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Income inequality and mortality: a test of competing pathways

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INCOME INEQUALITY AND MORTALITY: A TEST OF COMPETING PATHWAYS

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DEDICATION

This dissertation is dedicated to my parents, Don and Renae Winters, who have made their unconditional love and unwavering faith in me apparent throughout my entire life.

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ABSTRACT

Findings from numerous studies indicate that individuals living in more unequal societies are at greater risk for a variety of health problems. However, questions remain about the possible pathways that link health outcomes and income inequality. In general, the debate about *how* income inequality affects individual health centers around two issues: 1) whether the relationship is representative of the level of social cohesion within a given area, and/or 2) whether it is more indicative of the level of local investment in public health infrastructure. Each of these theories, then, represents a potential mediating mechanism through which income inequality impacts individual health.

The purpose of this dissertation is to examine the social cohesion and local investment mechanisms through which income inequality may impact individual-level health outcomes. By examining variation in levels of social welfare spending and civic engagement, I investigate which of these competing variables has a stronger mediating effect in the relationship between income inequality and individual health outcomes. To address this research question, I use data from the Integrated Health Interview Series (IHIS)—a collection of microdata based on the public use files of the U.S National Health Interview Survey (NHIS) which is linked to the National Death Index (NDI). Using multi-level modeling techniques, I simultaneously examine the role of environmental-level effects (i.e. degree of local investment/social cohesion) and individual-level effects (e.g. income) on the likelihood of individual mortality in metropolitan areas.

The findings presented in this dissertation contradict previous claims about the Income Inequality Hypothesis, which suggests that income inequality is detrimental to individual health. In addition, findings do not support the Social Cohesion or Local Investment Mechanisms as

mediating pathways through which income inequality impacts individual health. These results raise questions about the causal effects of income inequality, and the sensitivity of this relationship to level of aggregation and to what factors research choose to control.

CHAPTER ONE: INTRODUCTION

Income inequality is on the rise in many developed countries, and the United States is no exception. Although the distribution of income remained fairly stable during the period between WWII and 1970, income inequality began to steadily increase during the decades that followed, and today the U.S. ranks as having one of the highest levels of income inequality among Western industrialized nations (Weinberg 1996; OECD 2008). This increase in income inequality is driven by changes in the top and bottom of the income distribution. For example, in 2000, the income share held by the poorest twenty percent of Americans hovered around 5%, while the shares of income going to the richest 10% of Americans was approximately 30% (World Bank 2006). Given recent trends in globalization and data suggesting that the growing income disparity shows no signs of slowing down, many researchers have begun to examine the social consequences associated with living in a society characterized by economic inequality (Alderson & Nielsen 2002).

Why should we care about growing income disparities? One reason is that a growing body of evidence suggests that unequal societies are host to a range of modern social problems, including drug abuse, violence, obesity, and teenage pregnancy (Wilkinson & Pickett 2009; Gold et al., 2004; Pickett, Brunner, & Lobstein 2005). The wage distribution has also been found to play a large role in shaping individual health and wellbeing. Although not all the evidence is consistent, the majority of studies examining the effects of income inequality on health outcomes have found that high levels of income inequality are associated with a number of negative health outcomes, including lower life expectancy, higher infant mortality, and poorer average physical and mental health status.

Despite an extensive literature on the detrimental effects of income inequality on individual health, there is an ongoing debate about the precise mechanisms that explain this relationship. In general, questions about *how* income inequality affects health centers around two issues: 1) whether the relationship is representative of the level of social cohesion within a given area, and/or 2) whether it is more indicative of the level of local investment in the public health infrastructure. Each of these theories represents a potential mediating mechanism through which income inequality impacts on individual health outcomes.

Proponents of the social cohesion interpretation argue that awareness of relative income differences produces negative emotions that are then translated into poorer health via anti-social behavior, reduced civic participation, and less social capital. For example, awareness that one is of a lower social status may foster feelings of shame and mistrust that may ultimately lead to stress-induced behaviors such as smoking, drug use, and excessive drinking.

In contrast to this perspective, other scholars propose a more structural link between income inequality and health outcomes. Under the local investment interpretation, the inequality-health relationship is explained through the unequal distribution of material resources that are likely to improve the physical and mental wellbeing of all individuals. In other words, inequality affects individual health because it determines the availability of public and private resources that have the potential to reduce the probability of negative health outcomes, such as hospitals and/or health care personnel.

If the negative health effects of an unequal income distribution are contingent on social or material living conditions, it seems logical that community investment in either would largely mediate the relationship between income inequality and public health. However, research on the pathways through which this relationship operates is still heavily debated (for example see

Lynch et al. 2000 and Marmot & Wilkinson 2001). A review of the literature shows that there are very few studies that have tested these competing theories, and those that have done so have examined the association only at the national and/or state levels. Given the influence that lower levels of government and local organizations have about funding for public health infrastructure and various arenas for civic engagement, research on these potentially mediating mechanisms would benefit from an analysis of smaller areas (such as communities).

The purpose of my dissertation is to examine the social cohesion and local investment mechanisms as causal pathways through which income inequality may operate on individual health outcomes. By examining variation in levels of local investment and social cohesion, I hope to determine which of these competing variables has a stronger mediating effect on the relationship between income inequality and individual health. To address this set of research questions, I use the Integrated Health Interview Series (IHIS)—a collection of microdata based on the public use files of the U.S National Health Interview Survey (NHIS). These data contain a range of information on individuals aged 18 and over, including health-related behaviors and conditions, access to health insurance and medical care, and a variety of household and socio-demographic characteristics, such as race, age, income, and education. For the years 1986-2006, these person records also include information on the final mortality status of each individual, as reported by the National Death Index (NDI), a national database that stores death certificate records from state vital statistics offices

The IHIS also provides geographic identifiers for NHIS respondents so that data on the social environment may be linked to each person record, which allows researchers to situate individual-level outcomes within the broader social and economic context. The use of multi-level data allows me to investigate the association between community context and an

individual's probability of mortality, controlling for individual characteristics that may potentially impact on health. Using an event history regression technique, I simultaneously consider the effect of environmental-level factors (i.e. level of income inequality, level of local investment, degree of social cohesion) and individual-level characteristics (e.g. income) on the likelihood of mortality at the individual level. My analysis is organized around two objectives. First, I examine whether income inequality in metropolitan areas is related to individual mortality, over and above the effects of individual characteristics. Second, I test whether the inequality-mortality relationship is attenuated by social and structural mechanisms.

Given the policy implications of these research questions, an analysis of the role of income inequality as a contextual variable provides insight into whether or not income inequality matters in the determination of individual-level health and well-being. Discussion and testing of these relationships is important, given their clear political implications. If contextual variables have a limited impact on individual health, then policy makers should focus on the development and implementation of policies aimed at improving the absolute income for families and households. However, if income inequality alone is detrimental to individual health, then policies designed to alter the distribution of income would be more beneficial at reducing public health concerns. Furthermore, a comprehensive understanding of the competing pathways through which income inequality affects health outcomes would allow for the expansion of policies designed to buffer the negative effects of an unequal income distribution. For example, local governments dedicated to decreasing public health problems would be more knowledgeable about which types of policies are most effective at improving the health of individuals in their communities. Policy makers could then be more confident in deciding whether local finances should be spent on health-related infrastructure and services that improve public access to

medical resources, or whether investment is better served in the development of community organizations and social programs that may increase levels of civic engagement.

This dissertation contributes to the literature in two ways. First, it investigates whether variables related to the social cohesion mechanism or variables related to the local investment mechanism are more effective in mediating the relationship between income inequality and health. Previous studies examining these relationships have largely investigated such pathways separately and without comparing the relative strength of their intervening association with mortality. This dissertation includes such variables in the same multi-level models, which provides a more thorough examination of each contextual variable's ability to explain variation in mortality than found in previous research.

Second, these competing pathways have generally been assessed at the national or state levels. Studies that have been conducted at lower levels of aggregation such as metropolitan areas or census tracts have generally produced mixed results or found null effects for the income inequality-health relationship. However, this may be because prior studies have suffered from limited statistical power (Subramanian & Kawachi 2004). This dissertation uses more advanced analytical techniques and multi-level modeling to determine the independent contribution of community income inequality to individual health, and the mediating effects of social cohesion and local investment in this relationship, for Metropolitan Statistical Areas (MSAs). In addition, this dissertation uses longitudinal rather than cross-sectional data, which allows for an assessment of the causal nature of the relationship between income inequality and individual health outcomes, and avoids the limitations of using cross-sectional data to draw inferences about the variables of interest.

In Chapter 2, I provide a literature review of previous studies that have examined the relationship between income inequality and health. In addition, I discuss the two major theoretical pathways through which income inequality is believed to impact on individual health outcomes: the social cohesion mechanism and the local investment mechanism. Chapter 3 presents theoretical concept maps and a description of the hypotheses tested in this dissertation. Chapter 4 provides a description of the data sources, and all dependent, independent, and control variables employed for this dissertation. In addition, method of analysis, descriptive statistics, and key bivariate relationships are discussed in detail. In Chapter 5, I discuss results of the analyses, including the effects of the income distribution on individual health outcomes and the potential mediating properties of each of the two mechanisms for this relationship. Finally, in Chapter 6, findings are summarized, and conclusions, limitations, and directions for future research are discussed.

CHAPTER TWO: THEORETICAL BACKGROUND

THE PROBLEM: HOW DOES INCOME INEQUAILTY POSE A RISK TO INDIVIDUAL HEALTH?

In this chapter, I discuss the relationship between income inequality and health, providing a review of empirical evidence documenting the detrimental effects of income inequality for individual health outcomes even after account for individual-level characteristics, such as income. I provide a detailed description of the two major pathways through which income inequality is theorized to operate on individual health outcomes, and I review recent studies that have tested these mechanisms at the national, state, and local levels. Finally, I summarize the existing literature on these relationships and propose the three hypotheses that are tested in this dissertation.

INCOME INEQUALITY AND HEALTH

Research examining the determinants of health has traditionally focused on individual-level differences in socio-economic status (SES) and the various health outcomes associated with them (for a review, see Feinstein 1993). This extensive literature demonstrates a positive relationship between SES and health at the individual level. For example, early research has shown that impoverished individuals are much more likely to have diminished health outcomes than individuals who are not in poverty. More recently, scholars have recognized that the health effects of SES are not only associated with individuals living in extreme poverty, but also with individuals at higher levels of SES. This research demonstrates that there is a graded association between SES and health, and that individual morbidity and mortality vary among all levels of SES (Smith & Eggar 1992, Marmot et al. 1984, Bunker et al. 1989). For example, research has shown that the likelihood of mortality and infant mortality continues to drop as one goes up the

income hierarchy. This gradient effect is also well-documented for the prevalence of chronic disease (Adler et al. 1993) and several risk factors associated with chronic disease, such as smoking, cholesterol levels, and sedentary lifestyles (Winkleby et al. 1992).

Scholars have also suggested that individual health may be affected by the distribution of income within society. Such studies suggest that there is a direct link between income inequality and individual health. For example, several scholars have argued for the Income Inequality Hypothesis (IIH), or that it is inequality and not absolute income that matters in determining individual health outcomes (Wilkinson 1992, 1996; Kaplan et al. 1996; Kennedy et al 1996). Although the evidence is mixed, the majority of studies linking income inequality to health find that there is a significant negative association; that is, greater income inequality produces lower standards of health (Rodgers 1979 and Flegg 1982). For example, in a study of mortality across U.S states, Kaplan, Pamuk, Lynch, Cohen, and Balfour (1996) find a positive correlation between income inequality and all-cause mortality. This finding is supported by Kennedy, Kawachi, and Prothrow-Stith (1996) who use two different measures of inequality (the Robin Hood index and the Gini coefficient) to measure the effect of state income inequality on all cause and cause-specific mortality in the United States. The authors conclude that inequality of income is positively related to total mortality and infant mortality, even after adjusting for poverty.

More recent studies have identified the detrimental effects of income inequality on a range of cause-specific mortality outcomes. In a cross-national comparison of industrialized countries, Kim, Kawachi, Hoorn, and Ezzati (2008) find that income inequality has a positive relation to the prevalence of coronary heart disease, strokes, and a variety of related conditions including obesity and high blood pressure. These findings support other research that finds a

significant, positive association between income inequality and obesity for adults (Pickett et al. 2005) and for children (Pickett and Wilkinson 2007) at both the international and state levels.

Despite such evidence, other studies have shown that results related to the income inequality hypothesis are mixed. For example, Mellor and Milyo (2002) use data from the 1995-1999 Current Population Survey to examine the effect of income inequality on self-rated health status for both the general population and for individuals living in poverty in metropolitan areas. They conclude that the association between income distribution and health disappears after controlling for household income. Likewise, Ficsella and Franks (1997) assess the ecological relationship between income inequality and all-cause mortality and find that adjustment for individual household income renders the association insignificant. Findings by Deaton and Lubotsky (2003) suggest that the relationship between income inequality and mortality may also be confounded by the effects of racial composition. They find that the correlation between income inequality and mortality disappears when adjusting for the percent black at both the city and state the level. These findings raise questions about the causal effects of inequality and the sensitivity of this relationship to level of aggregation and to what factors researchers choose to control. According to Wilkinson (2007), income inequality is closely related to health at higher levels of aggregation within countries and states. In smaller areas such as counties or metropolitan areas, however, this relationship is less robust.

