

ABSTRACT

Title of Dissertation: EPIDEMIOLOGICAL TRANSITION AND
SHIFTING MORTALITY INEQUALITY: AN
EXTENSION OF FUNDAMENTAL CAUSE
THEORY

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The dissertation addresses two "public health puzzles" in US mortality inequality trends: (1) SES inequalities in mortality have been growing wider despite declines in overall mortality levels and the expansion of social welfare policies; (2) mortality inequalities present diverging trends across age groups, with declines at younger ages but growth at older ages. These puzzles challenge existing theories in explaining the complex dynamics of mortality disparities. The study aims to bridge this gap by proposing an alternative theoretical framework that combines Fundamental Cause Theory with the concept of epidemiological transition.

Previous research has focused primarily on socioeconomic factors as the main drivers of widening mortality disparities. However, this dissertation argues that mortality inequalities can evolve independently of socioeconomic factors due to shifts in disease patterns towards non-communicable diseases and advancements in health-beneficial innovations. By analyzing county-level US mortality rates from 1968 to 2020, this study reveals that mortality inequality related to infectious diseases declined in the early 1970s and remained stable over time. On the

other hand, mortality inequality related to non-communicable diseases remained at a low level during the 1970s but saw a significant increase since the 1980s. Further, this study found that mortality inequality from non-communicable diseases is more pronounced in middle-aged and older adults, and the age distribution of mortality inequality progressively shifts towards older ages.

This study contributes to the existing literature with a new theoretical perspective to understand the developments of mortality inequalities over time. This framework sheds light on the two "public health puzzles" and emphasizes the crucial role of disease patterns prevailing during specific historical periods in understanding the developments of mortality inequality. Furthermore, the study underlines the interplay of disease patterns, prevention/treatment innovations, and social and economic inequalities in collectively shaping the future of mortality and health disparities. It also sheds light on the social-political circumstances of medical innovation as well as behavioral factors over the life course in determining future population health and health inequalities.

EPIDEMIOLOGICAL TRANSITION AND SHIFTING MORTALITY
INEQUALITY: AN EXTENSION OF FUNDAMENTAL CAUSE THEORY

by

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Chapter One: Introduction

Overview

Over the past century, the United States (US) has made colossal achievement in population health. The US life expectancy at birth was only about 47 years in 1900; the number increased to almost 79 years in 2015 (although it plateaued since 2015 and then dropped by three years during Covid-19). Despite substantial improvement in overall population health and massive achievement in medicine and public health over the years, socioeconomic (SES) inequalities in mortality and health are pervasive, existing “pretty well everywhere” (Marmot, 2004 p.16) and “whenever and wherever it is sought” (Deaton, 2016 p.1703). Even worse, SES inequalities in mortality and health have been widening over time since at least the second half of 20th century (Chetty et al., 2016a; Crimmins et al., 2010; Cutler et al., 2011a; Ezzati et al., 2008a; Meara et al., 2008; Montez & Berkman, 2014; Murray et al., 2006; Olshansky et al., 2012; Pijoan-Mas & Ríos-Rull, 2014; Singh & Siahpush, 2006; Wang et al., 2013).

The enduring and widening inequality in health and mortality has become one of the most critical public policy challenges in the United States. This problem has become increasingly urgent due to the worrisome state of the US population's health in recent years. Studies show that the United States has the lowest level of life expectancy and higher prevalence of chronic conditions compared with other high-income countries (Woolf & Aron, 2013). The disadvantage has become much worse in recent years: the US experienced a plateau in life expectancy since 2015 (Harper et al., 2020), followed by a staggering increase in deaths and a decline of approximately three years in life expectancy during the COVID-19 pandemic (Davis &

Lederberg, 2000). The *Healthy People 2030* elevates health equality as the central focus for the next decade of US national health improvement goals (Pronk et al., 2021) and calls for systematic interventions to eliminate health disparities (Johnson et al., 2020; Pronk et al., 2021; U.S. Department of Health & Human Services, 2021).

To move toward health equality and find ways to reduce inequalities in health and well-being, we must have a better understanding of the complex mechanisms linking SES and health, especially how the disparities develop and change over time.

Two Puzzles in Existing Theories and Empirical Studies

We have made significant progress in both theoretical and empirical domains regarding the development and changes in health disparities over time, yet significant questions remain unanswered. Link and Phelan (1995) proposed that socioeconomic status (SES) is a fundamental social cause of health, whereby a person with higher SES, like education, income, and occupation, possesses “a wide range of broadly serviceable resources including money, knowledge, prestige, power, and beneficial social connections” (Link & Phelan, 1995). These varied resources can be used broadly and flexibly to protect individual health—for example, the knowledge of, access to, and ability to obtain health care and to engage in health-enhancing behaviors. Further, those with more resources also benefit from the broad contexts of their neighborhoods, social networks, and work/occupational environments. Because of the wide-ranging, flexible, and multiple purposes of these resources, those with higher SES can use them to benefit their health through a long and detailed list of risk and protective mechanisms (Link & Phelan, 1995) making the association between SES and health so robust and enduring despite changes over time in health outcomes and risk factors.

SES inequalities in health/mortality persist through these “flexible resources”. Empirical studies also found the growth of SES inequalities in US society leads to widening health/mortality inequalities over time. Rising income inequality (J. W. Lynch et al., 2000; Subramanian & Kawachi, 2004) and the deterioration in occupation structure for the less educated population (Ahonen et al., 2018; Burgard & Lin, 2013; Landsbergis et al., 2014, 2018; Ravesteijn et al., 2013) are associated with widening health/mortality inequalities. The widening health/mortality inequalities could also be reproduced through behavioral factors like smoking (Montez & Zajacova, 2013) and aggravated by social policies that limit access to medical care and health insurance in the US (Woolf & Braveman, 2011).

Despite all the theoretical and empirical evidence documenting health disparities, we still grapple with at least two puzzles or paradoxes. The US and other developed countries have seen an unprecedented decline in mortality, and a massive improvement in living conditions, nutrition, health care, insurance, medical techniques, and welfare policies during the 20th century (Elo, 2009; Preston, 1996; White & Preston, 1996). Nevertheless, health inequalities still endure and have been even widening. We have yet to find a satisfactory explanation for the contrasting trends observed between the progress in reducing overall mortality rates and the persistent failures in decreasing mortality inequalities. There is good evidence that rising SES inequality (e.g., income inequality or job market polarization, etc.) contributes to widening health/mortality inequalities. However, health and mortality inequalities have been widening not only in countries like the United States and the United Kingdom with huge and rising social inequality, but also in countries like Sweden and other Nordic countries with much more generous social welfare policies and equal income redistribution (Bambra, 2011; Mackenbach, 2012). Studies in European countries even found that the magnitude/increase of

mortality/morbidity inequality is not strongly associated with "the extent or intensity of welfare policies in a country" (Mackenbach, 2012), which makes this paradox even more puzzling. "Existing theories provide little insight into the issue," and this "public health puzzle highlights the limitations of existing theories" (Bambra, 2011).

Another paradox arises from the diverging trends in mortality inequalities across age groups. Most existing studies of mortality inequality focus on SES inequality in overall life expectancy, age-standardized mortality, or mortality at certain age groups (especially older ages where most deaths occur). These studies implicitly assume that mortality inequalities at different age groups or life stages (e.g., early life, middle life, and later life) have changed in the same or similar direction and magnitude. Nevertheless, very few studies have tested whether this implicit assumption is accurate. Only recently has research uncovered diverging trends in mortality inequality across different stages of the life course. Currie and colleagues demonstrated that mortality inequality declined for infants, children, and young adults in the US between the 1990s and the 2010s, while remaining relatively stable or showing minimal change for individuals in middle age; however, it revealed a significant and alarming increase in mortality inequality among older adults (Baker et al., 2021; J. M. Currie, 2018; J. Currie & Schwandt, 2016a; Schwandt et al., 2021). These divergent trends are impressive and puzzling because most existing explanations for adult health disparities (e.g., rising income inequalities, deterioration of jobs for the less educated, worsening health behaviors) should also affect children's and young adults' health and survival.

In sum, while previous studies have increased our understanding of the great gains in health and longevity, they have made little progress in solving these two remaining puzzles: 1) the growing inequalities by SES despite declining overall mortality levels and expansion of

social welfare policies, and 2) the declining disparities at younger ages but growing inequality at older ages. These two paradoxes underscore the need for new theoretical perspectives that foster a deeper understanding of the SES-mortality connection and the developments/changes in mortality and health inequality.

Shifting Disease Patterns and Health/Mortality Inequalities

This study attempts to address this critical gap by providing an alternative theoretical framework for understanding the changes of health/mortality inequalities over time. This framework relies on Fundamental Cause Theory (FCT) but links it to the historical context of the epidemiological transition. Fundamental Cause Theory (FCT) argues that SES inequalities in health endure over time even as conditions and risk factors change because when inequalities in some diseases have been reduced, the inequalities shift to new diseases (Link & Phelan, 1995; Phelan et al., 2004), which helps explain the persistence of SES inequalities in health. However, what is not well addressed in Fundamental Cause Theory (Miech, Pampel, Kim, et al., 2011a) is that shifts in health disparities by types of diseases do not occur in a vacuum but rather within the context of the epidemiological transition and shifting disease patterns. Based on this idea, I propose a new theoretical framework for understanding historical changes in health inequalities.

