g-index variable has on the death rates of the counties. The relationships between the CSDRs of the counties are significantly and positively related to the CSDRs, and they usually have the greatest standardized effects.

My county level hypotheses were generally supported in the analyses of this chapter. This research provides evidence that ASDRs (65-74, 75-84 and 85+) and CSDRs (neoplasm, heart disease and stroke) are impacted by a county's spatial relationship to other counties, as well as

by substantive properties and contexts specific to the county. This lends support to the importance of studying mortality beyond just the individual level. Another finding that can be taken from this county level analysis is the difference between individual and aggregate level mortality. Medicare proved to be a significant predictor for individual age at death, but was a sporadic predictor of county death rates. This suggests that the analysis of aggregate mortality may well necessitate the inclusion of variables that are not simply aggregates of individual characteristics.

To continue the comparison between the ASDR and CSDR models, I would like to offer a tentative reason why the predictors for ASDRs and CSDRs should not always be the same. The predictive variables utilized so successfully in explaining the variation in ASDRs were not as useful for explaining the variation in all CSDRs. The explained variance for the three ASDR models and the heart disease CSDR model ranged from 44 to 53 percent. The explained variance for the stroke and neoplasm CSDRs were both under 10 percent. This suggest perhaps that not only should different models be used when studying individual and county level mortality, but there should also be different

approaches considered when studying different causes of aggregate mortality.

The next and final chapter of my dissertation provides a discussion and conclusion for these analyses reported in this and the preceding chapters. In this final chapter, I will also offer suggestions for continuing this research and I will attempt to place my findings in the broader context of mortality research today.

CHAPTER VII

DISCUSSION AND CONCLUSIONS

In this final chapter of my dissertation I discuss the major features findings of the micro and macro level mortality analyses that I conducted and reported on in Chapters V and VI. I also consider how the different findings inform a larger conclusion about the state and general status of mortality in the United States. I also offer a critique of the methods I used in this dissertation, and I attempt also to place my findings in a larger context.

Micro-level Findings

One of the more interesting results in the micro-level analyses had to do with the effect of the race variables on age at death. Specifically, race behaved in the opposite way expected based on a review of prominent literature. Blacks, Mexican-Americans and Hispanics all showed a definite longevity advantage over Whites in my analyses. Scholars have found mixed evidence of Mexican-American and Hispanic longevity being similar to that of Whites, a phenomenon known as the Hispanic paradox (Markides and Eschbach 2005). My research found that Mexican-Americans have more than a year of life advantage when compared with Whites. This advantage increases to around four years when

insurance is introduced into the equation, suggesting that Mexican-Americans are better able than Whites to exchange access to life insurance into extra years of life. The same is true for Hispanics, i.e., non-Mexican Hispanics, although the magnitude of the longevity advantage over Whites is even larger than it is for Mexican-Americans. Though it is not unexpected to find that Mexican-Americans and Hispanics have similar ages at death compared to Whites, the longevity advantage shown by these analyses is unusual.

The advantage Blacks have over Whites as reported in my analyses in Chapter V was also unexpected. It has been well reported that Blacks are not likely to live as long as Whites and do not do as well at exchanging higher levels of socioeconomic status for longevity protection. This can be seen, slightly in my analysis, when I examined what happened to the Black longevity advantage when insurance variables were added to the analyses. Blacks consistently lost months of life advantage with the addition of insurance indicating that, as a group, they were not as successful at exchanging access to life insurance into extra months of life.

One variable that behaved as expected was sex. My analyses showed that being female provides a tremendous

longevity advantage over males. My research showed that females are expected to live around twenty-five years longer than men. Adding insurance halved this longevity advantage. In this instance access to insurance served to decrease the discrepancy between males and females. This is especially interesting given the history of the female versus male life expectancy differential. Before the Industrial Revolution many women regularly died in childbirth impacting women's overall life expectancy and causing it to be shorter than men's. Today in the United States, the maternal mortality rate, i.e., deaths to women due to puerperal causes, is a low 10 maternal deaths per 100,000 live births. But in the 19th and early 20th centuries it was much higher. This is no longer anywhere near a major cause of death for women. As death in child birth waned women began to outstrip men in terms of longevity. Today we can see this advantage the world over where women are expected to live eight years longer than men in the more developed countries of the world and three years more in the less developed countries (PRB 2009). It would seem that giving men access to insurance would serve to reduce this female advantage, and this is what I found in my research.