Although such findings have raised debate within the income inequality literature, extensive reviews by Wilkinson and Pickett (2006, 2009) and Lynch et al. (2004) demonstrate that, for the most part, income inequality and health are negatively correlated at almost every level of aggregation. A key shortcoming of this line of study is that scholars have yet to identify the exact causal mechanism through which income inequality affects health outcomes. In the

next section, I present a theoretical discussion of the proposed pathways through which income inequality influences health outcomes. I discuss the social cohesion and local investment mechanisms in detail, and provide an overview of existing research that has documented the mediating effects of these mechanisms in the inequality-health relationship. Finally, I identify the specific hypotheses being tested in this dissertation and provide information on the data and methodology adopted for disentangling the causal link in the relation between income inequality and health.

PATHWAYS LINKING INCOME INEQUALITY TO HEALTH

Although the majority of findings suggest that individuals living in more egalitarian societies do have better health outcomes, questions remain about the possible pathways that explain this relationship. The two most recognized theories for how income inequality influences health are the social cohesion mechanism and the local investment mechanism (Lynch et al. 2000; Marmot & Wilkinson 2001). Each of these theories describes a different pathway through which community income inequality impacts individual health outcomes. Discussion and empirical testing of these pathways is important, given their clear theoretical, empirical, and political implications for communities seeking to reduce public health problems. I describe each mechanism in more detail below.

The Social Cohesion Mechanism

The first pathway through which income inequality may affect individual health is social cohesion. Proponents of this interpretation argue that being exposed to uneven distributions of income produces negative emotions that are then translated into poorer health via anti-social behavior reduced civic participation, and lower social cohesion (Marmot & Wilkinson 2001). In

other words, visible differences in relative income produce negative psycho-social consequences that affect the ways individuals behave and interact. For example, Wilkinson (1996) argues that income inequality affects health outcomes because the awareness that one is of a lower social status fosters feelings of shame and mistrust that ultimately lead to stress-induced behaviors such as smoking, drug use, and excessive drinking. At the same time, perceptions of income inequality are argued to reduce social capital, trust, and self-efficacy, which have all been demonstrated to have positive effects on health (Berkman 1995).

Several studies support the notion that income inequality undermines social cohesion and yields harmful effects on health. In their analysis of the 50 U.S states, Kawachi et al. (1997) find evidence that greater income inequality is strongly associated with lower levels of social cohesion. Their analysis demonstrates that income inequality influences the quality of social relations—in states where income differences were large, there was low per capita density of membership in voluntary groups and low levels of social trust, as measured by the proportion of residents in each state who believed that people could be trusted. Other studies have shown that more unequal societies do not only weaken social affiliation, but may in fact be associated with increased racism (Kennedy et al. 1997), discrimination against women (Kawachi et al. 1999), and overall hostility (Williams, Feaganes, & Barefoot 1995). These studies and others are indicative of what Wilkinson (1999) identifies as a "culture of inequality," which is characterized by a lack of social cohesion and individuals who are less trusting and more violent.

The Local Investment Mechanism

Although an extensive literature focuses on the individual perceptions of inequality and social comparisons in an unequal environment, other scholars propose a more structural link between income inequality and health. Proponents of the local investment theory point to a lack of social

spending on health services, health education, and other key areas related to social welfare as the causal link between income inequality and poor individual health. In contrast to the social cohesion theory's emphasis on the connection between perceptions of relative deprivation and a decline in health, this interpretation of health inequalities calls attention to the unequal distribution of material resources that are likely to impact the level of physical and mental wellbeing of individuals. Under this interpretation, the relationship between income inequality and health is explained by systematic underinvestment in a variety of human, physical, health, and social infrastructure (Smith 1996; Lynch & Kaplan 1997; Kaplan et al. 1996; Lynch et al. 1998). In other words, inequality affects health and mortality because it determines the availability of public and private resources that have the potential to reduce the probability of negative health outcomes and death.

Prior research suggests that that the relationship between income inequality and level of government social welfare spending can either be positive or negative. On one hand, an increase in inequality may be associated with lower levels of social spending because the poor lack political influence and are less likely to vote (Mayer and Sarin 2005). If disadvantaged voters feel alienated or lack the resources to get to a voting booth, they will not support redistributive policies that could potentially benefit them the most. On one hand, higher levels of income inequality may lead to more government investment, specifically through democratic calls for redistribution and greater demand for progressive taxation. Romer (1975), Roberts (1977), and Meltzer and Richard (1981) have all suggested that a more unequal income distribution in society leads to a demand for pro-redistributive policies through the "selfish" voting behavior of the median voter. As the income gap widens between the richest and poorest citizens, the median

drops relative to the mean, and the median voter (i.e. the middle class) shows greater support for redistributive policies from which they now stand to benefit.

Building on this theory, Comeo and Gruner (2002) argue that individual preferences for or against redistributive policies are not solely determined by relative economic standing. Using cross-national data from twelve counties, they find that voting behavior is also influenced by non-economic rewards, such as a obtaining a higher social status. They also posit that individuals may be more favorable toward pro-redistributive policies if they believe income distribution is the result of exogenous factors such as family background, rather than individual failure or laziness. These findings are in line with Galasso and Profeta (2002) who argues that "fair voters" (i.e. individuals who support redistribution because they believe inequality is the result of structural disadvantage and not a lack of individual effort) reduce the political relevance of the "selfish" middle class voters.

The potential of government social spending to ameliorate the harmful effects of inequality has recently drawn some scholarly attention. A review of the literature suggests the connection between social welfare systems and population health is strongest at the national level. For example, in a cross-national comparison of nineteen countries from 1970-1996, Macinko et al. (2004) find a significant positive relationship between wage inequality and infant mortality rates, even after controlling GDP per capita. Using two different measures of income inequality, they report that the healthcare financing system of a country and the physician supply per 1000 population significantly attenuate the effect of wage inequality on infant mortality. Conley and Springer (2001) also find empirical support for the idea that governmental provision of health care services has a direct effect on population health outcomes. In a cross-national comparison, they model the impact of welfare spending on infant mortality and low birth weight

and find that the increased investment in public health—as measured by total public expenditure on healthcare—significantly enhances infant health outcomes. These studies support early research by Pampel and Paillai (1986), who found that total government medical expenditures is negatively associated with several measures of infant health, including overall infant mortality, neonatal mortality, and postneonatal mortality.

Research on the association between public expenditures and health at the sub-national level is limited, but also suggests that higher investments in public health may mediate the relationship. Dunn, Burgess, and Ross (2005) find that the relationship between income inequality and population health is partially explained by controls for public investment. In their study of 48 U.S states in 1987, they investigate the relationship between public service levels and all-cause mortality and find that total per capita expenditures on public services, including education and housing, significantly reduces the probability of death.

Kim and Jennings (2009), also studied the effects of social welfare systems on mortality in U.S states and found that the health of U.S citizens is heavily dependent on how state governments approach public programs. Using cross-sectional data for a 10-year-period, they analyzed three dimensions of social welfare systems and found that generous spending on education, progressive tax systems, and lenient welfare program rules help improve population health. In addition, research by Mayer and Sarin (2005) suggests that infants born in states with high levels of income inequality have a higher probability of dying within one year than infants born in states where income inequality is low. In an attempt to directly test the local investment explanation for the association between inequality and infant death, they find that increases in state per capita expenditure on health care did reduce the likelihood of neonatal mortality

Even fewer studies examining the mediating effect of government expenditures on population health have been conducted at the sub-state level, and the evidence is mixed. For example, Ronzio, Pamuk, and Squires (2004) examine the relationship between several local expenditure variables on premature death rates for U.S central cities with a population greater than or equal to 100,000. They find that spending on road infrastructure and police is associated with lower death rates, while per capita spending on other public expenditures, such as education and health services, was not a significant predictor. Lhila (2009) estimates the relationship between income inequality and low birthrate at both the state and county level, and assesses the role that government provision of healthcare has in altering the relationship between income inequality and child health. She finds that investments significantly reduce low birth weight at state level, but that county-level income inequality is not significantly related to low birth weight as a health outcome. This finding (or lack thereof) for counties is consistent with a previous income inequality literature showing that, for the most part, income inequality at lower levels of aggregation is only weakly associated with health outcomes (Wilkinson 1997).

There are two explanations for why income inequality is more closely related to health in studies with larger units of analysis. Subramanian and Kawachi (2004) argue that one reason is that studies using smaller sample sizes (census tract- or county-level) simply lack the statistical power to find an empirical correlation between inequality and health. Additionally, they suggest that the lack of correlation is because the operating mechanism through which income inequality affects health may exist at the state or national level, but not at lower levels of aggregation. In other words, decisions regarding social spending occur in political entities at the national or state level. This may or may not be true. However, further assessment is needed to disentangle the undoubtedly complex relationship between income inequality and health. Findings regarding the

way this relationship works at lower levels of aggregation are decidedly mixed. This dissertation adds to the literature by examining the role social cohesion and local investment in smaller areas in mediating the relationship between community income inequality and individual health.

SUMMARY OF THEORETICAL BACKGROUND

A review of the literature demonstrates evidence that income inequality does have detrimental effects for individual health, but that additional research is necessary in order to understand exactly *how* this association works. Scholars have theorized that one of the ways in which this relationship operates is through the *Social Cohesion Mechanism*. According to these theories, inequality operates on health through the perception of relative deprivation, which produces certain emotions that may have negative biological and psycho-social consequences for individuals. This research suggests that an individual's awareness that he or she is at a disadvantaged position in the social hierarchy can threaten their social esteem and cause them to feel insecure, anxious, or depressed. In this way, areas characterized by high levels of income inequality are socially hazardous environments.

Other research suggests that the inequality-health relationship can be explained through the *Local Investment Mechanism*. According to these theories, inequality operates on health outcomes through systematic underinvestment in infrastructure and other material resources that could reduce the occurrence of ill health or mortality. The few studies that have tested this theory have examined different measures for governmental provision of healthcare and find that the effect of income inequality on health is attenuated by higher levels of government expenditures. However, this mechanism has only been assessed at national and state levels. Given the influence of governments at lower levels of aggregation with regard to decisions about

the allocation of funds for health and other public infrastructure, research on this mediating mechanism would benefit from analyses at a lower level.

This chapter has highlighted the importance of further research that examines the way the *Social Cohesion Mechanism* and the *Local Investment Mechanism* work as pathways to explain variation in individual mortality. In Chapter 3, I present conceptual models for present study, outline the hypotheses that will be tested, and discuss my expectations for results as grounded in the existing literature.

CHAPTER 3: CONCEPTUAL MODELS AND HYPOTHESES

The purpose of this dissertation is to examine the *social cohesion* and *local investment* mechanisms as pathways through which income inequality may impact individual health outcomes. A review of the literature demonstrates support for the inequality-health relationship, and the potential that social cohesion and local investment to serve as a buffer in this relationship. As previously mentioned, however, such studies are often plagued by methodological concerns and mixed results. For example, much of the research examining the inequality-health relationship has been conducted using cross-sectional data at higher levels of aggregation, such as states or nations. The empirical strategy in this dissertation addresses the shortcomings of previous research by using multi-level modeling technique to better investigate the statistical relationships between the variables of interest.

To help address the gaps in existing literature, I first examine the effect of income inequality on individual risk of mortality within metropolitan areas (MSAs, CMSAs, and PMSAs). I then test the mediating effects of social cohesion and local investment in the income inequality-mortality relationship within these metropolitan areas. Using data on individuals, as well as contextual data on the MSAs, CMSAs, and PMSAs in which they live, I test the following three hypotheses:

> H1: Income inequality will be positively related to an individual's risk of mortality, even after controlling for individual income.
>
> H2: The positive effect of income inequality on an individual's risk of mortality will be reduced when contextual characteristics pertaining to community social cohesion are taken into consideration.
>
> H3: The positive effect of income inequality on an individual's risk of mortality will be reduced when contextual characteristics pertaining to local investment in public health infrastructure are taken into consideration.

Figure 1 presents a conceptual model for each of the three hypotheses tested in this dissertation. As Figure 1 demonstrates, Hypothesis 1 estimates the causal relationship between income inequality and the probability of individual mortality. As discussed in the literature review in the previous chapter, several studies have shown support for the Income Inequality Hypothesis (IIH) which suggests that it is income distribution and not absolute income that matters most in determining individual health outcomes (Wilkinson 1996, Kennedy et al. 1998). Based on this previous research, I expect that the relationship between income inequality and the probability of individual mortality will be positive, and that individuals who live in MSAs, CMSAs, or PMSAs that are characterized by higher levels of income inequality will be at a greater risk of mortality than individuals who live in MSAs, CMSAs, or PMSAs that are characterized by lower levels of income inequality.

Hypotheses 2 and 3 examine whether or not the inclusion of potentially mediating variables alters the relationship between income inequality and the probability of individual mortality. Hypothesis 2 tests the theory that social cohesion serves as a buffer against individual mortality. Scholars have theorized that income inequality undermines social cohesion within communities by creating a "culture of inequality" in which residents are less trusting of each other and demonstrate anti-social behaviors which may affect their health (Wilkinson, 1999). Based on this research, I expect that controlling for social cohesion will attenuate the positive effect of income inequality and the probability of individual mortality.

Hypothesis 3 tests the theory that local investment in public health infrastructure serves as a buffer against individual mortality. Scholars have emphasized the connection between income inequality and systematic underinvestment in local health infrastructure which may improve the health of residents (Lynch & Kaplan 1997). This research suggests that inequality

harms individual health because residents living in communities characterized by high levels of income inequality do not have access to material resources that are likely to improve their health. Based on this research, I expect that controlling for local investment will attenuate the positive effect of income inequality and the probability of individual mortality.