The main idea of this conceptual framework is that the SES inequalities in health/mortality shift alongside the changes in disease patterns and advances in medical innovation. The Epidemiological Transition Theory describes the declines in overall mortality as well as the shift in disease patterns from infectious diseases to degenerative diseases over the past several centuries (Omran, 1971). In this study, I propose that health and mortality inequalities stemming from infectious diseases have been substantially reduced. Then,

inequalities stemming from degenerative diseases have widened over time and account for the new emergent disparities.

Over the past centuries, the improved access to sanitation and living conditions, widespread vaccination efforts, and other related developments have reduced deaths from infectious diseases to a low level. Further, access to these prevention and treatment tools has become near-universal, leaving little room for SES inequalities. However, since the 1960s, numerous medical innovations (prevention/treatment) for chronic and degenerative diseases have emerged. The treatment of heart diseases (coronary care units, inhibitors, aspirin, statins, cardiovascular surgery, etc.) has improved largely since the 1960s. The improvement in screening/diagnostic techniques (e.g., blood glucose monitoring, colorectal cancer screening) and changes in lifestyle (e.g., smoking, diet, and physical activities) contribute to the decline in cardiovascular diseases, as well as many kinds of cancer and other chronic conditions (e.g., diabetes, COPD, etc.). However, it is important to note that as new knowledge, prevention, treatment interventions, and medical innovations emerge, they disproportionately benefit individuals with higher socioeconomic status (SES). This has resulted in an increase in health inequalities related to degenerative diseases, and hence, degenerative diseases have surpassed infectious diseases in their contribution to overall patterns of inequality. These patterns persist regardless of the extent or intensity of welfare policies or the level of SES inequalities within a country.

Further, the receding of pandemics and the increase in degenerative diseases led to a redistribution of disease burden and death from younger to older ages (Murray & Lopez, 1996). Degenerative diseases (e.g., heart diseases and cancer) and the rising inequality in degenerative diseases are much more apparent for middle-aged and older adults—with only a minor influence

on younger age groups. This helps explain the diverging mortality inequality trends across age groups.

This study aims to empirically test this theoretical framework and examine how well it helps us to understand the development and changes in health/mortality inequality over time. To be specific, I address the following questions:

- (1) How do SES inequalities in mortality change over time? And how do the trends vary by diseases (especially the difference between degenerative diseases and infectious diseases)?
- (2) How do trends in mortality inequalities vary across age groups? And how do rising inequalities from degenerative diseases help explain the diverging trends across age groups?
- (3) How has the emergence of medical innovations targeting degenerative diseases in the past decades (e.g., the improvement in treatment or prevention of heart diseases, cancer, diabetes, COPD, etc.) helped to explain the rise and fall of mortality inequalities for specific diseases?

The county-level US mortality data (1968-2020) by age, sex, and cause of death will be used to test the changes over time in all-cause and cause-specific mortality inequality, across age groups. Subsequently, I investigate the temporal aspects of medical innovations and the dissemination of these medical innovations across historical periods. The objective is to understand how well the emergence of medical innovations align with changes in mortality inequalities.

Significance of the Current Study

This work contributes to the existing literature in at least three ways. First, by extending fundamental cause theory and linking it to epidemiological transition under historical contexts, this study provides a new framework for understanding trends in SES inequalities in

mortality/health. This framework sheds light on the shifts in mortality inequalities among different diseases, helping elucidate the paradoxes surrounding persistent health inequality despite a decline in overall mortality rates and the expansion of social welfare policies. Additionally, it explains the contrasting trends in mortality inequalities across various age groups.

Secondly, this framework offers new insights about the future changes in mortality and health inequality, as well as the potential policy implications. As we look ahead, non-communicable diseases appear to be the predominant future burden. Mortality inequality has been gradually shifting towards older age groups, leading to the rising importance of life course experiences (the accumulation of risk and exposure during the whole life span) in determining future population health and health inequalities. Meanwhile, medical knowledge and technology affect the population's health as well as health inequalities through "a thick distribution of social, political and economic circumstances"(Link 2008, p.370). The social-political factors (economic resources, social network, healthcare, etc.), as well as the habit, lifestyle, and cultural capital (as described by Bourdieu) have been growing in influence on health disparities (Mackenbach 2017).

Thirdly, the framework and method used in this study have promising potential for future research in health and mortality disparities over time by race and gender as well as SES inequality in health during different stages of epidemiological transition (e.g., when infectious diseases are still the main burden of disease or when both infectious and degenerative diseases are dominant).

Chapter Two: Literature Review

The Robust and Widening SES Inequalities in Health

The positive association between SES and health has been widely examined in the United States (Preston et al., 1973). Extensive literature has documented a robust relationship between various measures of SES (e.g., education, income, wealth, occupational status, poverty rate, etc.) and a wide array of health outcomes (mortality, morbidity, self-reported health). Despite substantial improvement in overall population health and massive achievements in medicine and public health over the centuries, SES inequalities in health are very strong and persistent (Deaton, 2016 p.1703).

To make matters worse, studies found that SES inequalities in mortality and health have widened in the US. For example, SES inequality in mortality and life expectancy at the county level has been widening in the US since the 1980s (Ezzati et al., 2008b; Singh & Siahpush, 2006). The educational gap in mortality and life expectancy grew wider between the 1960s (Feldman et al., 1989a; Pappas et al., 1993) and the 1990s (Crimmins & Saito, 2001; Elo & Preston, 1996; Meara et al., 2008). The gap continued to widen during the first two decades of the 21st century in the US (Bound et al., 2015; Case & Deaton, 2015, 2020; Cutler et al., 2011b; Dowd & Hamoudi, 2014; Hayward et al., 2015; Sasson, 2016a, 2017; Zajacova & Lawrence, 2018). Figure 2.1 shows the widening educational disparities in life expectancy at 25 in the US from 1990 to 2017, with steeper improvements for the best educated, and the disturbing signs of flat trends (or even declines) for the least educated white men and women. And income-related inequality in life expectancy has been widening over time as well (Chetty et al., 2016a). Other developed countries such as Sweden, Finland, and Norway in northern Europe present similar

trends (Chetty et al., 2016b; Halpern-Manners et al., 2020; Kunst & Mackenbach, 1994; Mackenbach et al., 2003). In addition to mortality and life expectancy, the SES disparities in morbidity, disability, and self-reported health have also been widening over time (Kc & Lentzner, 2010; S. M. Lynch, 2006; Molla et al., 2004; Singh et al., 2017; Tsai, 2017; Zajacova & Lawrence, 2018).

[--Figure 2.1 about here--]

This persistent and growing health and mortality inequality has caused concern because the US has seen an unprecedented decline in overall mortality during the 20th century (Elo, 2009; Preston, 1996; White & Preston, 1996).

The Explanation for the Robust and Widening SES Inequalities in Health

Many mechanisms could mediate the link between SES and health. Those with higher socioeconomic status possess a broader range of economic, social-psychological, and interpersonal resources (S. M. Lynch, 2003) that may allow them to take better advantage of the information, new knowledge, behaviors, medical interventions, and beneficial environment to protect and improve their health (Adler & Rehkopf, 2008; Link & Phelan, 1995; Lynch et al., 2000; Montez et al., 2019; Pudrovska, 2014; Zajacova & Lawrence, 2018).

However, the SES-health link cannot be simplified or reduced to these proximate factors or mechanisms. During the 1980s and even 1990s, SES has been viewed as only a distal determinant of health. Based on this perspective, SES is essential because it affects the proximate determinants of health such as standard of living, sanitation, risks of exposure, access to health care, treatment of disease, or health-related behavior (Adler et al., 1994). This orientation simplifies the impact of SES on health to the proximal risk factors and assumes that attention and

policy interventions should be focused on these proximal risk factors (Link & Phelan, 1995). During the last century, most of the proximal factors linking SES to health (e.g., sanitation, clean water, crowded living conditions, and vaccines) have been identified and addressed in the US (Kadushin, 1964). Researchers expected the SES-health association to quickly wane or even disappear.

Nevertheless, the association did not disappear. The SES inequalities in mortality, life expectancy, and disease continued to persist and even increase over time. Indeed, the proximal factors like sanitation, clean water, immunization, crowded living conditions, and standard of living have primarily been addressed. However, studies found that these risk factors are replaced by new factors, including smoking, exercise, diet, which were of little importance before (Link & Phelan, 1995). Based on this observation, Link and Phelan proposed that SES is a fundamental social cause of health because SES embodies “a wide range of broad resources including money, knowledge, prestige, power, and beneficial social connections” (Link & Phelan, 1995). These resources are flexible, multi-purpose, and transformative from one situation to another. No matter the risks, diseases, and protective mechanisms, people of higher SES have more resources allowing them to know more about diseases, avoid the risk factors for poor health, and gain access to the means of protecting health and obtaining effective treatment to limit the consequences of disease (Link & Phelan, 1995). In this way, Fundamental Cause Theory helps to explain the persistent and enduring SES inequalities in health and mortality. The “flexible” resources are transformative from one situation to another, and the association between SES and health is reproduced via the replacement of risk factors, disease patterns, and protective factors dominating at that time (Link & Phelan, 1995; Phelan et al., 2004). When one

mechanism/mediating factor declines or is addressed, the alternative factors emerge and become more prominent (Phelan et al., 2004).