Marriage and poverty showed a similar pattern as the ascribed variables when insurance was added to the equation. However, and surprisingly, marriage was shown in my analyses to have a negative relationship with mortality. Including insurance with the ascribed variables did serve to decrease the longevity disadvantage of marriage and poverty giving support to the idea that access to insurance can be something of an equalizer in relationships of certain achieved statuses and mortality.

A person's own view of his/her health was shown to be positively associated with age at death. Adding insurance to the analysis increased the advantage of a positive view of health. Another way of interpreting this finding would be that among those who perceive themselves in poor health, when Medicare, Medicaid and/or private insurance were brought into the analysis, they were shown to more likely to be able to live a longer life than if they did not have access to the longevity protection that insurance provides. It would be interesting to continue to explore the relationship between perceived health and insurance to determine how people with insurance feel about their health. As I noted earlier in this dissertation, people without health insurance are more likely to not practice preventative medicine which could well impact their

perception of health and exacerbate the relationship between their self-classification of health and mortality.

Tobacco use has been linked to numerous diseases and has served to abbreviate the life expectancy of users, children of users and others who are constantly exposed to second hand tobacco smoke. Without insurance in the model tobacco users were shown to live, on average, almost six years less than non-users. Including insurance reduces the longevity disadvantage of smoking by two years. Smoking was not a significant predictor of death in the complete model indicating that achieved and health factors may have more of an impact on longevity than does smoking. There could also be an interaction between smoking and other variables that I did not pick up on in my analysis. This provides mixed support to the argument that having insurance can increase a person's life expectancy despite their own behavior.

The federally funded Medicare and Medicaid programs unfailingly showed a positive impact of around 15 years (for Medicare) to 29 years (for Medicaid) on age at death. The effect of private insurance was less dramatic but still provided a longevity advantage of about seven years over those without private insurance. It is fair to say that having Medicaid, Medicare or private insurance indeed

grants access to longevity and redistributes the longevity advantage and disadvantage among different groups. By having access to these insurances, people are given access to years of life despite their ascribed and achieved descriptors, their view of their health and their behavior.

What I also find interesting about the magnitude of the longevity advantage provided by insurance is how much greater the advantage is for Medicaid and Medicare when compared to private insurance. I had hypothesized that private insurance would provide some mortality protection, but I was unsure of the effect that federal health care would have. An especially exciting finding is that Medicaid, which is available for a limited number of years throughout a person's life and only in instances of great financial need, can have such a dramatic effect on age at death. If one accepts the idea that a person will only utilize Medicaid in instances of need, it is also possible to accept the idea that even a limited amount of insurance can provide longevity protection in certain situations.

All types of insurance served to increase length of life when the models controlled for race, but the effect was not as clear when looking at race-specific models. Medicaid usually only provided access to increased months of life for the White subset (and for blacks in the

demographic and complete models). This uneven ability to convert access to Medicaid into increased longevity for Mexican-Americans and (to an extent) blacks may well be seen as evidence that the racial inequality in life expectancy may be a product of the structure of Medicaid though more research must be done to sufficiently explore this discrepancy.

Macro-level Findings

As I stated earlier in my dissertation, I believe just looking at mortality only at the individual level is a mistake. People exist in a context, but this context is more than a culmination of the description of the place they live. The context in which people live reacts to the places around it and is defined by something greater than the specific descriptors of that context; it is also defined by the spatial relationship of the context. I therefore conducted a county level analysis in an attempt to ascertain whether or not there is a significant relationship between mortality and context that extends beyond the specific attributes of the county.

I began the county level analysis by looking at whether or not there was a relationship present between death rates across the continental U.S. The resulting statistically significant Moran's I coefficient provided

verification that death rates across the U.S. counties are
correlated. This first step was important because it would
be a test of the existence of a spatial relationship with
regard to mortality across the U.S. counties. This test
also enabled me to determine whether Tobler's first law of
geography applied to mortality and, therefore, whether
there was knowledge to be gained by studying aggregate
level mortality.

I next examined several substantive characteristics of
the counties and their relationships with age-specific
mortality and cause-specific mortality. Median age of the
county was shown to be a positive and significant predictor
of the three Age-Specific Death Rates as well as the heart
disease Cause-Specific Death Rate models. The predictable
performance of median age and death rate lends further
support to the well established relationship between age
and death. The lack of a relationship between median age
and the stroke and neoplasm CSDRs invites questions about
important and unimportant effects of certain types of
cause-specific mortality.