These two theories represent very different pathways through which income inequality may operate on individual health. In Chapter 4, I outline the methodological technique used to explore these relationships and describe the operationalization of all variable used to test the proposed hypotheses.

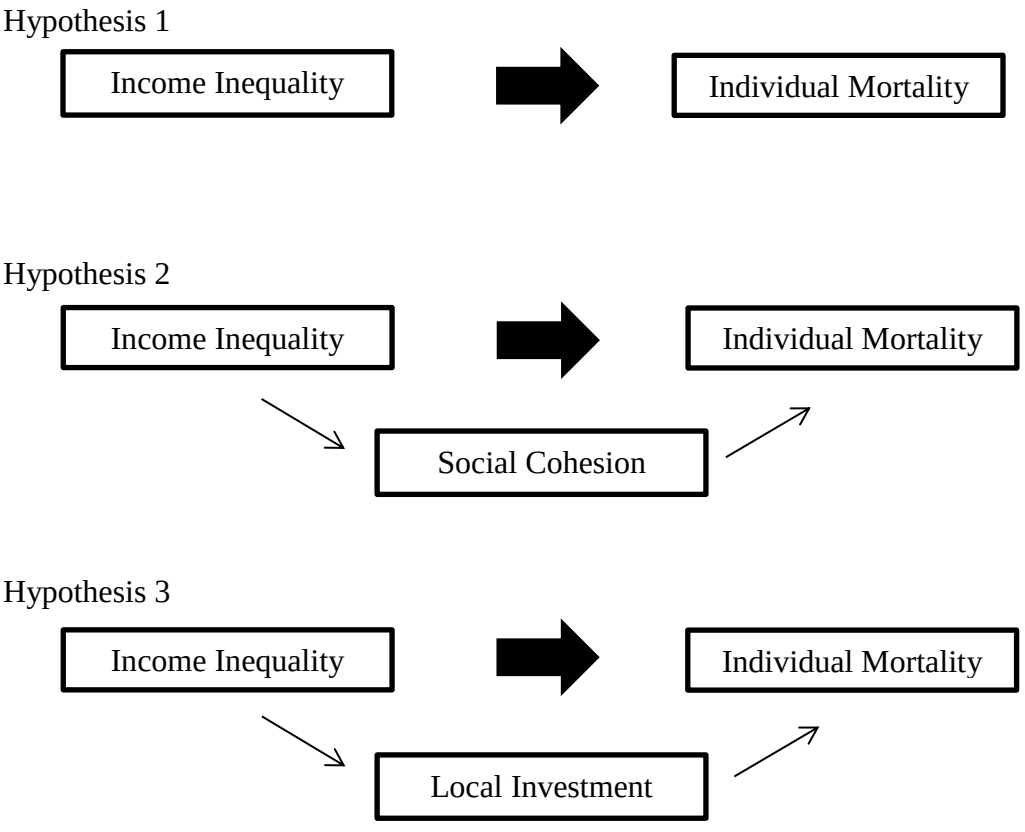

Figure 1. Conceptual Models for Hypothesized Relationships

CHAPTER FOUR: METHODOLOGY

This chapter will focus on the data and methods used in this dissertation. I begin with a description of the data, including both the individual-and contextual-level. I then describe the dependent, independent, and control variables and specify how they are operationalized. Finally, I discuss the type of analysis employed and explain why the method is appropriate for addressing the proposed research questions.

DESCRIPTION OF DATA

This dissertation uses data from a variety of sources. Individual-level data are drawn from the Integrated Health Interview Series (IHIS), a harmonized data set created from the public-use files of the National Health Interview Survey (NHIS) and covering the period from 1969-2009. The integration of these original data—a project funded by the National Institute of Child Health and Human Development (NICHD)—increases consistency in coding schemes across survey years and allows researchers to make cross-temporal comparisons. For example, the coding scheme for the educational attainment variable changes three times in the NHIS data over the period from 1969-2009. During the years from 1969-1981, educational attainment questions are grouped into intervals and reported as years of completed schooling. For the years 1982-1996, however, these data are reported as the number of years of completed schooling in single years. The coding scheme changes once again in 1997, as data are reported as the highest degree attained for those individuals with more than a high school education. To allow for comparability across survey years, the IHIS provides a bridging variable that recodes the educational attainment data into a single, consistent coding scheme for 1969-forward.

The IHIS database is composed of microdata collected from the NHIS, and as of 2010, includes information on over 7,000 integrated variables. Each year, approximately 100,000 persons in about 45,000 households are surveyed through NHIS. In addition to inquiries about core demographic characteristics, respondents are asked questions pertaining to several health related variables, including physical and mental health conditions, personal and family medical history, health behaviors and education, and access to medical care. Respondents are interviewed year to year, providing a longitudinal record of the health of each individual. Because the NHIS is linked with the National Death Index (NDI) for the years 1986-2006, these data also include information on each respondent's final mortality status, along with the year and cause of death. The combination of information regarding individual characteristics and mortality status can then be analyzed to predict the individual probability of death.

Contextual-level data are drawn from the 2000 Census of Population and Housing (STF3), the 2000 Census USA County Business Patterns File, and the 2000 Census American Community Survey. These data include second-level summary measures for median family income, as well as degree of social integration and health infrastructure within each MSA, that can be included in the models to determine their intervening influence on the income inequality-mortality relationship. These files also include several aggregate measures that can be used as second-level predictors or controls in the analysis, including economic structure, minority population, and population size. Individual-level data on respondents from the IHIS who participated in the NHIS between 1986 and 2006 (the years of the survey data that are linked with the NDI) will be merged with contextual data by MSA identifying codes that are made available by both NHIS and the Census. These merged data can then be used to investigate the association between a various individual- and contextual-level factors with individual mortality.

SAMPLE

The unit of analysis in this study is the individual. National Health Interview Survey (NHIS) data are collected through face-to-face interviews of individuals selected through a multistage probability sampling strategy that is carefully designed to produce nationally representative samples of the civilian, non-institutionalized population living in the United States.

Each respondent in the NHIS is assigned a unique IHIS-constructed value (NHISPID). This unique ID includes information indicative of the Metropolitan Statistical Area (MSA), Consolidated Metropolitan Statistical Area (CMSA), or Primary Metropolitan Statistical Area (PMSA) in which each respondent's household is located. Using this information, researchers are able to link IHIS data on individuals with geographic data from other sources, such as the U.S Census. For the purposes of this study, the individual-level data from IHIS were merged with demographic and economic information from the MSA's, CMSA's, and PMSA's in which respondents' households were located. The final sample in this analysis includes all respondents at least 18 years of age who were surveyed by NHIS between 1997 and 2001, and for which household location and final mortality status information was available. This resulted in a sample of n= 148,120 individuals living in 58 MSA's, CMSA's, and PMSA's across the United States. For a list of the MSA's, CMSA's, and PMSA's included in this analysis, see Appendix A.

MEASUREMENT

Outcome Variable

Individual Risk of Overall Mortality

The dependent variable in this dissertation is individual risk of o*verall mortality*, a summary measure frequently used in public health analyses that accounts for all causes of death. After their initial interview, each NHIS respondent is followed and re-interviewed each year until their

death. For each follow-up year that the respondent participates, they are assigned a zero (0), indicating that he or she is still alive, or a one (1) indicating that he or she has died. The individual's *final* mortality status, that is, whether or not they are deceased at the end of the follow-up period, then becomes a measure to contrast those persons who died at some point during the follow-up to those who survived the entire duration. If a respondent is not identified as deceased at the end of the follow-up period, he or she is presumed to still be alive.

Key Explanatory Variables

The key predictor variables in this dissertation include those contextual-level characteristics that are theorized to be associated with mortality, including income inequality, measures for social cohesion, and measures for local investment in health infrastructure.

Income Inequality

The Gini Index of family income inequality is used to measure the dispersion of income within each MSA. This measure ranges from a coefficient of 0—indicating that income is perfectly distributed among the population—to a coefficient of 100—indicating a condition of maximum inequality where one person in the population has all of the income. Various measures are available to quantify income inequality; however, the Gini Index is the most widely used measure and will allow for comparisons with other published works on the relationship between income inequality and health outcomes. In addition, this measure has been shown to be highly correlated with indicators of income inequality, including the Theil Index of income disparity and the Robin Hood Index (Kawachi and Kennedy 2007).

Social Cohesion

Two contextual-level measures of social cohesion are included in this analysis: the number of associations and the number of Third Places. Data on associations comes from the 2000 County Business Patterns. Associations include organization involved in religious, grantmaking, civic, professional, business, labor union, and political activities (North American Industry Classification System Industry Code 813). I measure associations as the number of organizations in the MSA/CMSA/PMSA per 1,000 persons.

Third Places refer to retail and other establishments that provide an area for interaction among community residents. Third places are identified as businesses in the following North American Industry Classification system categories: hair, nail, and skin care services (81211/), pharmacies and drug stores (44611/), drinking places (7224//), full service restaurants (7221//), limited service eating (7222//), cafeterias (722212), and grocery stores (445110). Data on Third Places come from the 2000 County Business Patterns. Like associations, Third Places foster civic engagement, but do so by serving as an institutional space for informal social relations (Oldenburg 1991). I measure third places as the number of third places in the each MSA/CMSA/PMSA per 1,000 persons.

The presence of associations and third places increases social interaction and networking within communities, and creates an environment that encourages social integration among the residents who live there. These types of organizations help to develop the "horizontal ties" between community members and increase the level of social cohesion among residents (Putman 1993). The integrative function of these locally oriented institutions and organizations has been shown to increase community well-being and offset the negative effects income inequality (Tolbert et al. 1998). Such research indicates that the social integration of residents increases

their ability to problem solve and address local issues, which may ultimately mediate the negative effects of inequality on individual health (Marmot & Wilkinson 2001).

Health Infrastructure

Two contextual-level measures of local infrastructure are included in this analysis: the number of Physicians, and the number of hospital beds, per 1000 residents in 2000. *Physicians* refer to the number of medical doctors who are working in in any medical field. *Hospital Beds* refer to all inpatient beds available in public, private, general, and specialized hospitals and rehabilitations centers, and can be used to indicate the availability of inpatient services (World Development Indicators Database).

Consistent with prior research, I adopt these variables as measures for local investment in public health infrastructure. Public and private expenditure on material resources—such as doctors and hospital beds—are indicative of local government's ability to meet the health-related needs of residents in the community (Area Resource File, 2009).The availability of health care materials and personnel increases the likelihood that individuals will receive both preventative and reactive health care; therefore, investment in this type of infrastructure may help to offset the negative health consequences experienced by individuals living in areas of high income-inequality (Smith 1996; Lynch & Kaplan 1997; Kaplan et al. 1996; Lynch et al. 1998).

Control Variables

This analysis also includes several control variables—at both the individual and contextual levels—to rule out spurious co-variation. Individual-level controls are taken from the IHIS and include demographic information (*age, sex, race, ethnicity,* and *self-reported health status*), as well as social and economic indicators (*region of residence*, *employment status*, *family income*,

educational attainment, and *marital status*). Contextual-level controls are taken from the 2000 Census of Population and Housing, Summary File 3, and include measures for *minority population*, *absolute income*, and *population size*. Below, I describe how each control variable is operationalized, and then briefly describe its significance to this particular study.

Individual-Level Control Variables

Age is a continuous variable, reporting the respondent's age in years since his or her last birthday (18-99+). Across all social groups, there is a general pattern of mortality by age, with death rates declining throughout infancy and early teen years, and increasing more rapidly as individuals reach elderly status (Heligman and Pollard 1980; Olshansky and Carnes 1997). Ignoring age in an examination of the individual probability of mortality would introduce severe bias; therefore, it is included in all analyses as a basic control. Due to confidentiality concerns, respondents under the age of 18 are excluded from the analysis.

Sex is a binary variable, indicating whether the respondent is male or female. Previous research indicates that there is a sex mortality differential in the United States, with females having a life expectancy an average of 8 years longer than their male counterparts (Peters et al 1998). Although the mortality gap between males and females continues to narrow over time, sex still plays influential role on overall mortality and therefore is included as a control in all analyses.

Race/Ethnicity is the self-reported (or interviewer reported) main racial background of the respondent, combined with his or her classification as having Hispanic/Spanish/Latino origin or ancestry. In this analysis, I distinguish between non-Hispanic Blacks, non-Hispanic Whites, other non-Hispanics, and Hispanics. The link between race/ethnicity and overall mortality is well documented, with White Americans typically enjoying longer life expectancies, better

health, and lower mortality rates than other racial minorities, particularly African Americans (Otten et al. 1990; Rogers 1992; Sorlie et al. 1992). In addition, previous research demonstrates a strong—albeit perplexing—relationship between health and ethnicity, with Cubans, Mexican Americans, and Other Hispanics experiencing lower age-adjusted mortality rates than Caucasian Americans (Anderson et al. 1997). To address concern over the influence of race or ethnicity on individual mortality outcomes, I include measures for both in this analysis.

Region is a binary variable indicating which region of the U.S the housing unit containing the respondent was located. Previous research indicates that there are regional disparities in a variety of health outcomes, with residents in the Southern United States typically experiencing higher mortality rates than residents living in other areas of the United States (Phelps 1997). To control for the influence of regional location, I include a variable that accounts for whether the respondent lives in the South or Non-South United States. [1]

Employment Status indicates whether a respondent was employed (a part of the labor force or actively seeking work), unemployed, or not a member of the labor force, during the previous 1 to 2 weeks. Whether or not an individual is employed can significantly impact on his or her life expectancy (Rogot, Sorlie, and Johnson 1992). Individuals experience a host of benefits from their jobs (including salary, possible access to health insurance, and social relations) of which the underemployed or unemployed may lack access. Research shows that those individuals who are not employed or not in the labor force generally experience higher mortality than their working counterparts (Rogers, Hummer, Nam 2000).