However, Fundamental Cause Theory is less able to explain the widening health and mortality inequalities in recent decades. Despite the many improvements in living conditions, insurance, public health programs, medical technology, and the welfare system during the past century, SES inequalities in mortality and health continue to increase (Elo, 2009; Preston, 1996; White & Preston, 1996). Empirical research suggests that widening health inequalities are associated with an increase in overall SES inequalities. For example, the rising income inequality (J. W. Lynch et al., 2000; Subramanian & Kawachi, 2004) and the deterioration in occupational structure and job insecurity for the less educated population (Ahonen et al., 2018; Burgard & Lin, 2013; Landsbergis et al., 2014, 2018; Ravesteijn et al., 2013) contribute to the widening health disparities. The widening SES gaps are reproduced through behaviors like smoking (Montez & Zajacova, 2013), and they are aggravated by unequal access to medical care and insurance in the US (Woolf & Braveman, 2011). The differences in state level policy within US, or the comparison of policy context between US and Canada/Europe shows that welfare-state policy helps explain the troubling trends and inequalities in US life expectancy (Avendano & Kawachi, 2014; Beckfield & Bambra, 2016; Montez et al., 2019, 2020; Van Dyke et al., 2018). Some other studies also attribute the widening health/mortality disparities to the compositional changes in SES whereby the expansion of education makes the less educated more negatively selected (Bound et al., 2015).

Although a country's rising level of SES inequality (e.g., income inequality or job market polarization) and welfare-state policy clearly contributes to widening mortality inequalities, it cannot explain the rising health inequalities in countries like Sweden and other Nordic countries

with much more generous welfare policies and income redistribution (Bambra, 2011; Mackenbach, 2012). Studies in European countries even found that the magnitude of mortality/morbidity inequality and the speed of rising inequality is not strongly associated with “the extent or intensity of welfare policies in a country” (Mackenbach, 2012), which makes this paradox even more puzzling. “Existing theories provide little insight into the issue,” and this “public health puzzle highlights the limitations of existing theories” (Bambra, 2011). So far, we still do not have a satisfactory answer as to why the health/mortality inequality continues to widen so universally, no matter the extent of welfare policies and the unprecedented decline in overall mortality.

Diverging Mortality Inequalities Across Age groups

Most research on health disparities focuses on general health measures and pays less attention to the heterogeneity of health and mortality inequality trends at different age groups/life course stages (e.g., early life vs. middle life vs. later life). The overall SES-mortality gradient and its widening trend over time have been well documented; most studies look at mortality in adults (especially older ages where most deaths occur) or age-adjusted mortality and life expectancy at birth. The age-adjusted mortality rate and life expectancy are summary measures which collapse the age-specific rates into a number and are also dominated by mortality at older ages because that is where most mortality happens. In most cases, these studies assume implicitly that inequalities in health and mortality in early, mid, and later life change in the same direction and magnitude.

Although SES-Health gradients exist throughout the entire life course, and trends in the gradient at different life stages may vary in magnitude and direction, few studies have assessed

whether the trends are similar for people at different life stages or age groups. One notable exception is work by Meara and colleagues (2008) which found that the widening disparities in life expectancy at age 25 between the 1980s and 1990s was due primarily to the widening gap for older adults 65+, with almost no change for adults 25-44 (see Figure 2.2) (Meara et al., 2008).

[--Figure 2.2 about here--]

However, this study did not consider mortality trends among infants, children, and young adolescents. It was not until recently that several studies found that mortality inequality had declined for infants, children, and young adults in the US from 1990 to 2010s, with stable or minor change for those in middle life, but it increased dramatically for older adults (Baker et al., 2021; J. M. Currie, 2018; J. Currie & Schwandt, 2016a, 2016b; Schwandt et al., 2021). Using county-level mortality data in the US, Currie & Schwandt ranked all US counties from the poorest to the richest and grouped all the counties into 20 county-bins with almost equal population size, and then compared the county-level SES differences in mortality in 1990 and 2010. The mortality rate decreased over time among all age groups for counties at any rank. However, mortality inequality between the richest (top 5%) and the poorest (the bottom 5%) county-bin declined for younger age groups (men under age 50 and women under 30), primarily because the poorest counties experienced greater gains in health. For middle aged adults (men 50-64 and women 30-44), mortality inequality remained stable or increased slightly, but it increased dramatically for both men and women at older ages as the wealthiest counties experienced a faster gain in elder health).

Similar age group variation in mortality inequality has been found among subgroups of US population (US blacks) and in other developed countries. Based on a similar approach,

previous studies (J. Currie & Schwandt, 2016a, 2016b) found that African Americans experienced a significant decrease in inequality in early and middle life mortality but an increase in inequality in later life (J. Currie & Schwandt, 2016a). They also compared the trends in mortality inequality between US and Canada, finding that Canada experienced no change in inequality among children and young adolescents but a significant increase in inequality for men over age 24 and women aged over 14 (Baker et al., 2019). Two more recent studies of mortality inequalities in European countries also indicate a flattening of mortality inequality in early life, with increases in inequality concentrated in older ages (Baker et al., 2021; Schwandt et al., 2021a).

These divergent trends are impressive and puzzling. Most existing explanations for adult health disparities (e.g., rising income inequalities, deterioration of jobs for the less educated, worsening health behaviors) should also affect children's and early/middle aged adults' health. Clearly, a better explanation is needed for these divergent trends.

Extension of Public Health Resources for Children

As mentioned above, past research has made little progress in understanding the divergent health inequality trends at different life stages. Existing explanations about widening health inequality (e.g., the rising income inequality, the structural change in the labor market) should affect health at all ages. The changes in social and economic structures (e.g., rising income inequality) seem inadequate to explain this diverging trend across life course stages.

The only existing explanation comes from Currie (2018), who attributed it to the extension of health insurance (e.g., Medicaid /SCHIP) for pregnant women and children under 18, as well as the expansion of other public programs supporting poor women and young

children including the earned income tax credit (EITC), food stamps (SNAP), and state-level pre-kindergarten (pre-K) programs (J. M. Currie, 2018). This line of research shows that from the 1980s to 2000s, eligibility for Medicaid/SCHIP expanded dramatically for poor pregnant women and children in the US (J. Currie et al., 2008)¹, thereby improving access to health care to infants and young (J. Currie & Gruber, 1996), older children aged 9-17 (J. Currie et al., 2008). In addition, it led to a decline in adolescent mortality for black children (but not for white children) aged 15-18 (L. R. Wherry & Meyer, 2016). These findings led Currie and her colleagues to argue that the expansion of public insurance and health resources can also explain the reduction in health inequality among infants and children (Baker et al., 2019; J. M. Currie, 2018)

Further, studies show that the expansion of public insurance eligibility has positive long-term effects on outcomes over the life course: better health, fewer hospitalizations, lower mortality, improved education, and lower poverty in adolescence and adulthood (Brown et al., 2015; J. Currie et al., 2008; L. Wherry et al., 2018; L. R. Wherry et al., 2016; L. R. Wherry & Meyer, 2016). This long-term effect may also explain at least some of the reduction in inequality for those up to about age 30 (Baker et al., 2019).

All these studies are insightful and convincing. The expansion of public health insurance and other public resources must play a role here. However, this explanation is only part of the story and is not satisfying enough for at least two reasons.

¹ Some background information about the expansion of Medicaid and SCHIP is shown in (Currie, Decker, and Lin 2008, p.1568).

First, the expansion of public health insurance and other public resources does not close the disparities of life chances at early life. Recent studies consistently show that the inequality in early-life parenting, childcare investment, and education has been very significant and widening in the US (Altintas, 2016; Federal Interagency Forum on Child and Family Statistics, 2021; Kornrich & Furstenberg, 2013). Studies also found rising income inequality in the US is associated with a widening gap in parental financial investment in children (Schneider et al., 2018), and the gap has been accentuated by the growth of single-parent families (Bianchi et al., 2004). The disparities in children's parents in risk behaviors like smoking, and even health outcomes including the incidence of obesity, poor/fair health, and loss of work because of illness are still very significant (Bianchi et al., 2004).

Second, in terms of public insurance and other public resources, the expansion of eligibility happens not only among infants and children but also among older adults. The social security and health insurance programs for older adults were created and improved much earlier than the programs for infants and children. During the 20th century, the federal social security system (since the 1930s) and Medicare (since the 1960s) were created to provide income and routine health care for all older adults during their retirement years. Then the Supplemental Security Income (SSI) since the 1970s, and Medicaid also provide income and healthcare resources for the most vulnerable older adults (Street et al., 2016). In 2001, only 0.8% of older adults aged 65 and above were uninsured, but the number for children under age 18 was about 11.7% (Mills & Bhandari, 2003). According to recent Current Population reports, children under 19 still have a much higher percentage of people uninsured (about 5.6% in 2020) than older adults aged 65 and above (about 1% in 2020) (Berchick et al., 2019; Kinney, 2020). If health insurance and access to health care are the main stories, we should also observe a decline in older

adults' health disparities. In contrast, in Currie and Schwandt's study (2016a, 2016b), we have observed a tremendous increase in mortality inequality among older adults.