I showed that death rates were significantly impacted
by the relationship the death rates of the county had with
the other counties in the U.S. In all six ASDR and CSDR
models the greater the relationship between county DRs, as

measured by the Getis index, the higher the predicted DR. I was also able to show that the spatial relationship among the 85+ ASDRs and among the stroke and neoplasm CSDRs had the largest impact on the respective death rates, and the second largest impact in the heart disease CSDR, and the 65-74 and 75-84 ASDRs. This is a significant finding in the study of aggregate mortality. A possible implication of these results is that the relationship between counties can impact county death rate behavior more so than Medicare expenditures, more than population size and, in some cases, more than the mean age of the county. This may prove particularly useful for identifying why pockets of high death rates exist across the U.S.

Informing Micro and Macro Study

Another interesting finding of my dissertation is the relationship between Medicare and mortality. Medicare is a significant predictor of additional years of life at the individual level, but a sporadic indicator at the county level. Medicare was a significant predictor of the 65-74 ASDR, and also the neoplasm and stroke CSDRs. It is important not to compare these micro and macro level findings directly since the individual level Medicare variable reflects whether or not a person has access to Medicare, and the county level variable reflects the per

capita expenditures to Medicare. But it is worth speculating as to why Medicare has such a dramatic impact on age at death but not on a county's death rate. It could be due to inconsistent measures of Medicare across levels of analysis, or it could be due to the utilization of Medicare. The individual may be better able to exchange Medicare for access to longevity protection, but the county may not be as effective in exchanging Medicare for decreased death rates.

The discrepancy between Medicare's relationship with mortality at the individual and county level also points to the importance of studying mortality at more than one level of analysis. Individual predictors will not always be the same as aggregate predictors. A common and faulty criticism of aggregate level mortality studies is that aggregate mortality measures are simply the measure of aggregated individual level data and therefore unnecessary for analysis; that is, by understanding mortality at the individual level you understand mortality at the aggregate level. The discrepancy between Medicare's performance at the micro and macro levels can, cautiously, be taken as evidence that relationship of place is an important consideration in the study of mortality and deserves

further exploration to understand how the unique context of place impacts mortality.

Reevaluation of the Mortality Methods

A disadvantage of my individual level research analyses is the cross sectional approach of this design. Another way to approach this analysis would be to use a time series statistical model to estimate mortality, such as a Cox proportional hazard model. Unfortunately, the micro-data I used in this dissertation were not appropriate for Cox modeling. But it would be useful to explore whether the length of time a person has insurance further impacts his/her age at death. Another method that may inform what is happening would be logistic regression. By including the living portion of the sample and using age as a predictive variable it could be possible to discern what factors lead to an increased likelihood of dying. These results could then be used to inform a race-specific analyses, limited to the decedents, to determine if access to health insurance translates into longevity protection.

A major limitation of the county level analyses is the small number of predictor variables. My county level analysis was a brief foray into the relationship between spatial autocorrelation and mortality. Though the explained variance for the ASDRs and heart disease CSDR

were 40 percent or so higher, many variables that are usually associated with death rates were not included in these analyses. It would be interesting to see the overall predictive capability of the county level models with the inclusion of pollution measures, economic measures and measures of health.

Were I now to begin again this research project knowing now what I know, I would attempt to find more common predictors of mortality between the individual- and the aggregate-level analyses. For example I would search out Medicaid and private insurance county expenditure figures, the relationship of poverty between the counties and let the individual and county level findings inform the question of whether mortality is different at different measurements.

Significance of This Research

I have shown that the spatial relationship of county-level death rates with the death rates of other counties is an important consideration in the study of county level mortality. Spatial context proved to be a significant and consistent predictor of death rates whether the rates reflected age or cause of death, whereas other predictor variables dealing with characteristics of the counties themselves proved to be inconsistent and not always viable.

It is clear from this analysis that insurance provides longevity protection for individuals. But this protection does not seem to erase inequality in mortality between racial groups. This access to longevity protection is something that can be extended to every single person. With the possible coming of universal health care in the U.S., it will be interesting to see if this access to longevity protection continues to reduce disparity in longevity.

When I began thinking about and developing the overall issues and questions to be addressed in this dissertation, I was particularly interested in the discrepancy in longevity among groups and what a society could do to attenuate those discrepancies. It was my thought that studying the discrepancy between individuals would provide some evidence and suggestions about how to abbreviate the longevity inequity among adults in the U.S. I was able to provide evidence for how it may be possible to inject some equality in access to longevity at the individual level through access to insurance, especially federally funded insurance. But I was not able at the county level to provide similar evidence. By continuing this county level research and continuing to measure the spatial relationships between predictive variables, it may be

possible to present a more accurate picture of county level
mortality and how the society may be able to begin to
eliminate the disparity.

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