[1] States included in the South/Non-South division correspond to the U.S regions recognized by the U.S Census Bureau. South: Delaware, Maryland, District of Columbia, Virginia, West Virginia, North Carolina, South Carolina, Georgia, Florida, Kentucky, Tennessee, Mississippi, Alabama, Texas, Arkansas, Oklahoma, Louisiana.

Family Income is an interval variable indicating the respondent's total combined family income during the past 12 months. Family income is defined as any money income received from jobs, retirement income, or social security, as well as unemployment payments and money from other types of public assistance. Family income is commonly adopted in research related to health, and is found to be consistently related to mortality and other health outcomes for individuals. In addition, family income is considered the most accurate indication of an individual's economic status, as it represents not only a respondent's individual income, but access to income collected by other family members from whom they are most likely sharing and benefitting. Individuals are often hesitant to report their incomes. In cases where individuals had a nonresponse to questions regarding income, I imputed family income based on regression models of age, sex, and race/ethnicity, region of residence, employment status, educational attainment, marital status, and self-reported health. This imputation allowed me to preserve a majority of cases that otherwise would have had to have been dropped from the analysis.

Educational Attainment is an interval variable indicating the respondent's highest grade of school or year of college completed, in intervals. In this analysis, I distinguish between those respondents who have less than a high school education (0-11 years), those who are high school graduates (12 years), and those who have more than high school education (13+ years). There is a well-documented inverse-gradient relationship between education level and mortality, with individuals receiving health benefits from each additional year of education (Adler et al. 1994). To address variation in my sample on educational attainment, and its potential influence on individual mortality, educational attainment is includes as a basic control variable.

Marital Status indicates the respondent's legal marital status, including whether he or she was currently married, divorced, widowed, or never married. Studies show that individuals who are married experience a lower mortality risk than unmarried individuals and are more likely to engage in positive health behaviors (Lillard and Waite 1995).

Self-reported health is a binary variable indicating the respondent's self-reported general health at the time of the first interview. A respondent's underlying health condition at the beginning of the survey period may subsequently influence the likelihood that he or she will die during the follow up period; therefore, it is necessary to control for baseline health. An individual reporting their health as being excellent, very good, or good were categorized as "healthy"; in contrast, individuals reporting their health as fair or poor were categorized as "unhealthy."

Contextual-Level Control Variables

Minority population is measured using two variables: the percentage of the population that is non-Hispanic Black and the percentage of the population that is Hispanic. Previous research indicates that higher minority racial concentration is significantly related to higher mortality (LaVeist, 1992; McLaughlin and Stokes, 2002). By including *Percent Black* and *Percent Hispanic* in the current analysis, I control for such effects.

Absolute Income is operationalized as total household income per person. The economic structure of a community is both directly and indirectly related to the health of its citizens, as it may facilitate the consumption of goods and services that influence health, including food, housing, education, and access to medical care (Preston 2007). I include a measure for absolute income which (in addition to the measure for *Income Inequality*) controls for the influence of economic structure on health.

Population Size is a measure for the natural logarithm of the MSA, CMSA, or PMSA population size. Population size serves as a measure for degree of urbanization, which has been linked to changes in living standards and social behaviors which may influence population health. Although living in an urban environment may increase access to better health care, it may also "concentrate health risks and introduce new hazards" (Bulletin of World Health Organization, 2010).

DATA PREPARATION & ANALYSIS

This dissertation uses discrete-time hazards modeling to determine the influence of contextual-level variables on the likelihood of individual mortality, while individual-level characteristics are held constant. Discrete-time hazards models are appropriate for data that includes a *risk set* and a specified *hazard* of interest to the researcher (Allison 1984). In this analysis, the risk set refers to the 148,120 individuals "at risk" of dying during the follow-up intervals after their initial interview. The hazard or "event of interest" is the death that may or may not occur of each individual within the risk set. This type of analytical technique allows individual mortality risk to be assessed within the framework of several explanatory variables, and for comparisons to be made between individuals who died during the follow-up period versus those who "survived" the entire duration of the interview period (Allison 1984; Rogers, Hummer, & Nam 2000).

Data are prepared for this type of analysis by first constructing a person-year file, and then merging that person-year file set with contextual-level data. A person-year file was generated by creating a separate record for every year a person in the sample "survived" until the year of their death, or before the end of the follow-up period, whichever came first. The number of years a person contributes to the person-year file will vary, depending on when and if they die

during the follow-up period. The time of year the initial interview of each NHIS respondent takes place will also vary, as interviewers work continuously throughout the year. To address this inconsistency and keep the person-year data as accurate as possible, all participants are considered "at risk of dying" for one-half year in addition to each subsequent full year until their death or until the end of the follow-u period (Rogers, Hummer, & Nam 2000). For example, persons who participated in the 1997 NHIS and survived to end of the follow-up period in 2000, each contribute 3.5 years. Similarly, persons interviewed in 1997 who died in 1998 contribute just 1.5 years. This type of data transformation—where individual person- records are transposed to create a person-year file—is routine in studies of mortality and particularly useful in cases where individuals are interviewed and then followed over time (Rogers et al. 1996). The person year data file in this analysis includes 1,098,839 person-year records for the 148,120 persons in the sample. An example of a person-year data file can be seen in Appendix B.

Once the person-year file was complete, it was merged with contextual information that corresponded to the 58 MSAs, PMSAs, or CMSAs within which each respondent resides. Linking the individual- and contextual-level data provides information about the type of environment each respondent lives in, and allows for an examination of the influence of environmental characteristics on the probability of each respondent's mortality. Independent and control variables from the U.S Census Summary File 3 are available at the MSA, CMSA, and PMSA level and are directly merged with the person-year file by each respondent's personal NHIS identification code. Key variables from the Census Bureau, however, are only available at the county-level. These county data were first aggregated up from the county to the corresponding MSA, PMSA, or CMSA in which they are located, and then merged to the person-year file by the NHIS person identification code.

The discrete-time hazard modeling used in this analysis is accomplished using the SURVEYLOGISTIC specification in SAS. There are three reasons survey logistic regression was chosen as the appropriate method of analysis for this dissertation. First, the survey logistic regression procedure is designed especially for logistic regression with survey data collected using a multi-stage sampling design that may include a variety of different methods—including stratification, clustering, and unequal weighting techniques—to identify a sample population. The SURVEYLOGISTIC command incorporates the complex sampling design of NHIS into this analysis and allows for the statistically accurate estimation of mortality models that compare individuals who died during the follow-up period to those individuals who survived the entire duration of the follow-up period (Rogers, Hummer, & Nam 2000).

Second, the survey logistic regression command produces results comparable to the statistical techniques used in other studies of mortality. For example, several studies have used continuous-time (Cox Proportional) hazard modeling where survival time is measured from date of initial interview to the date of death, rather than in yearly intervals (e.g. LeClere et al., 1998). Although this type of statistical technique is commonplace in "survival" or "event-history" analyses, discrete-time hazard modeling has been shown to consistently yield results that are similar to those produced in continuous-time analyses (Rogers, Hummer, and Nam 2000). Allison (1984:22) notes the equivalency of results produced by continuous-time and discrete-time hazard models, and argues that the decision to use one method over the other "should generally be made on the basis of computational cost and convenience." For convenience purposes, all results in this dissertation are produced using discrete-time hazard modeling.

And third, most public health research reports findings in terms of odds-ratios; that is, the odds of a specified event (e.g. mortality) occurring in one group (e.g. males) versus the odds of it

occurring in another group (e.g. females). The SURVEYLOGISTIC command fits logistic regression models for discrete responses, which can then be reported in the form of odds ratios by exponentiating the regression coefficient produced by the discrete-time hazard models (Hosmer and Lemeshow 1989, Rogers, Hummer, & Nam 2000). Given the convenience and accuracy of this type of analysis, as well as the familiarity of most readers with interpretation of logistic regression models, all results are reported in terms of odd ratios of death. Individuals who died during the follow-up period are coded one (Mortality =1) and individuals who survive the duration of the follow-up period are coded zero (Mortality = 0). Using this coding scheme, odds ratios above 1 can be interpreted as indicating a higher risk of death for that particular category, while an odds ration below 1 signifies a reduced risk of mortality (Rogers, Hummer, & Nam 2000).

PRELIMINARY ANALYSES

Descriptive Statistics

Tables 1 and 2 list the descriptive characteristics for all individual- and contextual-level variables included in the analyses. Table 1 presents the means and standard deviations for all individuals included in the sample. Of notable interest is the small proportion of sample individuals who died during the follow-up up period (Mortality =1). Of all individual IHIS respondents included in the present analysis, less than 1% had a final mortality status indicative of a death occurring between the survey period lasting from 1997-2000. Although this is a relatively small proportion of individuals relative to the overall sample size, it totals 9,475 individuals which allows for comprehensive investigation of variance in individual mortality.

The descriptive statistics for all contextual-level variables are reported in Table 2. Income inequality (Gini coefficient) across all 58 of the MSAs, CMSAs, and PMSAs included in

the sample ranges from a low of 37.96 to a high of 53.47, with an average of 43.78 (SD=3.24). With regard to other key predictor variables in this analysis, the mean number of associations per 1000 people is 0.87 (SD=0.23) and the mean number of Third Places per 1,000 people is 2.25 (SD =0.24). These variables serve as a measure of social cohesion and range from 0.50 – 1.44 and 1.65 – 3.52, respectively, across all MSAs, CMSAs, and PMSAs included in the sample. The mean number of Physicians per 1,000 people is 3.30 (SD =1.70) and the mean number of Hospital Beds per 1,000 people is 3.27 (SD=0.98). These variables serve as measures for local investment in public infrastructure and range from 1.55 – 21.00 and 2.09 – 9.62, respectively, across all MSAs, CMSAs, and PMSAs included in the sample.

Bivariate Analyses

Tables 3 and 4 present results from bivariate analyses between the dependent variable in this dissertation, individual mortality, and the key predictor variables of interest. The purpose of bivariate analyses is to explore three questions connected to the hypotheses being tested in this dissertation. First, what is the relationship between income inequality and individual mortality? The link between income distribution and individual mortality is well established within the literature. However, very few studies have examined this relationship at lower levels of aggregation. This portion of the bivariate analysis examines the inequality-mortality relationship at the MSA/CMSA/PMSA level, and is an important first step before introducing variables into the analysis that may mediate this relationship.

Table 1. Descriptive Statistics: Individual-Level Variables	
	Mean (Std. Dev.)
Outcome Variable	
Mortality (% Died)	00.86
Individual's Characteristics	
Age	46.69 (16.52)
Sex	
% Female	53.22
% Male	46.78
Race	
% non-Hispanic White	75.04
% non-Hispanic Black	15.30
% non-Hispanic Other	09.65
% Hispanic	22.79
Region	
% South	33.93
% Non-South	66.06
Employment Status	
% Employed	68.69
% Unemployed	02.32
% Not in Labor Force	28.99
Family Income	44.41 (22.16)
Educational Attainment	
% < 12 years	17.76
% 12 years	30.45
% 12+ years	51.79
Marital Status	
% Married	57.81
% Divorced	12.57
% Widowed	05.70
% Never Married	23.92
Self-reported Health	
% Healthy (Excellent/Very Good/Good Health)	90.24
% Not Healthy (Fair/Poor Health)	09.96
Notes: Individual data obtained from 1997-2000 IHIS (N= 1,098,839)	

Table 2. Descriptive Statistics: Contextual-Level Variables	
	Mean (Std. Dev.)
Key Explanatory Variables	
Average Income Inequality (Gini Coefficient)	43.78 (3.24)
Social Cohesion	
# Associations per 1,000 persons	0.87 (0.23)
# Third Places per 1,000 persons	2.25 (0.24)
Public Health Infrastructure	
# Physicians per 1,000 persons	3.30 (1.70)
# Hospital Beds per 1,000 persons	3.27 (0.98)
MSA/PMSA/CMSA Characteristics	
Minority population	
Percent Black	13.79 (8.18)
Percent Hispanic	17.30 (15.58)
Per Capita Income	23.42 (3.04)
Ln Population Size	6.50 (0.31)

Notes:
MSA data constructed from 3 sources:
2000 Census of Population and Housing
2000 Census USA County Business Patterns File
2000 Census American Community Survey.
(N=58)

Second, what is the relationship between the level of social cohesion and individual mortality? The primary purpose of this dissertation is to investigate the manner in certain contextual-level characteristics may buffer the effects of income inequality on individual mortality. This portion of the bivariate analysis will provide basic insight into the potentially mediating properties of community social cohesion in the relationship between income inequality and the probability of individual mortality. And third, what is the relationship between investment in local infrastructure and individual mortality? As with the previous question, the importance of testing this association has important implications regarding the mediate properties of variables related to local infrastructure in the inequality-mortality relationship.

Table 3 reports the bivariate correlations between the probability of individual mortality and all individual-level control variables. With the exceptions of region of residence and having a marital status of "divorced," all individual-level control variables are highly significant and in the predicted direction. These results indicate that—at least at a bivariate level—the characteristics of individuals, including their age, sex, race, employment status, family income, educational attainment, marital status and self-reported health, play an important role in determining the probability that they die during the follow-up period.