Certainly, one could argue that experiencing adverse health conditions during early life may accumulate and lead to poorer outcomes in later stages (life course perspectives). However, this explanation does little to clarify the widening mortality inequality observed among older adults while the mortality disparities in early life are narrowing. In fact, the life course perspective, including the cumulative advantage/disadvantage theory, tends to give less emphasis to the persistence of health and mortality inequality, as well as the divergent trends observed across different stages of the life course. Fundamental cause theory (FCT) argues that inequality increases when medical knowledge, innovation, or the ability to control diseases increasingly benefits those with more resources. This perspective helps elucidate the enduring nature of mortality inequality over time. However, it does not fully account for the universal expansion of health inequalities, irrespective of the level of welfare policies, nor does it account for the divergent trends observed across different age groups.

Evolution in Disease Patterns and Shifting SES Inequalities in Health/Mortality

The two paradoxes about health disparities point to a significant gap in the literature. New mechanisms and explanations should be introduced to advance the understanding of the changes in health inequalities over time. Previous empirical studies and theoretical frameworks mainly attribute the widening health inequalities to the changes in overall SES inequalities but pay less attention to changes in disease patterns. Link and Phelan (1995) propose that SES is a fundamental social cause of health. As mentioned above, accessing and effectively utilizing resources that can help to “avoid risks or minimize the consequence of disease” is essential to

understanding health inequalities. Yet not all diseases are associated with the same degree of disparities. According to Link and Phelan (1995), when the causes, risk factors, preventions, and treatment of the diseases are unknown or unavailable to anyone, the health inequalities are minimal because socioeconomic resources are of little use. However, when new knowledge, medical interventions, or protective factors emerge, those with more resources have advantages in getting access to this knowledge, prevention, and treatment and will benefit first and most, thereby resulting in widening health inequalities (Phelan et al., 2004). In other words, the expanded capacity to control diseases and death creates health disparities (Phelan & Link, 2005).

Based on Fundamental Cause Theory, the association between SES and health is reproduced and endurs via the replacement of risk factors, disease patterns, and protective factors dominating at that time (Link & Phelan, 1995; Phelan et al., 2004). Clouston et al. (2016) extended the FCT by proposing a four-stage ideal type about the rise and fall of mortality inequality: (1) when there are few treatments for a disease, then there are few disparities; (2) but once there are new treatments available, those with more “flexible resources” get them first and hence the growth of disparities; (3) then, when treatments become more and more universally accessible, the disparity narrows and (4) finally could disappear. This four-stage ideal type (as displayed in Figure 2.3) helps us understand the nature and dynamics of mortality inequality for different diseases or different stages of a disease’s development. Health inequalities endure over time through “a continuing stream of widening, and newly emergent disparities that counter the effects of other diminishing disparities” (Miech et al., 2011). When one mechanism/mediating factor declines or is addressed, alternative factors emerge and become more prominent (Phelan et al., 2004). The widening disparity in overall mortality is not the result of uniform growth in all-

causes of death, but rather because the widening disparities in some diseases outweigh the narrowing disparities in others (Clouston et al., 2016; Miech et al., 2011).

[--Figure 2.3 about here--]

However, what is not well addressed in Fundamental Cause Theory (Miech, Pampel, Kim, et al., 2011a) is how the “continual stream of widening and narrowing inequalities” is embedded in the historical context. The “continual stream of widening and narrowing inequalities”—i.e., shifts in health disparities across different types of diseases—does not happen in a vacuum but instead follows the shift in disease patterns in real historical context (referred to as the epidemiological transition). The trends in health inequalities change alongside shifts in disease patterns and the replacement of relevant risk factors over time. To understand the changes in health inequalities, we must focus on changes in disease patterns—the epidemiological transition—in historical context.

Overall mortality levels declined and shifted tremendously over the past several centuries. In 1971, Omran first proposed the Epidemiological Transition Theory as the process of decline in infectious diseases and then gradually replaced by degenerative diseases (Omran, 1971). According to Omran(1971), the shift in disease burden was comprised of three stages. The first stage, the “age of pestilence and famine,” lasted until middle of 19th century, during which time mortality levels were high and fluctuating. The second stage, the “age of receding pandemics” ended in the mid- 20th century, saw overall mortality decline primarily due to lower mortality from infectious and parasitic diseases (thanks to the improvement in medical technologies like vaccines, sanitation, and standard of living) (Omran, 1971). As more people survive to adulthood, they face an elevated risk of degenerative disease, leading to Stage 3 of the transition: the “age of degenerative and man-made diseases” (Omran, 1971). Then, a fourth stage

of epidemiological transition was proposed in the mid-1980s, arguing that since the 1960s, major degenerative diseases (especially heart disease, stroke, and some kinds of cancer) have experienced considerable improvements in medical technology. The utilization of treatment (e.g., blood pressure control, coronary artery bypass surgery, etc.), and lifestyle changes (e.g., smoking, exercise, and diet) led to the delayed onset and progression of these degenerative conditions—hence Stage 4 of the transition: the “age of delayed degenerative diseases” (Olshansky & Ault, 1986).

The trends and patterns of mortality inequalities evolved along with the shifts in disease patterns noted above, via the emergence of medical innovations for prevention and treatment, and the replacement of risk factors and protective factors dominating at that time.

The Decline of Infectious Diseases

The disease burden of infectious diseases has declined over time largely due to various factors. These include the discovery of microorganisms in the 19th century, the improvements in “sanitation and hygiene, the discovery of antibiotics, and the implementation of universal childhood vaccination programs”(CDC, 1999), as well as the improved nutrition and rising standard of living (R. G. Rogers & Hackenberg, 1987). Here, I provide only a few examples to illustrate this point. During the 1930s-1950s, huge achievement has been achieved in “sewage disposal, water treatment, food safety, organized solid waste disposal, and public education about hygienic practices (e.g., food handling and handwashing)” in the United States (CDC 1999, p.637). In the 1940s, penicillin was developed and widely used in treatment for infectious diseases(CDC, 1999).

The introduction of vaccines largely eliminated deaths from diphtheria, tetanus, poliomyelitis, smallpox, measles, mumps, rubella, haemophiles influenzae type b meningitis (CDC, 1999). The vaccine for smallpox was recommended for the US public since the 18th century, and by 1977, smallpox had been even eliminated. The national efforts to promote vaccines among children started in 1955 with the polio vaccination. In 1963, the measles vaccine became available to the public and the deaths declines since 1965. The widespread implementation of pneumonia vaccine for infants and children (pneumococcal conjugate vaccine, PCV7) was licensed in 2000 (Musher et al., 2022; Pulido & Sorvillo, 2010), and leads to tremendous reduction in pneumococcal diseases in children and adults as well (might because reduction in transmission from children to adults)(Cillóniz et al., 2016). The Influenza vaccine emerged in 1985, even though the vaccine did not appreciably alter mortality rates (Doshi, 2008a).

The universal access to many vaccines, as well as sanitation, improved nutrition, and better living conditions, primarily reduced or even eliminated the deaths from infectious diseases. The overall mortality from infectious diseases has declined rapidly(Armstrong et al., 1999; Doshi, 2008b; Feigenbaum et al., 2019). The health inequalities from many infectious diseases have been also vastly reduced(Link & Phelan, 1995; Phelan et al., 2004).

The Evolution of Disease Burden from Infectious to Degenerative Diseases

As more people survive to adulthood, they face an elevated risk of degenerative disease; as a result, degenerative diseases gradually become the major cause of death (*Stage 3: the Age of Degenerative and Man-made Diseases*) (Orman, 1971). Since the 1960s, mortality rates from major degenerative diseases declined steadily (*stage four: the Age of Delayed Degenerative*

diseases). The decline in mortality from degenerative diseases is driven by the improvement in prevention/treatment for major degenerative diseases during the past decades.

Medical innovations related to cardiovascular diseases serve as the prime example. The mortality rate from cardiovascular diseases declined by over 50% from 1940 to the 1990s and continued to decline by about 20% from the 1990s to the 2010s (Sidney et al., 2016a). The treatment of heart diseases has improved largely since the 1960s. The first coronary care units (CCU) were built in the United States in 1963 (DAY, 1963), and became the most important milestone in the treatment of acute myocardial infarction (AMI). More options for medical treatment and new drugs also emerged in the second half of the 21st century. The development and widespread use of Angiotensin-converting enzyme (ACE) inhibitors and a series of updated inhibitors (during the 1970s-1980s), blood pressure lowering drugs (during 1950s to 1970s), aspirin (in the 1970s), statins to control cholesterol (1980s to 2000s), beta-blockers to manage abnormal heart rhythms (1990s and 2000s) all contributed to a reduction of death from cardiovascular diseases (Chang & Lauderdale, 2009; Elwood, 2006; Miner & Hoffhines, 2007; Skinner & Staiger, 2005). Cardiovascular surgery has also steadily improved since the 1950s (Cooley & Frazier, 2000). The first "modern" coronary artery bypass grafting (CABG) was performed in 1964(Head et al., 2013). The techniques continue to advance thereby improving myocardial protection: for example, the use of grafts, minimal invasive & hybrid revascularization, and use of cardiac catheterization have improved steadily since the 1970s(Bourassa, 2005; Head et al., 2013; Melly et al., 2018).