Table 4 reports the bivariate correlations between the probability of individual mortality and all contextual-level variables, including both key explanatory variables and controls. With regards to average income inequality, the Gini coefficient is positively correlated with individual mortality. This finding is expected and consistent with the existing literature, which demonstrates a significant, positive association between the probability of individual mortality and higher levels of income inequality.

Bivariate correlations between the measures for social cohesion and measures for public health infrastructure also conform to expectations grounded in previous research. With the exception of the number of association per 1,000 persons, all measures for social cohesion and local infrastructure exhibit significant, negative correlations with the probability of individual mortality. These findings indicate that, at least at a bivariate level, the number of third places per 1,000 people, the number of physicians per 1,000 people, and the number of hospital beds per 1,000 people is associated with a reduced probability of individual mortality. In addition, such associations demonstrate the potential for community social cohesion and local infrastructure to mediate the relationship between income inequality and individual mortality.

SUMMARY OF PRELIMINARY ANALYSES

The descriptive and bivariate analyses discussed in this section provide basic insight into the relationships between the probability of individual mortality and the key explanatory variables of interest in this dissertation: income inequality, social cohesion, and local infrastructure.
In addition, these exploratory analyses demonstrate the interrelationships between the individual mortality and several important individual- and contextual-level control variables.

With consideration of the fact that these are bivariate analyses and that any findings presented here may be tenuous, the preliminary results have important implications regarding the three questions posed at the beginning of this section. First, income inequality does appear to be associated with the probability of individual mortality. This association holds regardless of whether income inequality is measured as a continuous variable, or whether it is broken down into quartiles indicating lower to higher levels of income inequality. In addition, these bivariate analyses indicate that the relationship between inequality and mortality may only operate at the higher levels of income inequality.

Table 3. Pearson Correlation Coefficient Between Mortality and Individual-Level Variables

Individual's Characteristics	
Age	0.103***
Sex	
Female	-0.004***
Race	
non-Hispanic White	0.005***
non-Hispanic Black	0.002*
non-Hispanic Other	-0.010***
Hispanic	-0.011***
Region	
South	-0.000
Employment Status	
Employed	-0.071***
Unemployed	-0.006***
Not in Labor Force	0.074***
Family Income	-0.041***
Educational Attainment	
% < 12 years	0.031***
% 12 years	0.006***
% 12+ years	-0.028***
Marital Status	
Married	-0.012***
Divorced	0.00
Widowed	0.067***
Never Married	-0.023***
Self-reported Health	
Healthy (Excellent/Very Good/Good Health)	-0.065***
Not Healthy (Fair/Poor Health)	

Notes:
^p=.10 *p < .05 **p<.01 ***p<.001
Bivariate analyses include survey weights

Table 4. Pearson Correlation Coefficients Between Mortality, Key Explanatory Variables, and Contextual-Level Control Variables

Key Explanatory Variables	
Average Income Inequality (Gini Coefficient)	0.003***
Social Cohesion	
# Associations per 1,000 persons	0.000
# Third Places per 1,000 persons	-0.003**
Public Health Infrastructure	
# Physicians per 1,000 persons	-0.0002^
# Hospital Beds per 1,000 persons	-0.007***
Contextual-Level Controls	
Minority population	
Percent Black	0.002*
Percent Hispanic	-0.003**
Per Capita Income	-0.005***
Ln Population Size	-0.003**

Notes:
^p = .10 *p < .05 **p<.01 ***p<.001
Bivariate analyses include survey weights

Second, the level of social cohesion in a community—as indicated by the number of third places per 1,000 persons—is associated with the probability of individual mortality. The bivariate analyses regarding the third place variable indicate that a significant, negative relationship exists between social cohesion and the probability of an individual dying during the follow up period. Although this finding is indicative of a direct effect, it suggests that the examination of social cohesion as a mediating variable in the inequality-mortality relationship is worth pursuing. And third, the level of investment in public health infrastructure—as indicated by the number of physicians per 1,000 persons and the number of hospital beds per 1,000 persons—as associated with the probability of individual mortality. Like social cohesion, the measures for the number of physicians and hospital beds indicate a direct, negative association between investment in local infrastructure and the probability of an individual dying during the follow-up period. These significant associations also suggest that a more in-depth analysis of the mediating properties of such variables is worth pursuing.

CHAPTER FIVE: RESULTS

In this chapter, I present the results from baseline and multivariate analyses that examine the influence of both individual- and contextual-level predictors on the risk of individual mortality. The purpose of this analysis is two-fold: One, to explore and establish the relationship between income inequality and the probability of individual mortality, controlling for a variety of contextual and individual characteristics. And two, to explore the possible pathways through which income inequality may operate on the probability of individual mortality. Previous research on the inequality-health relationship has shown support for the Income Inequality Hypothesis (IIH), which is grounded in the assumption that an unequal income distribution can have detrimental effects for individual's health, above and beyond absolute income or individual income. This chapter will present in-depth analyses of the inequality-health relationship, using all-cause mortality as the outcome variable. Scholars have recently proposed two mechanisms through which income inequality may lead to poor health for individuals: the *social cohesion mechanism* and the *local investment mechanism*. In this chapter, I explore the potentially mediating properties of each of these mechanisms within the inequality-mortality relationship.

The analyses presented in this chapter are carried out in three steps. In step 1, I explore the interrelationships between individual risk of mortality and all individual- and contextual-level control variables. In step 2, I present baseline and multivariate models examining the inequality-health relationship, controlling for a variety of individual and contextual characteristics. Finally, in step 3, I explore the mediating properties of variables related to the *social cohesion mechanism* and *local investment mechanism* in the inequality-health relationship.

CONTROL MODELS

All analyses in this dissertation include several individual- and contextual-level control variables. Before testing hypotheses related to association between income inequality and mortality—and the mechanisms that may potentially mediate this relationship—I examined the relationship between all control variables and my dependent variable, all-cause mortality. I limit my discussion of most control variables to this section of the paper, and then return to any key findings regarding these variables in the conclusion section.

Table 5 presents the survey logistic regression results for the effects of all individual and contextual control variables on the probability of individual mortality. Model 1, which controls for only the contextual characteristics of each MSA/CMSA/PMSA, shows that both minority population and per capita income are associated with the probability of individual mortality. This model demonstrates a positive association between the percent of the population that is Black and the odds of individual mortality ($p < .10$), a negative association between the percent of the population that is Hispanic and the odds of individual mortality ($p < .01$), and a negative association between per capita income and the odds of individual mortality ($p < .001$). With the exception of Percent Black, these contextual-level effects remain significant, even after the addition of individual-level controls (Model 3).

Models 2 and 3 demonstrate the effects of individual-level controls on individual mortality, with and without contextual-level controls. The significant influence of individual-level control remains consistent across these two models, indicating that the basic demographic, social, and economic characteristics of individuals play a large role in the probability of their mortality, regardless of the social and economic characteristics of MSAs, CMSAs, and PMSAs in which they reside.

Table 5: Survey Logistic Regression Predicting the Relationship Between Individual and Contextual Control Variables on Individual Risk of Mortality

	Model 1	Model 2	Model 3
Individual-Level Controls			
Age		1.08***	1.08***
Sex (Female =1)		0.56***	0.56***
Race			
White (Contrast)		------	------
Black		1.09**	1.10**
Other		0.70***	0.71***
Hispanic		0.80***	0.82***
Region			
(South =1)		1.06**	1.07**
Employment Status			
Employed (Contrast)		------	------
Unemployed		1.40**	1.40**
Not in Labor Force		1.74***	1.74***
Family Income		0.996***	0.996***
Educational Attainment			
< 12 years		0.99	0.99
12 years (Contrast)		------	------
12+ years		0.86***	0.86***
Marital Status			
Married (Contrast)		------	------
Divorced		1.34***	1.35***
Widowed		1.21***	1.21***
Never Married		1.60***	1.61***
Self-reported Health (Healthy=1)		0.44***	0.43***
Contextual-Level Controls			
Minority population			
Percent Black	1.00^		1.00
Percent Hispanic	0.99**		0.99*
Per Capita Income	0.96***		0.99*
Ln Population Size	0.95		1.01
Adjusted R^2	0.00	0.20	0.20
-2*Log-likelihood	60825.162	48710.856	48706.923

Reported Figures are Odds Ratios
^ p <.10 *p < .05 **p<.01 ***p<.001
(N= 1,098,839)

The results of Model 3 indicate that there are significant mortality differentials that exist between individuals based on demographic characteristics such as sex, race, ethnicity, and baseline health. As expected, there is a significant mortality gap between men and women, with women exhibiting 44% lower odds of mortality than men during the follow-up period. Compared to whites, Black individuals displayed more than 10% higher odds of dying during the follow-up period, while individuals falling into the broad racial category of "other" were at a reduced risk of dying compared to whites. In addition to race, ethnicity also appears to play a role regarding the probability of an individual dying during the follow-up period. The odds of mortality for those individuals having Hispanic origin are 18% lower than for non-Hispanics. Likewise, individuals who categorized themselves as "healthy" during their initial survey interview had 57% lower odds of dying during the follow-up period than participants whose self-reported health status during their first interview was "unhealthy."

The important influence of socioeconomic variables on the probability of individual mortality is also apparent in Model 3. Controlling for all other factors, the odds of mortality for individuals who were unemployed or not in the labor force was 40% and 74% higher, respectively, than for individuals who were employed. Although there was not a significant difference in the odds of dying for individuals with less than 12 years of education compared to those with a high school degree, there was a significant protective effect for individuals who educational attainment went beyond high school. Individuals with 12+ years of education experienced 14% lower odds of dying during the follow-up period, compared to those with only 12 years of schooling. Family income is also shown to have a significant negative association with probability of mortality, within individuals reporting higher family incomes being more likely to survive the follow up period than individuals reporting lower family incomes.

In addition to the demographic and socioeconomic variables discussed above, marital status demonstrated significant effects with regard to mortality risk. Compared to individuals who were married, individuals who were divorced, widowed, or never married were at a higher risk of dying during the follow-up period. The effect of marital status is especially strong for individuals who reported never being married; compared to married individuals, the odds of a individuals who had never been married dying increased by 61 percent. Region also appears to have significant influence over final mortality status, with individual living in the Southern United States experiencing over 7% odds of mortality than those living in the non-South.

In summary, significant mortality gaps exist between individuals, depending on their various demographic, social, and economic characteristics. All findings regarding the control variables are consistent with previous literature, including the protective effects of being female, white, of Hispanic origin, employed, married, having an education beyond high school, living in the non-South, and having a high family income. Although these individual-level disparities in mortality are not of key interest in this particular study, the significance of such variables indicates the importance of including them as controls in all subsequent analyses conducted in this dissertation.

INCOME INEQUALITY AND MORTALITY

In this section of the analysis, I present and discuss the findings from survey logistic regression analyses that test the following hypothesis:

> H1: Income inequality will be positively related to an individual's risk of mortality, even after controlling for family income.

Table 6 presents the results of survey logistic regression predicting the relationship between income inequality and individual risk of mortality. To ensure that the results of these analyses

are robust to contextual and individual controls, I enter each set of controls incrementally and note their effects on the key variables of interest: average income inequality and individual risk of mortality. Models 1 and 2 serve as a baseline models and examine the influence of average income inequality on individual risk of mortality, without including any of the individual-level variables that control for the demographic, social, or economic characteristics of respondents. Models 3, 4, and 5 examine the influence of average income inequality on individual risk of mortality, while controlling for the demographic and/or economic characteristics of respondents, as well as contextual controls. And finally, models 6 and 7 examine the inequality-mortality relationship after the inclusion of a wide range of individual and/or contextual level controls

There are two important findings regarding the baseline models (Model 1 and Model 2) in Table 6. First, income inequality is not significantly related to the probability of individual mortality until contextual-level controls are introduced in Model 2. This finding is unexpected, since preliminary analyses did indicate a significant, positive bivariate correlation between the Gini Coefficient and individual mortality. However, a causal relationship between these two variables is not supported using survey logistic regression. In Model 2, income inequality is significantly associated with the probability of individual mortality in the expected direction, after controlling for the economic structure (per capita income) and population characteristics (minority population , population size) of the MSAs, CMSAs, and PMSAs in which residents live. The odds ratio for the Gini coefficient in this model indicates that the odds of individual mortality during the follow-up period increased by 3.7% for every standard deviation increase in income inequality.

Second, three of the four contextual-level controls introduced in Model 2 do show significant associations with individual mortality, and in the expected directions. In line with the

control models (Table 5), the absolute economic wellbeing of each MSA/CMSA/PMSA is associated with the likelihood of individual mortality in the expected direction. The odds ratio for per capita income (0.958) is indicative of a small, yet significant negative association between absolute income and the probability of individual mortality, even when a measure for income distribution is included in the model.

The association between minority population and individual risk of mortality changes slightly after including a measure of income inequality into the model. Without the Gini Coefficient, both percent Black and percent Hispanic demonstrated significant negative associations, ($p < .10$) and ($p < .01$), respectively, with the probability of individual mortality (see Table 5, model 1). Once the measure for income inequality was introduced into the analysis, however, percent Black was reduced to insignificance. Percent Hispanic does remain significant, indicating that individuals living in MSAs, CMSAs, or PMSAs characterized by a higher Hispanic minority population had reduced odds of dying during the following up period, even when income distribution is considered.

Table 6: Survey Logistic Regression Predicting the Relationship Between Income Inequality and Individual Risk of Mortality

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Key Explanatory Variables							
Average Income Inequality (Gini Coefficient)	0.999	1.037***	1.017*	1.005	0.999	0.993	0.996
Individual-Level Controls							
Age				1.098***	1.090***	1.080***	1.080***
Sex (Female =1)				0.660***	0.602***	0.565***	0.565***
Race							
White (Contrast)				------	------	------	------
Black				1.514***	1.251***	1.102**	1.102**
Other				0.781***	0.743***	0.713***	0.718***
Hispanic				0.035***	0.869***	0.823***	0.827***
Region							
(South =1)						1.071**	1.070**
Employment Status							
Employed (Contrast)						------	------
Unemployed						1.403**	1.404**
Not in Labor Force						1.742***	1.743***
Family Income			0.971***		0.985***	0.996***	0.996***
Educational Attainment							
< 12 years						0.992	0.991
12 years (Contrast)						------	------
12+ years						0.863***	0.864***
Marital Status							
Married (Contrast)						------	------
Divorced						1.348***	1.350***
Widowed						1.212***	1.212***
Never Married						1.614***	1.615***
Self-reported Health (Healthy=1)						0.439***	0.439***
Contextual-Level Controls							
Minority population							
Percent Black		0.998	0.999	1.001	1.003		1.00
Percent Hispanic		0.990***	0.991***	0.997^	0.998^		0.999^
Per Capita Income		0.958***	0.990^	0.980^	0.994^		0.992^
Ln Population Size		0.904^	0.921	0.998	1.002		1.020
Adjusted R^2	0.0000	0.0012	0.0324	0.1836	0.1894	0.2043	0.2044
-2*Log-likelihood	60875.394	60804.38	58954.04	49951.03	49605.09	48708.81	48706.70

Reported Figures are Odds Ratios
^ p <.10 *p < .05 **p<.01 ***p<.001
(N= 1,098,839)

Models 3, 4, and 5 in Table 6 examine the inequality-mortality relationship after the inclusion of controls for important demographic and economic variables related to individual respondents. Model 3 examines the influence of average income inequality on individual risk of mortality, controlling for family income. Model 4 examines the influence of average income inequality on individual risk of mortality, controlling for important demographic characteristics of individuals, including age, sex, race, and ethnicity. Model 5 examines the influence on average income inequality on individual risk of mortality after controlling for the age, sex, race, and ethnicity, as well as family income. Models 3, 4, and 5 all include contextual-level controls for economic structure and population characteristics, as introduced in Model 2.

Models 3, 4, and 5 demonstrate that the demographic and economic characteristics of individuals are core predictors of individual health, and are highly influential in explaining the relationship between income inequality and individual mortality. Across all three of these models, there are significant mortality gaps between individuals based on age, sex, race, ethnicity, and family income. As was seen in the control models presented in the previous chapter, models 3, 4, and 5 demonstrate that the odds of an individual dying are significantly less for individuals who are female, white, who are of Hispanic origin, and/or who have higher family incomes.

Findings related to the demographic and economic characteristics of individuals are particularly relevant to Hypothesis 1, which predicts that income distribution will be positively associated with the probability of individual mortality, even after controlling for family income. Comparing model 2 to model 3 in Table 6, results for the Gini coefficient indicate that the inclusion of a control for family income does dampen the effects of income inequality on the probability of individual mortality, but that income inequality continues to have a significant,

positive association with the odds of individual mortality (odds ratio = 1.017, $p < .05$). Although the findings in model 3 indicate initial support for Hypothesis 1, the results of model 4 demonstrate that the inequality-mortality relationship is not robust to basic demographic controls. After the inclusion of individual-level controls for age, sex, race, and ethnicity, the Gini coefficient is no longer a significant predictor of the probability of individual mortality. With regard to the effects of contextual-level controls, model 4 and 5 show that the effects of variables related to absolute economic well-being and population characteristics are also reduced after the introduction of individual-level variables.

Models 6 and 7 in Table 6 examine the inequality-mortality relationship after the inclusion of all individual- and contextual-level controls. Model 6 examines the influence of average income inequality on individual risk of mortality, controlling for all demographic, economic, and social characteristics of respondents (age, sex, race, ethnicity, region of residence, employment status, family income, educational attainment, marital status, and self-reported health). And finally, Model 7 is the full model which examines the relationship between income inequality and the probability of individual mortality, while controlling for individual-level characteristics of respondents as well as the contextual-level characteristics of the MSAs, CMSAs, and PMSAs in which they reside.

The findings in models 6 and 7 are consistent with the results from other models in Table 6. As was seen in models 4 and 5, once the demographic characteristics of individuals are taken into account, the Gini coefficient is not shown to be a significant predictor of individual mortality. Likewise, the majority of individual-level controls related to the economic and social characteristics of individuals demonstrate significant effects on the probability that an individual dies during the follow-up period. In terms of directionality, size of effect, and significance, the

results of the individual-level measures remain consist with the control models presented in Table 5.

INCOME INEQUALITY, MORTALITY, AND SOCIAL COHESION

In this section of the analysis, I present and discuss the findings from survey logistic regression analyses that test the following hypothesis:

> H2: The positive effect of income inequality on an individual's risk of mortality will be reduced or brought to insignificance when contextual characteristics pertaining to community social cohesion are taken into consideration.

Table 7 presents the results of survey logistic regression predicting the relationship between income inequality and individual risk of mortality, with and without the inclusion of variables that measure social cohesion. Model 1 serves as a baseline analysis and examines the influence of average income inequality on the probability of individual mortality, controlling for contextual-level characteristics (minority population , per capita income, and population size) as well as individual-level characteristics of respondents (age, sex, race, ethnicity, region of residence, employment status, family income, educational attainment, marital status, and self-reported health). Models 2 and 3 also examine the influence of average income inequality on individual risk of mortality; however, in addition to the individual- and contextual-level control variables included in Model 1, these models also include variables that measure the level of social cohesion with each MSA, CMSA, or PMSA.

During preliminary bivariate analyses, the two variables that measure social cohesion (# of associations and # of third places per 1,000 persons) were shown to have a highly significant positive correlation (Pearson Correlation Coefficient 0.335, $p = <.001$). To avoid any issues with

multicollinearity, and to have a more accurate understanding of the effects of these two key explanatory variables, they were analyzed independently of each other.

In Model 1 of Table 7, I examine the relationship between average income inequality and the probability that an individual dies during the follow-up period, while controlling for the effects of respondent and MSA, CMSA, and PMSA characteristics. This model is included in Table 7 to serve as a baseline analysis of the inequality-mortality relationship, so that the mediating effects of the social cohesion measures may be tested in Models 2 and Model 3. As demonstrated in prior models, however, the effect of average income inequality (as measured by the Gini coefficient) is not significant after the inclusion of individual-level controls. Again, this finding is unexpected and inconsistent with prior research demonstrating that an unequal income distribution will negatively influence individual health outcomes, even after controlling for individual-level characteristics.

Models 2 and 3 examine the relationship between income inequality and the probability of an individual dying during the follow-up period, after the inclusion of variables measuring the level of social cohesion in the MSAs, CMSAs, or PMSAs in which respondent's reside. In addition to all individual-level and control variables, Model 2 includes a measure for the number of association per 1,000 persons, a variable that accounts for the presence of formal organizations and membership societies within the specified area.

Table 7: Survey Logistic Regression Predicting the Relationship Between Income Inequality and Individual Risk of Mortality, with and without Social Cohesion Variables

	Model 1	Model 2	Model 3
Key Explanatory Variables			
Average Income Inequality (Gini Coefficient)	0.996	0.997	1.007
Social Cohesion			
# Associations per 1,000 persons		0.895^	
# Third Places per 1,000 persons			0.847**
Individual-Level Controls			
Age	1.080***	1.080***	1.080***
Sex (Female =1)	0.565***	0.565***	0.564***
Race			
White (Contrast)	------	------	------
Black	1.102**	1.104**	1.100**
Other	0.718***	0.718***	0.716***
Hispanic	0.827***	0.830***	0.835***
Region			
(South =1)	1.070**	1.078**	1.058**
Employment Status			
Employed (Contrast)	------	------	------
Unemployed	1.404**	1.402**	1.403**
Not in Labor Force	1.743***	1.741***	1.742***
Family Income	0.996***	0.996***	0.996***
Educational Attainment			
< 12 years	0.991	0.991	0.990
12 years (Contrast)	------	------	------
12+ years	0.864***	0.864***	0.862***
Marital Status			
Married (Contrast)	------	------	------
Divorced	1.350***	1.350***	1.351***
Widowed	1.212***	1.212***	1.214***
Never Married	1.615***	1.618***	1.619***
Self-reported Health (Healthy=1)	0.439***	0.439***	0.439***
Contextual-Level Controls			
Minority population			
Percent Black	1.00	1.000	0.999
Percent Hispanic	0.999	0.998^	0.996*
Per Capita Income	0.992^	0.993^	0.991^
Ln Population Size	1.020	1.007	0.998
Adjusted R2	0.2044	0.2044	0.2044
-2* Log-Likelihood	48706.707	48705.314	48702.557
Reported Figures are Odds Ratios			
^ p <.10 *p < .05 **p<.01 ***p<.001			
(N= 1,098,839)			

Similarly, Model 3 includes a measure for the number of Third Places per 1,000 persons, a variable that accounts for the presence of informal retail and social establishments within the specified area. The presence of associations and third places is theorized to promote civic engagement and social integration among residents, and these variables have been adopted in previous research as measures for social cohesion.

In model 2 of Table 7, I estimate the effect of associations in a model that includes all individual- and contextual-level control variables, in addition to the Gini coefficient for average income inequality. The Gini coefficient was not shown to be a significant predictor of individual mortality in Model 1; therefore, the effect of the associations variable as included in Model 2 cannot be interpreted as "mediating" the relationship between income inequality and the probability of individual mortality. The effects of the association variable may, however, be interpreted as having a direct effect on the probability of individual mortality. The measure for associations in Model 2 does show a significant, negative direct effect on the probability of individual mortality (Odds Ratio = 0.895, $p < .10$). This finding suggests that the odds of individual mortality decrease as the number of associations per 1,000 persons increases, and that individuals living in MSAs, CMSAs, or PMSAs characterized by a higher concentration of associations were at a reduced risk of dying during the follow-up period than those respondents who lived in areas with a lower percentage of associations.

In model 3 of Table 7, I estimate the effect of Third Places in a model that includes all individual- and contextual-level control variables, in addition to the Gini coefficient for average income inequality. As with model 2, the effect Third Places can only be interpreted in terms of direct effects, rather than having mediating properties in the association between income inequality and mortality. The measure for Third Places in Model 3 does show a significant,

negative direct effect on the probability of individual mortality (Odds Ratio = 0.847, p <.001). This finding suggests that the odds of individual mortality decrease as the number of third places per 1,000 persons increases, and that individuals living in MSAs, CMSAs, or PMSAs characterized by a higher concentration of third places were at a reduced risk of dying during the follow-up period than those respondents who lived in areas with a lower percentage of associations.

INCOME INEQUALITY, MORTALITY, AND LOCAL INFRASTRUCTURE

In this section of the analysis, I present and discuss the findings from survey logistic regression analyses that test the following hypothesis:

> H3: The positive effect of income inequality on an individual's risk of mortality will be reduced or brought to insignificance when contextual characteristics pertaining to local investment in public health infrastructure are taken into consideration.

Table 8 presents the results of survey logistic regression predicting the relationship between income inequality and individual risk of mortality, with and without the inclusion of variables that measure investment in local health infrastructure. Model 1 serves as a baseline analysis and examines the influence of average income inequality on the probability of individual mortality, controlling for contextual-level characteristics (minority population , per capita income, and population size) as well as individual-level characteristics of respondents (age, sex, race, ethnicity, region of residence, employment status, family income, educational attainment, marital status, and self-reported health). Models 2 and 3 also examine the influence of average income inequality on individual risk of mortality; however, in addition to the individual- and contextual-level control variables included in Model 1, these models also include variables that measure the level of local health infrastructure with each MSA, CMSA, or PMSA.

Table 8: Survey Logistic Regression Predicting the Relationship Between Income Inequality on Individual Risk of Mortality, with and without Local Infrastructure Variables

	Model 1	Model 2	Model 3
Key Explanatory Variables			
Average Income Inequality (Gini Coefficient)	0.996	0.996	0.995
Public Health Infrastructure			
# Physicians per 1,000 persons		1.001	
# Hospital Beds per 1,000 persons			1.008
Individual-Level Controls			
Age	1.080***	1.080***	1.080***
Sex (Female =1)	0.565***	0.565***	0.565***
Race			
White (Contrast)	------	------	------
Black	1.102**	1.102**	1.101**
Other	0.718***	0.718***	0.719***
Hispanic	0.827***	0.826***	0.826***
Region			
(South =1)	1.070**	1.072**	1.076**
Employment Status			
Employed (Contrast)	------	------	------
Unemployed	1.404**	1.404**	1.404**
Not in Labor Force	1.743***	1.743***	1.743***
Family Income	0.996***	0.996***	0.996***
Educational Attainment			
< 12 years	0.991	0.991	0.991
12 years (Contrast)	------	------	------
12+ years	0.864***	0.864***	0.865***
Marital Status			
Married (Contrast)	------	------	------
Divorced	1.350***	1.350***	1.350***
Widowed	1.212***	1.212***	1.212***
Never Married	1.615***	1.615***	1.641***
Self-reported Health (Healthy=1)	0.439***	0.439***	0.439***
Contextual-Level Controls			
Minority population			
Percent Black	1.000	1.000	1.000
Percent Hispanic	0.999	0.999	1.002
Per Capita Income	0.992^	0.991^	0.993^
Ln Population Size	1.020	1.024	1.036
Adjusted R2	0.2044	0.2044	0.2044
-2*Log-likelihood	48706.707	48706.695	48706.496
Reported Figures are Odds Ratios			
^ p <.10 *p < .05 **p<.01 ***p<.001			
(N= 1,098,839)			

During preliminary bivariate analyses, the two variables that measure local health infrastructure (# of physicians per 1,000 persons and # of hospital beds per 1,000 persons) were shown to have a highly significant positive correlation (Pearson Correlation Coefficient 0.708, p = <.001). To avoid any issues with multicollinearity, and to have a more accurate understanding of the effects of these two key explanatory variables, they were analyzed independently of each other.