Similar dramatic improvements over time have been seen for cancer screening and treatment. The overall cancer mortality rate declined by about 1% per year since 1990, with an average of about 2% decline in lung cancer, colorectal cancer, breast cancer, and prostate cancer

(Byers, 2010). The progress in medical knowledge about diet and lifestyle contributes to the decline in cardiovascular diseases, as well as many kinds of cancer and other chronic conditions(e.g., diabetes, lung conditions, etc.). The Surgeon General's Report on smoking was released in 1964(The Council of State Governments, 2009), warning of smoking's consequences on health especially lung cancer. Since then, cigarette consumption declines largely(Centers for Disease Control and Prevention, 1999a). Following the Surgeon General's Report, more actions in tobacco control policies also helped reduce cigarette consumption(Levy et al., 2016; Moritsugu, 2006).

The changes in smoking behavior directly impact mortality trends in degenerative diseases, especially lung cancer (Giovino, 2002) and chronic obstructive pulmonary disease (COPD). Trends in lung cancer closely follow the decline in smoking (Giovino, 2002), with a delay of about 20 years(Rubin et al., 2014). The male lung cancer mortality peaked in 1990 and then dropped by 2% per year in 1990s. female lung cancer rate continued to increase in the 1990s peaking in the early 2000s, and then declined (Byers, 2010), which might be caused by the gender differences in smoking cessation rate (Osler et al., 1999). Stroke mortality has declined by more than 75% from the 1950s to 2008 (Towfighi & Saver, 2011). The control of hypertension, hyperlipidemia, and tobacco use has made significant contributions to the decline in mortality rates, while improved acute stroke care has also played a substantial role, albeit to a lesser extent (Lackland et al., 2018; Towfighi & Saver, 2011).

The changes in diet and physical activities also play an important role in the decline of cardiovascular diseases, cancer, COPD, diabetes, and many other kinds of chronic diseases. Until the first half of the early 20th century, high calorie diets were still deemed beneficial, especially to help recovery from infectious diseases (Kritchevsky, 1998). The American Heart Association

published the first recommendations regarding diet in 1961(Page et al., 1961), recommending less intake of saturated fat and cholesterol to reduce the risk of heart attack and stroke (Walker, 1977). In 1988, the Surgeon General's Report on Nutrition and Health was published, stating that diet plays an important role in five of the leading causes of death in the US (coronary heart disease, stroke, atherosclerosis, diabetes, and some types of cancer)(Nutrition Policy Board, 1988). This report was another landmark paralleling the Surgeon General's Report on smoking in 1964. In 1997, the American Diabetes Association (ADA) provided guidelines and recommendations for lifestyle interventions in the prevention and management of diabetes, highlighting lifestyle, diet, nutrition, and physical exercise in reducing the risk of diabetes.

The improvements in diagnostic and treatment technology like screening techniques also emerged during the past decades. The colon cancer death rate peaked in 1985 in the US and then started to decline, which could be attributed to colorectal cancer screening techniques since 1980s(Byers, 2010; Wang et al., 2012). Of course, the changes in lifestyle and diet, and the use of non-steroidal anti-inflammatory drugs (e.g., aspirin) may also have contributed (Byers, 2010). The breast cancer mortality rate also declined since 1990 due to the use of mammographic screening for earlier diagnosis and better treatment after the diagnosis (Byers, 2010; Gold et al., 2012).

The prevention and treatment for COPD and diabetes have also improved in the last decades. In the early 1960s, inhalers and mechanical ventilators became available for COPD treatment. In the 1980s, when home oxygen therapy made it possible for COPD patients to get oxygen outside the hospital, it soon (Kvale et al., 1980; Stuart-Harris et al., 1981), became the main treatment for advanced COPD patients(Petty, 1990). Drugs that help restore pulmonary function (e.g., steroids) are more and more widely used in the 1990s(Petty, 2006).

For Diabetes, a series of studies revealed that intensive control of glucose levels, blood pressure, and lipid levels could largely reduce the complications of diabetes mellitus in 1990s (Diabetes Control and Complications Trial Research Group, 1993; Gæde et al., 2003; UK Prospective Diabetes Study (UKPDS) Group, 1998). Newer improvements in insulin to control chronic complications were developed since 1990 (Karamanou, 2016). In 1997, American Diabetes Association (ADA) provides guidelines and recommendations for lifestyle interventions in the prevention and management of diabetes, highlighting lifestyle, diet, nutrition, and physical exercise in reducing the risk of diabetes (American Diabetes Association, 1997). Several studies have reported notable improvements in national diabetes care since the 1990s (Ali et al., 2013; Raghavan et al., 2019). Over the past two decades, there has been a significant decline in diabetes-related complications (Gregg et al., 2014; McEwen et al., 2011), as well as a reduction in mortality rates associated with diabetes (Gregg et al., 2018; Raghavan et al., 2019).

Consistent with the Fundamental Cause Theory, the improvement in prevention/treatment technologies for degenerative diseases and lifestyles knowledge are associated with the increase in health inequalities. At the early years of the *“Age of Degenerative and Man-made Diseases”*, few SES inequalities are observed for major degenerative diseases. For example, one epidemiological study proposed that “there seems to be little evidence of a systematic relationship between social class and coronary disease” during the 1960s (Feldman et al., 1989; Phelan & Link, 2005; Syme et al., 1964). Along with the emergence of these medical innovations (prevention/treatment) for degenerative diseases especially after the 1960s, diseases and deaths from heart disease, stroke, and some sorts of cancer have been more preventable and treatable (Phelan & Link, 2005). Meanwhile, mortality disparities from degenerative diseases increased. Mortality inequality from heart diseases has vastly increased since 1970 (Feldman et

al., 1989; Phelan & Link, 2005; Singh et al., 2015). For example, studies found that higher SES groups used to have higher cholesterol levels in 1960s and 1970s because they had richer high-cholesterol food. But the association reversed after the emergence of statins (statins were very expensive)(Chang & Lauderdale, 2009). Singh found that individuals in the most deprived counties had 11% higher CVD mortality in 1969, but the number increased to 40% higher mortality in 2007-2011(Singh et al., 2015).

The SES inequality in cancer mortality has experienced similar reversals. Whereas during the 1950s, 1960s, and 1970s, individuals in low SES countries had lower cancer mortality rates by the late 1970s, patterns reversed so that cancer mortality was almost 20% higher for the low SES in 1997(Singh, Miller, Hankey, et al., 2002). The rising inequality is mainly driven by lung, colorectal, and prostate cancer mortality (Phelan & Link, 2005; Singh, Miller, & Hankey, 2002b; Singh, Miller, Hankey, et al., 2002). During the 1950s and 1960s, lung cancer mortality was higher among those with higher SES (Phelan & Link, 2005) but disparities narrowed over time and then started to reverse and widen during the 1970s and 1980s for men and 1990s for women(Singh, Miller, & Hankey, 2002a). These reversals in mortality inequality are associated with changes in smoking beliefs and behaviors in the US (Link, 2008; Phelan et al., 2010). Similar patterns have been found between screening techniques and mortality inequality for colorectal cancer(Wang et al., 2012),

Previous studies have demonstrated that unequal access to prevention/treatment resources in breast cancer, COPD, and diabetes is linked to disparities in mortality rates. For instance, lower rates of mammograms have been associated with an increased risk of developing breast cancer (Lundqvist et al., 2016; Miranda et al., 2011). Similarly, disparities in the distribution of smoking, exposure to harmful particles, and access to therapy techniques have been linked to

inequality in COPD mortality (Gaffney et al., 2021; Pleasants et al., 2016), and making COPD one of the most strongly SES-related disease(Pleasants et al., 2016). Additionally, the unequal utilization of blood sugar management techniques and the lifestyle (e.g., diet and exercise) has been associated with disparities in diabetes-related mortality (Barnard-Kelly & Chernaiovsky, 2020; Lutfey & Freese, 2005; Ricci-Cabello et al., 2010). However, the temporal relationship between emergence of these medical innovations and the changes in mortality inequalities has been less studied.

The Most Recent Evolution of the Epidemiological Transition

The success in reducing deaths from infectious diseases and the delayed onset of degenerative diseases led to a pervasive climate of overconfidence--the illusion of “disease eradication” in the 20th century(Snowden, 2008). However, the epidemiological transition should not be seen as static stages but rather as an evolving process (Bühler et al., 2008; Olshansky et al., 1998; J. Rogers & Nelson, 1997). Since the 1980s, the progressive and triumphalist assumption of the epidemiological transition--about the eradication of infectious disease--did not allow for the emergence of new kinds of infectious diseases in the future.

The emergence of new infectious diseases such as HIV in the 1980s, and the reemergence of some old infectious diseases (e.g., drug-resistant strains) led to an overall increase in infectious diseases in the 1980s and 1990s(CDC, 1999), and suggests how fragile advances in health remains for human society(Snowden, 2008). In 1992, the Nobel Prize for Medicine winner, Joshua Lederberg warned that “age of euphoria was over”(Snowden, 2008), and that infectious diseases “will not be conquered during our lifetimes ... We can also be confident that new diseases will emerge, although it is impossible to predict their individual emergence in time

and place”(Davis & Lederberg, 2000). This seems to be a prophecy. In 2020, Covid-19 caused staggering numbers of deaths in the US and worldwide. Average US life expectancy dropped by about 3 years, creating even larger SES and racial disparities (Davis & Lederberg, 2000).