In Model 1 of Table 8, I examine the relationship between average income inequality and the probability that an individual dies during the follow-up period, while controlling for the effects of respondent and MSA, CMSA, and PMSA characteristics. This model is included in Table 8 to serve as a baseline analysis of the inequality-mortality relationship, so that the mediating effects of the local infrastructure measures may be tested in Models 2 and Model 3. As demonstrated in prior models, however, the effect of average income inequality (as measured by the Gini coefficient) is not significant after the inclusion of individual-level controls. Again, this finding is unexpected and inconsistent with prior research demonstrating that an unequal income distribution will negatively influence individual health outcomes, even after controlling for individual-level characteristics.

Models 2 and 3 examine the relationship between income inequality and the probability of an individual dying during the follow-up period, after the inclusion of variables measuring the level of investment in local infrastructure in the MSAs, CMSAs, or PMSAs in which respondent's reside. In addition to all individual-level and control variables, Model 2 includes a measure for the number of Physicians per 1,000 persons, a variable that accounts for the number of medical doctors who are working in any medical field within the specified area. Similarly, Model 3 includes a measure for the number of hospital beds per 1,000 persons, a variable that

accounts for the presence of inpatient beds available in public, private, general, and specialized hospitals and rehabilitations centers within the specified area. The presence of physicians and hospital beds is indicative of the level of expenditure on health infrastructure within an area, and higher investments is theorized to reduce the negative health outcomes of individuals by providing them with health care personnel and material resources heath care needs.

In model 2 of Table 8, I estimate the effect of Physicians in a model that includes all individual- and contextual-level control variables, in addition to the Gini coefficient for average income inequality. The Gini coefficient was not shown to be a significant predictor of individual mortality in Model 1; therefore, the effect of the Physicians variable as included in Model 2 cannot be interpreted as "mediating" the relationship between income inequality and the probability of individual mortality. The effects of the Physicians variable may, however, be interpreted as having a direct effect on the probability of individual mortality. The measure for physicians in Model 2 is not shown to have a significant direct effect on the probability of individual mortality.

In model 3 of Table 8, I estimate the effect of Hospital Beds in a model that includes all individual- and contextual-level control variables, in addition to the Gini coefficient for average income inequality. As with model 2, the effect Hospital Beds can only be interpreted in terms of direct effects, rather than having mediating properties in the association between income inequality and mortality. The measure for Hospital Beds in Model 3 is not shown to have a significant direct effect on the probability of individual mortality.

SUMMARY OF HYPOTHESIS TESTS

The purpose of the analyses presented in this chapter is to examine the relationship between average income inequality and the probability of individual mortality, and to explore two

possible pathways through which inequality may operate on individual health: 1) the *social cohesion mechanism*, and 2) the *local infrastructure mechanism*. Generally speaking, the baseline and multivariate models produced from these analyses provided no support for the three hypotheses proposed in this dissertation. In chapter 6, I provide a more detailed summary of the findings presented here, as well as discussion of the implications of these findings, limitations of the study, and directions for future research.

CHAPTER SIX: DISCUSSION AND CONCLUSIONS

The aim of this dissertation has been to advance prior research exploring the impact of income inequality on the health of individuals. Scholars have long documented a gradient effect between individual socio-economic status and health (Smith & Eggar 1992, Marmot et al. 1984, Bunker et al. 1989), but more recently—and perhaps more interestingly—researchers have discovered a link between high levels of income inequality and a number of negative health outcomes, including lower life expectancy, higher risk of infant mortality, obesity, and poorer average physical and mental health (Wilkinson & Pickett 2009; Pickett, Brunner, & Lobstein 2005). This dissertation has drawn on recent developments in the public health literature to test two possible pathways through which income distribution may operate on individual health: the *social cohesion mechanism* and the *local investment mechanism*. Guided by findings of this recent research, the overarching objective of this dissertation has been to re-examine these associations in consideration of some of the major methodological and theoretical limitations that have plagued previous analyses.

The methodological approach and theoretical framework developed in this dissertation have addressed two important limitations of current research examining the inequality-health relationship. First, I investigated whether variables related to the *social cohesion mechanism* and variables related to the *local investment mechanism* are effective at mediating the relationship between income inequality and health in metropolitan areas. Previous studies examining these relationships have investigated such pathways only at the national or state level. This dissertation has filled an important gap in the literature by exploring these relationships at lower levels of aggregations; namely, Metropolitan Statistical Areas (MSAs), Consolidated Metropolitan Statistical Areas (CMSAs), and Primary Metropolitan Statistical Areas (PMSAs).

Second, I used longitudinal data and employed a multi-level analytical strategy in order to assess the causal nature of the relationship between income inequality and individual health, and the mediating mechanisms through which this relationship may operate. For the most part, previous research has relied on cross-sectional data in order to draw inferences about the variables of interest. A key contribution of this dissertation to the larger bodies of inequality and health research is that it takes advantage of multi-level data, which has allowed me to better assess the causal relationships between of contextual-level variables variable of interest (income inequality, social cohesion, local investment) and individual-level variables of interest (individual mortality).

SUMMARY OF FINDINGS

The analyses conducted in this dissertation test hypotheses regarding the effect of income inequality on individual risk of mortality, and the mediating properties of the *Social Cohesion* and *Local Investment* mechanisms in this relationship. The first step in the analysis was to assess the influence of income inequality (as measured by the Gini coefficient) on the probability that an individual died during the follow-up period. I then controlled for the demographic, economic, and social characteristics of individual respondents to investigate the influence of such characteristics on the inequality-mortality relationship. And finally, I included key contextual-level explanatory variables related to the level of social cohesion and local investment within MSAs, CMSAs, and PMSAs to investigate the impact of these ecological factors on the inequality-mortality relationship. Table 9 summarizes the findings for each hypothesis tested, as outline in Chapter 3.

Hypothesis 1, which predicted that income inequality would be positively related to an individual's risk of mortality—even after controlling for family income—was *not supported*.

Results indicated a significant, positive association between average income inequality and individual mortality in basic models that controlled only for contextual-level characteristics (Table 6, Model 2). Furthermore, the effect of the Gini coefficient remained significant (albeit reduced) after the inclusion of an individual-level measure for family income. The influence of the Gini—even after the inclusion of a control for family income—indicated preliminary support for Hypothesis 1. However, once controls for individual-level characteristics (specifically, age, sex, race, and ethnicity) were included in the model, the effect of average income inequality on the probability of individual mortality was no longer significant (Table 6, Model 4).

As can be seen across all models that included individual-level control variables, it was the personal characteristics of individual survey respondents (age, sex, and race, region of residence, employment status, family income, educational attainment, marital status, and self-reported health) that played the largest role in predicting the odds of their death during the follow-up period. The association between basic demographic, economic, and social factors and odds of mortality are well documented (Rogers, Hummer, and Nam 1999) and not surprising. What was unexpected, however, was that these individual-level controls completely mediated the effects of income distribution on individual mortality. Such findings are inconsistent with the Income Inequality Hypothesis (IIH) and indicate that the association between inequality and health is not robust to individual-level attributes, at least not in metropolitan areas.

Hypothesis 2, which predicted that the positive effect of income inequality on an individual's risk of mortality will be reduced or brought to insignificance when contextual characteristics pertaining to community social cohesion are taken into consideration, was *not supported*. Given the unexpected findings regarding the relationship between average income inequality and the probability of individual mortality, the interpretation of results regarding the

social cohesion mechanism were made with caution. Both variables measuring the level of social cohesion within MSAs, CMSAs, and PMSAs (# of associations per 1,000 persons and # of third places per 1,000 persons) exhibited significant, negative associations with the probability of individual mortality (Table 7, Models 2 and 3). However, without evidence of an association between income inequality and individual mortality, such findings can only be interpreted as having direct—rather than mediating—effects on an individual's risk of mortality.

The presence of associations and third places is theorized to provide space for public interaction, thereby increasing social integration among residents, and ultimately, social cohesion within communities. In the past, scholars have utilized a civic community perspective to explain the connection between social and economic structures, and community well-being (Tolbert 2005; Tolbert et al. 1998). This perspective emphasizes the important role of locally oriented institutions and organizations, such as businesses, voluntary organizations, professional societies, and churches for increasing interaction among community residents. Scholars have linked high levels of collective efficacy—mutual trust and willingness to help each other—to positive health outcomes for individuals (Ewing et al. 2003). The findings in this dissertation (with regard to associations and third places) support the idea that increased social cohesion among community residents may be beneficial to individuals' health.

Table 9: Summary of Hypotheses and Findings

Hypothesis	Findings
Hypothesis 1: Income inequality will be positively related to an individual's risk of mortality, even after controlling for individual income.	Not supported. Multilevel results show that when controls for the demographic characteristics of individuals were included in models, the significant, positive association between income inequality and the individual risk of mortality disappears.
Hypothesis 2: The positive effect of income inequality on an individual's risk of mortality will be reduced or brought to insignificance when contextual characteristics pertaining to community social cohesion are taken into consideration.	Not supported. Both measures for social cohesion (associations and third places) were found to have significant, negative effects on the risk of individual mortality; however, given the lack of significant findings regarding the inequality-mortality relationship, these associations can only be interpreted as having direct effects. Multilevel models do indicate that associations and third places have a direct effect on an individual's risk of mortality.
Hypothesis 3: The positive effect of income inequality on an individual's risk of mortality will be reduced or brought to insignificance when contextual characteristics pertaining to local investment in public health infrastructure are taken into consideration.	Not supported. Neither measure for local infrastructure (physicians and hospital beds) was found to be significant associated with the probability of an individual dying during the follow-up period. Multilevel results do not indicate a direct effect between physicians/hospital beds and an individual's risk of mortality.

Hypothesis 3, which predicts that the positive effect of income inequality on an individual's risk of mortality will be reduced or brought to insignificance when contextual characteristics pertaining to local investment in public health infrastructure are taken into consideration, is *not supported*. Neither of the variables measuring the level of local investment in public health infrastructure (#of physicians and # of hospital beds per 1,000 persons) was shown to have a significant association with the probability of individual mortality. As discussed above, the findings the social cohesion and local infrastructure measures used in this dissertation could not be interpreted in terms of mediating the inequality-mortality relationship. In terms of direct effects, however, either of the key explanatory variables measuring local investment in public health infrastructure was significantly related to individual risk of mortality.

IMPLICATIONS OF FINDINGS

Relevance for Future Studies

The results of this dissertation contribute to a growing body of literature and empirical research that examines the effects of income distribution on the health of individuals. Although the findings of this study were contradictory to my expectations regarding the influence income inequality on individual mortality, such results still have important implications for future research in this area of study. As discussed below, the lack of support for the Income Inequality Hypothesis in this dissertation is especially relevant for the methodological and theoretical considerations of future studies of the inequality-health relationship.

One of the most important implications of the findings presented in this dissertation is the necessity of using multi-level modeling in any type of analysis that examines the relationship between income inequality and individual health. As the results of this study indicate, variables accounting for the demographic, economic, and social characteristics of individuals are

extremely important for explaining individual risk of mortality. In addition to directly explaining much of the variation in individual mortality, the individual-level controls included in this analysis were shown to mediate the negative effects of income inequality on the risk of individual mortality. These findings—based on multilevel data—a very different from the results of previous studies that have showed an association between income inequality and aggregate measures of health (Waldmann 1992; Wilkinson 1992; Kaplan et al.1996; Kennedy et al. 1996).Given these inconsistencies in findings, future studies examining the influence of income inequality on health outcomes should use a methodological technique that allows for simultaneous consideration of individual- and contextual-level control variables.

There are two reasons that it is important to use multi-level analysis to examine the influence of income inequality and other contextual characteristics on health outcomes. First, studies using aggregate data may be limited in regards to the inferences that can be drawn about individual-level exposure effects (Sheppard 2003). Research on public health often examines mortality *rates* within and between populations from some pre-defined ecological area (usually countries, states, or counties). Data are widely available and inexpensive for this type of analysis; however, their use is controversial because of the possible disconnect between the level of inference and the level of analysis (Piantadosi et al. 1988; Willett & Stampfer 1990). Interpretations of the inequality-health relationship based solely on aggregate-level data (i.e. when the dependent and independent variables are both at the ecological level) should be made with caution, as they may be statistically inaccurate and more likely the product of an ecological fallacy (Mellor and Milyo, 2002).

Second, some prior has suggested that the ecological associations between income inequality and health found in previous research may simply reflect the well-documented

influence of individual income on personal wellbeing, and not a true aggregate-level relationship (Backlund, Sorlie, & Johnson 1996; Ecob and Davey Smith 1999, Gravelle 1998, Subramanian & Kawachi 2004). This argument involves concern over the statistical power of some methodological strategies, which can and should be remedied through the adoption of more advanced statistical techniques. There are enormous social and political consequences that may stem from identifying the inequality-health relationship as a "statistical illusion," particularly when it comes to the development and implementation of policies designed to improve the health of individuals. Future research should address these methodological concerns and theoretical disagreements by adopting multi-level models.