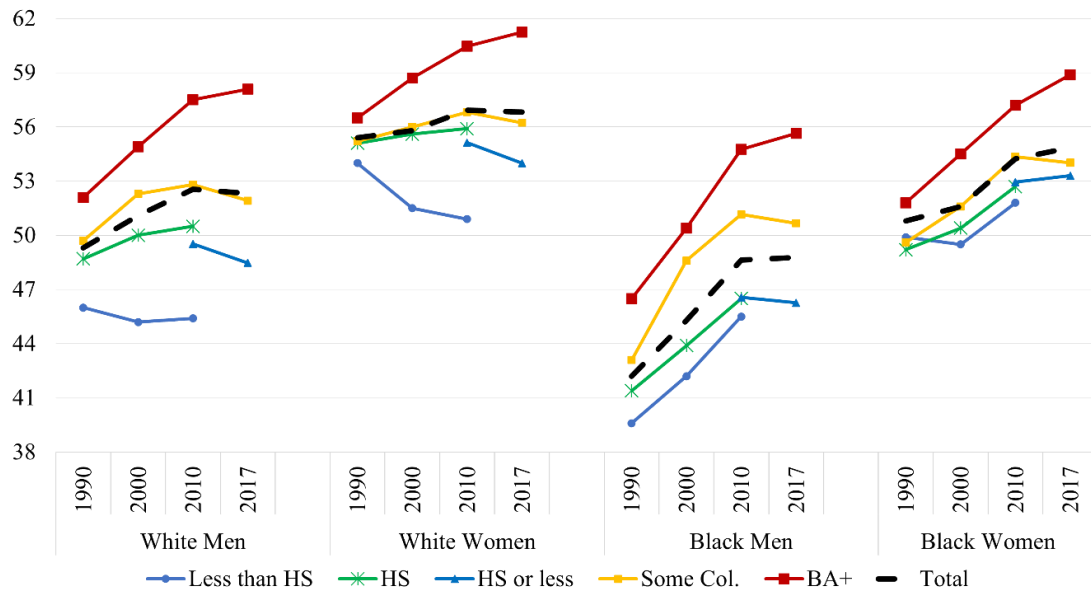
Meanwhile, the declines in heart disease had slowed in the United States since the 1980s(Jones & Greene, 2013). The decline in cardiovascular diseases plateaued or even reversed since 2000 (McClellan et al., 2019; Sidney et al., 2016b). At the same time, studies show that obesity has increased by 50% since the 1960s and has been linked to the increase in risks of cardiovascular diseases and diabetes (Olshansky et al., 2005). The prevalence of diabetes morbidity has been rising dramatically in recent decades, both among older adults, adults, and children (Jones & Greene, 2013; Ludwig & Ebbeling, 2001). These more recent changes in disease patterns suggest a more complicated future that does not necessarily fit the overly optimistic model of the epidemiologic transition.

To sum up, the SES inequality in mortality and health has been rising, and most studies attribute it to the widening overall SES inequality. But existing studies have made little progress in explaining the increase in mortality/health inequalities as the overall mortality declines, even in societies with generous welfare policies and relatively equal income redistribution. It also remains unexplained why mortality inequalities present diverging trends across different age groups/life course stages. This study attempts to fill these two gaps by providing a new theoretical framework, which links fundamental cause theory to the shifting disease patterns under the evolution of the epidemiological transition. The key principle of this framework is that the shifting disease patterns as well as the emergence of medical innovations (e.g., prevention/treatment technology, medical knowledge about lifestyles, public health intervention, etc.) affect the trends in health inequalities, independent of the changes in SES inequality. The

degenerative diseases outpaced infectious diseases in contributing to overall inequality. In that case, SES inequality in health will increase no matter the extent or intensity of welfare policies or the level of SES inequalities in a country. Even worse, the countries with more advanced welfare policies may go further in the Epidemiological Transition, making the inequalities from degenerative inequalities more influential to the overall health or mortality inequality. Also, considering that degenerative diseases affect adults and older adults most, with minor influence on early-life and young adolescence, it could be imagined that the widening mortality and health inequalities are more salient among later life than earlier life stages. Linking the changes in disease patterns could help explain the two paradoxes mentioned above, move forward the health inequality theory, and enrich the policy implications of health equity.

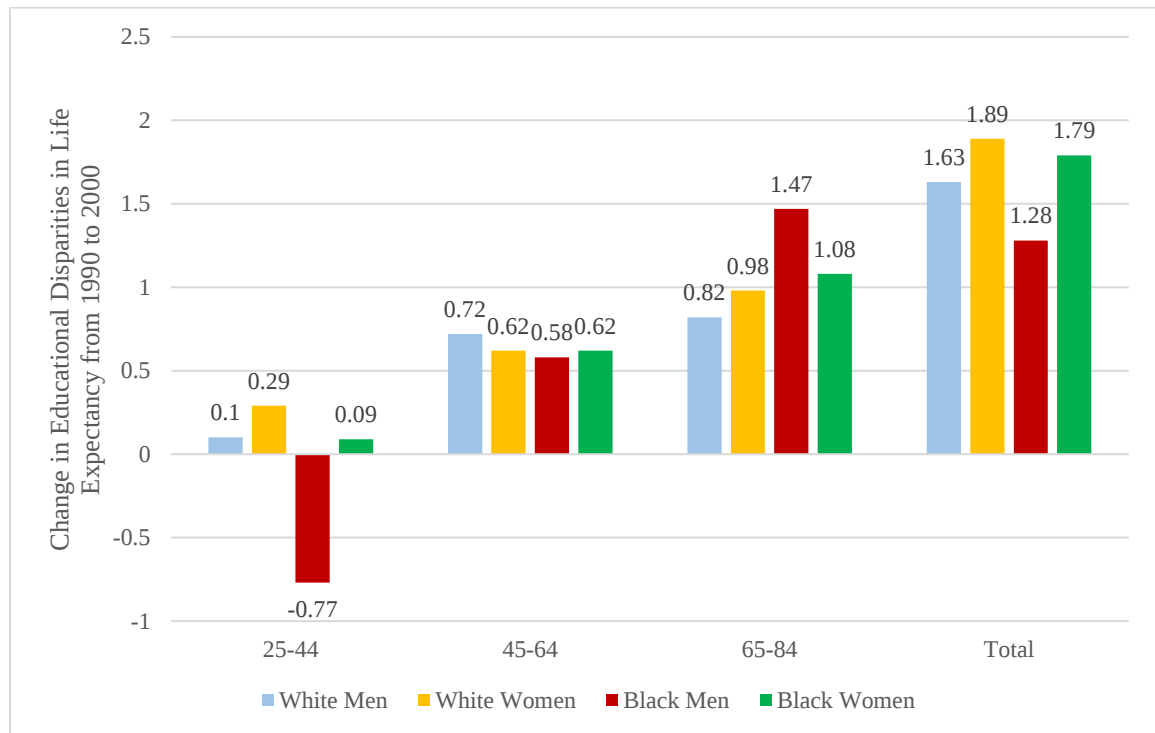
Tables and Figures

Figure 2. 1 The Educational Differences in Life Expectancy at Age 25 In The US, 1990-2017.



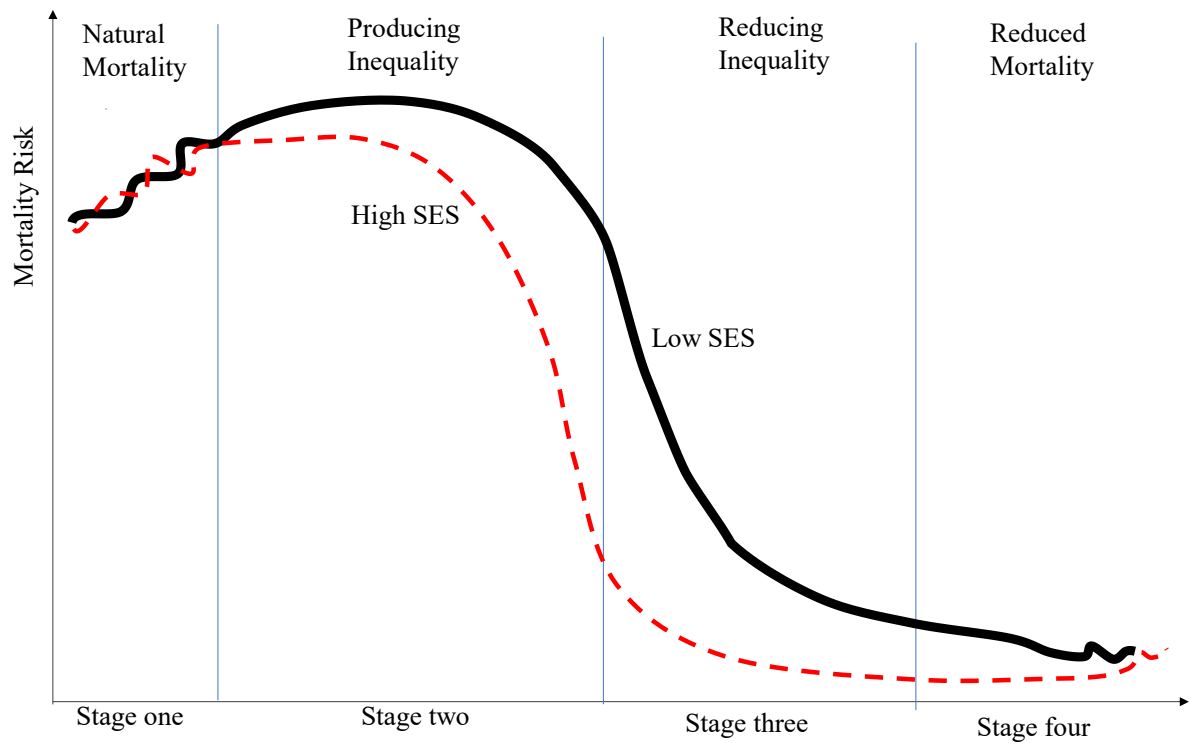
Notes: adapted from Sasson, I. (2016). Trends in Life Expectancy and Lifespan Variation by Educational Attainment: the United States, 1990–2010. *Demography*, 53(2), 269–293. Sasson, I., & Hayward, M. D. (2019). Association Between Educational Attainment and Causes of Death Among White and Black US Adults, 2010-2017. *JAMA*, 322(8), 756.