In the past, researchers have been severely limited in their access to high-quality data that would allow them investigate the effect of contextual-level characteristics on health, while simultaneously controlling for the attributes of individuals. As data on individual health become more widely available and accessible, however, scholars should ensure that they are accurately testing their theoretical assumptions about the inequality-health relationship by adopting a multi-level study design. Future studies testing the Income Inequality Hypothesis should adopt multi-level modeling scheme, as this currently represents the most methodologically sound technique for investigating the complex relationships that exist between community characteristics and individual health (Daly, Duncan, Kaplan, & Lynch 1998; Kennedy, Kawachi, Glass, & Prothrow-Smith 1998). By merging and analyzing data from *both* the ecological and individual level, researchers will be able to avoid any type of inferential fallacy, isolate the contextual effects of income inequality from the influence of individual income, and more accurately estimate the causal links between income inequality, social cohesion, local infrastructure, and individual health outcomes.

A second important implication of the findings presented in this dissertation is that the relationship between income inequality and individual health is extremely complex, and that more research is needed in order to understand exactly how income inequality may work to influence individual health. Extensive reviews by Wilkinson and Pickett (2006, 2009) and Lynch et al. (2004) demonstrate that, for the most part, there is a significant, negative association between income inequality and health. As the findings in this dissertation and other studies suggest, however, the causal effects of inequality on health outcomes may be sensitive to the level of aggregation and to what researchers choose to control.

There is an extensive literature finding support for the Income Inequality Hypothesis, which suggests that it is income inequality, and not absolute income, that matters most in determining individual health outcomes (Wilkinson 1992, 1996; Kaplan et al. 1996; Kennedy et al 1996). However, the majority of studies demonstrating such support have examined this relationship at the national or state level. At lower levels of aggregation, such as census-tracts, counties—or in the case of this dissertation—metropolitan areas, findings regarding the empirical relationship between income distribution and health are less clear. Scholars have yet to explain exactly why such relationships may be statistically significant at some levels of analysis, but not others. The findings presented in this dissertation indicate that further research is needed to understand why income inequality may be a significant predictor of health at the national and state level, but not at lower levels of aggregation, such as MSAs, CMSAs, or PMSAs.

Research that has tested the Income Inequality Hypothesis also shows that results regarding the inequality-health relationship will vary from study to study, depending on the control variables included in the analyses. For example, some prior research has found that the

negative influence of income inequality on individual health disappears once controls for household income (Ficsella and Franks 2007) or racial composition of communities (Deaton and Lubotsky 2003) are controlled. The findings of this dissertation contribute the literature by showing the simultaneous effects of several individual- and contextual-level control variables on the inequality-health relationship, and help guide future research in terms of what control variables are most important to include in analyses.

Use of The Integrated Health Interview Series (IHIS) will prove useful in future studies of adult mortality in the United States. The IHIS—which is linked with the National Death Index—provides a rich source of data for any type of research aimed at identifying factors associated with a range of individual health outcomes. In addition to the benefits of large sample sizes and relatively small amounts of missing data, the IHIS provides information on several variables that pertain to individual health outcomes. For example, special topics data include individual health behaviors (e.g. smoking, weight control, alcohol intake), access to medical care (e.g. insurance, medication, dental, vision), health education (e.g. food knowledge, heart attack knowledge), and many more subjects related to the health-related activities of individuals. Most health-related research on individuals—including this dissertation—included variables measuring the effects of demographic and/or socioeconomic factors on mortality and other indicators of health. Future research can incorporate these additional variables and better investigate the correlates of physical and mental well-being, some of which may prove to extremely important in predicting health outcomes for individuals.

Furthermore, future studies could compare the effects of different measures for income inequality on the individual health outcomes, or the spatial concentration of income inequality on individual health outcomes. This dissertation used the Gini Index, as it is the most widely used

measure in previous research on the inequality-health relationship. However, other measures for income distribution are available (e.g. Theil Index or Robin Hood Index) and would provide additional insight into how these relationships operate. Previous research has examined the influence of income inequality within geographic areas overall. Perhaps future research could examine the spatial concentration of inequality within communities and the way income segregation can influence health outcomes, particularly for those individuals living on the border between neighborhoods characterized by a high rate of poverty or affluence.

In addition to new variables of interest, future studies should explore cross-level interaction effects between contextual-level variables (e.g. income inequality) and individual-level variables (e.g. family income). The purpose of including interaction terms into analyses of health would be to examine how family income may condition the relationship between income inequality and individual health. In this dissertation, it was found that individual attributes had a significant moderating effect between income inequality and individual mortality. For example, when individual-level controls are entered into the model one by one, it is the effect of age that renders the income inequality-mortality relationship insignificant. Future research should examine the potential interactions between the measures of inequality (Gini coefficient) and variables related to the demographic and economic characteristics of individuals to determine whether the effects of average income inequality may be more or less pronounced for individuals with a certain level of family income, or of a certain age, race, or ethnic background.

Relevance for Policy

Findings in this dissertation, especially those related to the protective effect of community social cohesion on individual health, are useful not only for the methodological or theoretical foundation of future research, but for policy decisions as well. A third important implication of

the findings in this dissertation relates to the types of policies that could stem from research that investigates the effect of contextual-level characteristics on individual health outcomes. This dissertation tested the two most recognized pathways through which income inequality is believed to operate on individual health: the social cohesion mechanism and the local investment mechanism. Although findings did not support hypotheses predicting the mediating effects of these pathways in the inequality-health relationship, the results of this dissertation do have implicate the *direct* effect of social cohesion to reduce poor health outcomes for individuals. This finding may be useful in informing public policies that aim to reduce negative health outcomes for individuals within communities.

There is often debate about whether public health policies should focus on individuals' own personal responsibility regarding health-related behaviors, or should address more structural factors that may be related to individual health outcomes. Although I do not disagree that many negative health outcomes for individuals could be avoided or remedied through changes in individual behaviors, the direct effect of social cohesion on individual health as revealed in this dissertation indicate that a broader focus that includes community-based policies is warranted. In terms of informing public policy, this finding supports the implementation of public health policies that are designed to increase social integration and community involvement. The results of this study indicate that increased funding for community programs and activity centers that are likely to increase civic engagement may be extremely productive in reducing the negative health outcomes.

LIMITATIONS

The findings of this dissertation make a contribution to both the sociological and public health literature; however, there are several notable limitations that should be discussed. First, the years

of longitudinal data included in the present analyses are few, ranging only from 1997-2001. Data on individuals, as provided by the Integrated Health Interview Series (IHIS) is currently linked with the National Death Index (NDI) for the years 1986-2006. This linkage allows researchers to access information on respondent's final mortality status, along with year and cause of death. The link between IHIS and NDI proves to be a great resource, as it allows researchers to analyze covariates of the risk of mortality. Unfortunately, the mortality weight variable (provided by IHIS) to be use in analyses of the sample adult population is only available for survey years 1997 forward. As survey respondents who have missing data may differ from those who do not have missing data, ignoring the mortality-weight variable could lead to biases in the mortality analyses. For this reason, analyses in this study are limited to the years 1997 forward.

Second, the age of the data used in these analyses is slightly outdated. The individual-level data used in this study are based on individuals who participated in the National Health Interview Survey from 1997 until 2001, and the contextual-level data are based on the census data from 2000. Although the age of the data does not preclude the methodological strengths of this analysis, it should be noted that findings are based on data that are more than a decade old, and all interpretations should take this fact into consideration.

As mentioned previously, the IHIS is linked to the NDI until 2006. However, the National Health Interview Survey (from which IHIS data is gathered) identification codes for MSAs, CMSAs, and PMSAs are only available from the Centers for Disease Control and Prevention until 2001. This was a severe data limitation for the current study in terms of the number of years available for a longitudinal study. As more data on individuals becomes available, however, and are able to be linked to contextual data on metropolitan areas and the

National Death Index, the sample of individuals who die during the follow-up period will be higher, allowing for a more thorough investigation of the covariates of adult mortality.

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APPENDIX A: LIST OF MSAS, CMSAS, AND PMSAS USED IN THE ANALYSIS

Geographic Identifier	Geography
0520	Atlanta, GA MSA
0640	Austin--San Marcos, TX MSA
1122	Boston--Worcester--Lawrence, MA--NH--ME--CT CMSA
1280	Buffalo--Niagara Falls, NY MSA
1520	Charlotte--Gastonia--Rock Hill, NC--SC MSA
1602	Chicago--Gary--Kenosha, IL--IN--WI CMSA
1642	Cincinnati--Hamilton, OH--KY--IN CMSA
1692	Cleveland--Akron, OH CMSA
1840	Columbus, OH MSA
2082	Denver--Boulder--Greeley, CO CMSA
2162	Detroit--Ann Arbor--Flint, MI CMSA
3120	Greensboro--Winston-Salem--High Point, NC MSA
3280	Hartford, CT MSA
3362	Houston--Galveston--Brazoria, TX CMSA
3480	Indianapolis, IN MSA
3600	Jacksonville, FL MSA
3760	Kansas City, MO--KS MSA
4520	Louisville, KY--IN MSA
4920	Memphis, TN--AR--MS MSA
5082	Milwaukee--Racine, WI CMSA
5120	Minneapolis--St. Paul, MN--WI MSA
5360	Nashville, TN MSA
5560	New Orleans, LA MSA
5720	Norfolk--Virginia Beach--Newport News, VA--NC MSA
5880	Oklahoma City, OK MSA
5960	Orlando, FL MSA
6200	Phoenix--Mesa, AZ MSA
6280	Pittsburgh, PA MSA
6442	Portland--Salem, OR--WA CMSA
6480	Providence--Fall River--Warwick, RI--MA MSA
6820	Rochester, MN MSA
6922	Sacramento--Yolo, CA CMSA
7040	St. Louis, MO--IL MSA
7160	Salt Lake City--Ogden, UT MSA
7240	San Antonio, TX MSA
7320	San Diego, CA MSA
7602	Seattle--Tacoma--Bremerton, WA CMSA
8280	Tampa--St. Petersburg--Clearwater, FL MSA
8960	West Palm Beach--Boca Raton, FL MSA
19221920	Dallas, TX PMSA; Dallas--Fort Worth, TX CMSA

APPENDIX A continued

19222800	Fort Worth--Arlington, TX PMSA; Dallas--Fort Worth, TX CMSA
44724480	Los Angeles--Long Beach, CA PMSA; Los Angeles--Riverside--Orange County, CA CMSA
44725945	Orange County, CA PMSA; Los Angeles--Riverside--Orange County, CA CMSA
44726780	Riverside--San Bernardino, CA PMSA; Los Angeles--Riverside--Orange County, CA CMSA
49922680	Fort Lauderdale, FL PMSA; Miami--Fort Lauderdale, FL CMSA
49925000	Miami, FL PMSA; Miami--Fort Lauderdale, FL CMSA
56020875	Bergen--Passaic, NJ PMSA; New York--Northern New Jersey--Long Island, NY--NJ--CT--PA CMSA
56025015	Middlesex--Somerset--Hunterdon, NJ PMSA; New York--Northern New Jersey--Long Island, NY--NJ--CT--PA CMSA
56025190	Monmouth--Ocean, NJ PMSA; New York--Northern New Jersey--Long Island, NY--NJ--CT--PA CMSA
56025380	Nassau--Suffolk, NY PMSA; New York--Northern New Jersey--Long Island, NY--NJ--CT--PA CMSA
56025600	New York, NY PMSA; New York--Northern New Jersey--Long Island, NY--NJ--CT--PA CMSA
56025640	Newark, NJ PMSA; New York--Northern New Jersey--Long Island, NY--NJ--CT--PA CMSA
61626160	Philadelphia, PA--NJ PMSA; Philadelphia--Wilmington--Atlantic City, PA--NJ--DE--MD CMSA
73625775	Oakland, CA PMSA; San Francisco--Oakland--San Jose, CA CMSA
73627360	San Francisco, CA PMSA; San Francisco--Oakland--San Jose, CA CMSA
73627400	San Jose, CA PMSA; San Francisco--Oakland--San Jose, CA CMSA
88720720	Baltimore, MD PMSA; Washington--Baltimore, DC--MD--VA--WV CMSA
88728840	Washington, DC--MD--VA--WV PMSA; Washington--Baltimore, DC--MD--VA--WV CMSA

APPENDIX B: EXAMPLE PERSON-YEAR DATA FILE

MSA/CMSA/PMSA	NHIS Person ID	Sex (Female =1)	Age	Mortality Status (Death =1)	Year
0520		1	32	0	1997
0520		1	33	0	1998
0520		1	34	0	1999
0520		1	35	0	2000
0640		0	52	0	1997
0640		0	52.5	1	1998

Notes:
This example person-year file includes information on two individuals: A female respondent who survived the follow-up period (contributing 3.5 person years) and a male respondent who died during the follow up period (contributing 1.5 years).

Most of the individual-level data are non-time-varying; however, the age of individuals in the person-year was adjusted each year the respondent was followed.

VITA

Lisa Winters was born in Carlsbad, New Mexico. She attended Oklahoma State University in Stillwater, Oklahoma, where she received her Bachelor of Science degree in political science with a minor in sociology and women's studies in 2005. She received her Master of Arts degree in sociology from University of Memphis in 2008. She will receive her Doctor of Philosophy degree in sociology from Louisiana State University during the fall 2012 commencement ceremony.