Figure 2. 2 The Changes in Educational Differences of Life Expectancy at Age 25 (Any College vs. HS or Less) From 1990 to 2000.



Notes: adapted from Meara, E. R., Richards, S., & Cutler, D. M. (2008). The gap gets bigger: Changes in mortality and life expectancy, by education, 1981-2000. *Health Affairs*, 27(2), 350–360.

Figure 2. 3 The Ideal Type of Four Stages of Mortality/Health Inequality



Notes: Adapted from Clouston, S. A. P., Rubin, M. S., Phelan, J. C., & Link, B. G. (2016). A Social History of Disease: Contextualizing the Rise and Fall of Social Inequalities in Cause-Specific Mortality. *Demography*, 53(5), 1631–1656. <https://doi.org/10.1007/s13524-016-0495-5>

Chapter Three: The Theoretical Framework

To better understand the two paradoxes in mortality inequality trends (widening mortality inequalities and diverging mortality inequalities across life course stages), this study provides an alternative theoretical framework—which is referred to as ***epidemiological transition and shifting health/mortality inequalities***—for understanding historical changes in health inequalities. This framework relies on Fundamental Cause Theory (FCT) but links it to the historical context of the epidemiological transition. Here, the epidemiological transition refers to the evolution of disease patterns in real historical contexts, from mostly infectious diseases to mostly degenerative diseases (described in classical Epidemiological Transition Theory of Omran, 1971); it also encompasses the delayed onset of degenerative diseases up until recent decades which have seen a slowdown in the decline in heart disease and the reemergence of some infectious diseases. Based on the Fundamental Cause Theory, health/mortality inequality endures over time through the shifts in health disparities by types of diseases as a “continual stream of widening and narrowing inequalities” (Miech, Pampel, Kim, et al., 2011a). I propose that the “continual stream of widening and narrowing inequalities” does not happen in a vacuum, but under shifts in disease patterns alongside the epidemiological transition.

Existing studies mainly focused on the rise and fall in social and economic inequalities in explaining health inequalities. I emphasize the importance of shifts in disease patterns and progress in medical innovation in understanding the changes in mortality/health inequality, independent of the changes in social/economic inequalities. **The core argument in this conceptual framework is that: the shifting disease patterns as well as the emergence of medical innovations (e.g., prevention/treatment technology, medical**

knowledge about lifestyles, public health intervention, etc.) affect the trends in health inequalities, independent of the changes in SES inequality. As universal access to vaccines, sanitation, and better living conditions primarily reduced or even eliminated the deaths from infectious diseases, the mortality inequalities from infectious diseases have been vastly reduced. However, the SES inequalities from degenerative diseases have increased along with the emergence of these medical innovations for degenerative diseases since the 1960s (the Age of Delayed Degenerative diseases). As a result, degenerative diseases outpaced infectious diseases contributing to overall patterns of inequality. In the following, I will summarize the propositions of my theoretical framework, and the hypotheses that are used to test the propositions.

The fundamental mechanisms of this conceptual framework are summarized in the illustration graph below (see Figure 3.1). This framework tries to explain how social changes affect the trends in health inequalities over time. Previous studies mainly focus on the pathway on the right side of the graphs (social change→ rise and fall of social/economic inequality→health inequality). This framework adds one more pathway (social change→shifting disease patterns & medical innovation→health inequality). The Epidemiological Transition theory links the *mortality trends* to changes in *disease patterns* (Omran, 1977). In this study, I add *mortality inequality* into this framework, and emphasize that the social changes in the past decades led to overall mortality decline and changes in the distribution of disease, which affects the shifts in health/mortality inequalities by types of diseases and finally shapes the overall mortality inequality and health inequalities we observed.

Of course, this framework does not negate the significance of socioeconomic factors. On the contrary, the changes in disease patterns and medical innovation result from complex

social, economic, medical, and environmental changes. The transition in mortality inequality is the consequence of the long-term and complex socioeconomic transition, and it also provides an underlying context for health inequality research. Moreover, this transition in mortality inequality interacts with other health inequality explanations, like changes in broader socioeconomic inequalities and life course processes. They do not conflict with each other. In contrast, they are compatible.

[--Figure 3.1 about here--]

Epidemiological Transition and Shifting Health/Mortality Inequalities

1. *The SES-health link is maintained through the critical role of “flexible resources.”* Based on the Fundamental Cause Theory, SES is a fundamental social cause of health because people of higher SES have more flexible resources (money, knowledge, prestige, power, and beneficial social connections). These resources allow them to learn more about diseases, avoid the risk factors for poor health, gain access to the factors protecting health, and effectively access treatment to limit the consequences of disease (Link & Phelan, 1995).
2. *For each disease, health inequality rises when newfound knowledge and technology allow for more capacity in the prevention/treatment of the disease (as those with resources are the first to take advantage of the innovations) and declines when these innovations become more universally available.* New inequalities are created when newfound knowledge and technology (e.g., diet and nutrition, prevention) allow more control of diseases. The inequalities will decrease when the “health-beneficial knowledge and technology become more universally accessible and evenly distributed throughout the population” (Clouston et al., 2016).

3. *The overall health inequalities endure because as inequality in some diseases decline/diminishe, the inequality expands in other types of diseases where innovations emerge (as those with resources are the first to take advantage of the innovations).*

Mortality has declined for hundreds of years due to modifications over time of relevant mechanisms of diseases and risk factors. However, SES inequalities persisted over time despite changes in disease patterns and risk factors. As a result, at any given time, social inequalities have been widening for some diseases but narrowing for others. The widening disparity in overall mortality is caused by “a continual stream of widening and newly emergent disparities counteract the effects of any diminishing disparities”(Richard Miech et al. 2011).

These three propositions above have been systematically tested in previous studies (Clouston et al., 2016; Miech et al., 2011; Phelan et al., 2004). In this research, I only reemphasize one testable hypothesis:

- *Hypothesis 1.* SES disparities in all-cause mortality have increased over time, but this masks divergent trends across causes of death. Different diseases (causes of death) show divergent magnitude and direction in mortality inequality trends. At any given time, SES inequalities in mortality are not changing uniformly but narrowing for some diseases while widening for others.

4. *This shift of inequality across types of diseases is not random but is embedded in shifts in disease patterns under the epidemiological transition.*

4a. *Infectious diseases have declined with universal access to technologies like vaccines, sanitation, and standard of living. As a result, mortality inequalities from infectious diseases have been vastly reduced.*

4b. As more people survive infectious diseases and live to older ages; they face an elevated risk of degenerative diseases. However, as newfound medical knowledge and technology continue to emerge, the disparities have widened as individuals with valued resources benefit more than others. As a result, degenerative diseases outpaced infectious diseases in contributing to overall patterns of inequality.

Health and mortality inequality shifted alongside the declines in overall mortality and the modification of relevant mechanisms and risk factors over time. As deaths from infectious diseases largely declined, degenerative diseases become the major killer (Stage 3: the Age of Degenerative and Man-made Diseases) (Omran, 1971). From the 1960s, improvements in medical technology (e.g., blood pressure control, coronary artery bypass surgery) and lifestyle changes (e.g., smoking, exercise, and diet) reduced the mortality risks from major degenerative diseases (including heart disease, stroke, and some kinds of cancer, and some other chronic diseases)—the Age of Delayed Degenerative diseases (Olshansky & Ault, 1986). Meanwhile, SES inequality has increased along with these innovations (prevention/treatment) for degenerative diseases. The following hypotheses are constructed to test proposition 4 above:

- *Hypothesis 2.* The mortality inequality from infectious diseases has been largely reduced.
- *Hypothesis 3.* Since the 1960s, mortality inequality from degenerative diseases has widened and now dominates mortality inequality. The degenerative diseases have outpaced infectious diseases in contributing to overall patterns of inequality.

Degenerative diseases are a diverse set of diseases that have each followed a unique trajectory of innovation in diagnostics and treatment. As stated above, the widening mortality

inequality from degenerative disease is attributed to widening mortality inequalities from cardiovascular diseases (CVD), cancer, as well as many other degenerative diseases.

- *Hypothesis 4a.* The mortality inequalities from cardiovascular diseases (heart diseases and stroke, etc.) have been widening alongside the improvements of treatment techniques and changes in lifestyles.
- *Hypothesis 4b.* The mortality inequalities from cancer have been widening alongside the improvements of changes in lifestyles like smoking behavior (especially for lung cancer) and medical techniques like the diagnosis and treatment in Colorectal and prostate cancer.
- *Hypothesis 4c.* Not just CVD and cancer, the mortality inequalities from other degenerative diseases (e.g., COPD and diabetes, etc.) also increased in the second half of 20th century alongside the changes in lifestyle and prevention/management knowledge.

Here, "temporal" linkage between innovations and inequality serves as the essential foundation for testing these hypotheses: inequality will widen after a significant change/innovation has occurred in the detection, diagnosis, or treatment of a disease, and it will follow different patterns depending on the disease².

Diverging Trends in health/mortality Inequality Across the Age Groups

This framework also helps to explain the diverging health inequality trends across age groups/life course stages. The shifts in disease patterns from infectious diseases to

² One thing to be noted is that the crucial role of cardiovascular diseases (CVD), cancer, and other degenerative diseases in contributing to overall mortality inequality stems not from their high death rates but from advancements in prevention and treatment innovations. Of course, medical innovation always particularly targets diseases with high mortality rates.

degenerative diseases change the distribution of deaths across age groups/life course stages, making the trends in mortality inequality differ by age groups (early life vs. mid-life vs. later life).

5. *The shift in disease patterns differs by age groups/life course stages, making the trends in mortality inequality differ by life course stages. Specifically, the receding of infectious diseases and an increase in degenerative diseases has led to a redistribution of disease burden and death from young to older ages (Omran, 1977). Because degenerative diseases need to accumulate through the life course, and the illness and death are much more concentrated in older age groups, there should be a widening mortality/health inequality in later life with only a minor increase at younger ages.*

Based on Epidemiological Transition Theory, the first three stages of transition are characterized by “a substitution of one set of diseases to another”, but the fourth stage is characterized by “a substitution of the ages at which degenerative diseases tends to kill” (Olshansky & Ault, 1986, p.374). It justifies the emergence of a fourth stage because the “age distribution of mortality from degenerative diseases has shifted progressively toward older ages” (Olshansky & Ault, 1986). The medical innovations and changes in health behavior do not eliminate (but just delay) death from degenerative diseases (as shown in Figure 3.2 below).

[--Figure 3.2 about here--]

In other words, medical innovations and changes in health behavior slow the aging process, and "the timing of aging-related disease and disability incidence" has been pushed closer to the end of life (Levine & Crimmins, 2018). The later age of onset, slower progress, and lower severity in degenerative diseases have also been documented a lot in “compression of morbidity” theory (Fries, 1980; Fries et al., 2011) and “dynamic equilibrium”

theory(Manton, 1982). Empirical studies have also found that the biological age of the US population has become younger between 1988 and 2010—also known as "the 60 is the new 50"—especially among older adults(Levine & Crimmins, 2018).

Currie's study (2016a, 2016b) pointed out that the mortality inequalities have been narrowing in early life and widening in later life. This theoretical framework goes further and proposes that there is a life course pattern or age pattern in the mortality inequalities trends: for successive periods, as the onset of degenerative diseases is delayed or moved progressively toward older ages, the critical importance of "flexible resources" in producing mortality inequalities is also delayed to older ages. As a result:

6. *The age distribution of mortality inequality also shifts progressively toward older ages. In other words, for successive times, the distribution of mortality inequality will move toward older ages of later life.*

This leads to the following hypotheses about diverging trends in mortality inequality across the age groups/life course stages:

- *Hypothesis 5.* The increase of mortality inequalities is more salient among later life stages than earlier life stages. This diverging trend in mortality inequality across the life course is because:
 - *Hypothesis 5a.* The inequality in infectious disease diminished at younger ages as vaccines and treatments became more universally available.
 - *Hypothesis 5b.* The widening inequalities from degenerative diseases affect later life mainly, with ~~little~~ less influence on early life.
- *Hypothesis 6.* Age distribution of mortality inequality is shifted progressively toward older ages. Specifically, for successive periods, the percent distribution of SES inequalities in mortality has been more and more concentrated toward later life.

Further, mortality from infectious diseases has been reduced in early life, and degenerative diseases are more related to people's later life. As a result, violent death (e.g., accidents, homicides), and behavioral related death (e.g., suicide and alcoholism) have a greater impact on mortality inequality particularly among young and middle-aged adults. Recent increases in death from suicide, along with the alcohol-related death and death from drug overdose (also known as "death of despair"), leads to great concern in the U.S. middle aged adults, especially for those without a bachelors' degree (Case & Deaton, 2015, 2017, 2020; Committee on Population, & National Academies of Sciences, Engineering, 2021; Knapp et al., 2019; Masters et al., 2017; Monnat, 2017; Sasson, 2016; Stein et al., 2017). This leads to the following hypothesis:

- *Hypothesis 7.* Mortality inequality from social pathology, including violent deaths, suicide, and alcohol/drug-related deaths, increases over time especially among adolescents and younger adults.

Most Recent Evolution of Epidemiological Transition

The Epidemiological Transition theory describes the changes in disease patterns over the past centuries. Since 1980s, the disease pattern in the United States continues to evolve with some new features going beyond Omran's original Epidemiological Transition Theory. Nevertheless, the theoretical framework in this research does not necessarily rely on the original Epidemiological Transition Theory. The core logic of this framework is that health inequalities shift alongside the evolution of disease patterns and the characteristics of the diseases dominating a particular historical time. As we understand more about the characteristics of newly emergent diseases, the framework above helps us predict how these diseases affect the trends of health inequalities.

7. *The characteristics of new emerging diseases (e.g., risk factors and prevention/management knowledge, etc.) provide insight into these diseases' health/mortality inequality trends.*

For example, the reduction in cardiovascular diseases mortality rates had slowed in the United States since 1980s (Jones & Greene, 2013), and the mortality rates even stagnated since 2000 (McClellan et al., 2019; Sidney et al., 2016b). The reasons for the stagnation are not well understood (Acosta et al., 2022; Lopez & Adair, 2019; Sidney et al., 2016), but it appears that the stagnation indicates a slowing of the medical innovation and prevention/treatment tools that previously reduced CVD and exacerbated inequalities.

At the same time, some degenerative diseases like diabetes and chronic liver conditions have been rising dramatically reflecting behavioral changes related to diet, obesity, and alcohol use (Jones & Greene, 2013; Ludwig & Ebbeling, 2001). These new changes lead to the following hypotheses about the patterns of mortality inequality:

- *Hypothesis 8a.* The increase of mortality inequality from cardiovascular diseases (e.g., heart diseases and stroke) has been slowed in the last two decades.
- *Hypothesis 8b.* Meanwhile, the mortality inequality from other degenerative diseases like diabetes and liver diseases has been rising and plays a more and more important role in affecting overall mortality inequality.

The emergence of HIV and reemerging infectious diseases (e.g., drug-resistant strains) lead to an overall increase in infectious diseases in the 1980s and 1990s (CDC, 1999). New treatment and drugs are then invented to address these (Rubin et al., 2010). In 2020, Covid-19 caused staggering death in the US, leading to about a three-year drop in life expectancy (Davis & Lederberg, 2000). The poor population is disproportionately exposed to SARS-CoV-2 due to a lack of remote work opportunities and overcrowded living conditions

(Clouston et al., 2021; Dukhovnov & Barbieri, 2022; Zhang & Kolady, 2022). These new changes could also exacerbate the patterns of mortality inequality:

- *Hypothesis 9.* The mortality inequality from infectious diseases increased during the 1980s and 1990s, and then increased dramatically in the Covid-19 of 2020.

To examine the theoretical propositions and hypotheses, I will utilize county-level US mortality rates (1969-2020) from the National Center for Health Statistics (NCHS). The aim is to investigate trends in county-level socioeconomic inequality in mortality and then examine how mortality inequality evolves with shifts in disease patterns and improvements in medical innovation.

In Chapter Four, the focus will be on analyzing changes in socioeconomic inequality in all-cause mortality over time in the United States. I will use age-standardized county-level US mortality data (1968-2020) categorized by sex and county, along with county-level socioeconomic data, to test changes over time in all-cause mortality inequality. Additionally, exploration will be carried out into how these trends in mortality inequality vary across different age groups.

Moving on to Chapter Five, separate analyses will be conducted based on the cause of death. This approach will enable the presentation of variations in mortality inequality trends related to different types of diseases. Specifically, the shift of mortality inequalities towards chronic diseases over the past several decades will be demonstrated. Furthermore, connections will be established between these trends and key temporal changes in medical knowledge. This will illustrate how the mortality inequalities of various diseases are influenced by the emergence of corresponding medical innovations. For instance, exploration will extend to how advancements in the treatment or prevention of heart diseases, cancer, diabetes, COPD, and other conditions have impacted mortality disparities. The objective here

is to comprehend how well the emergence of medical innovations aligns with changes in mortality inequalities.

Tables and Figures

Figure 3. 1 The Conceptual Model of Disease Pattern and Transition of Mortality Inequalities.

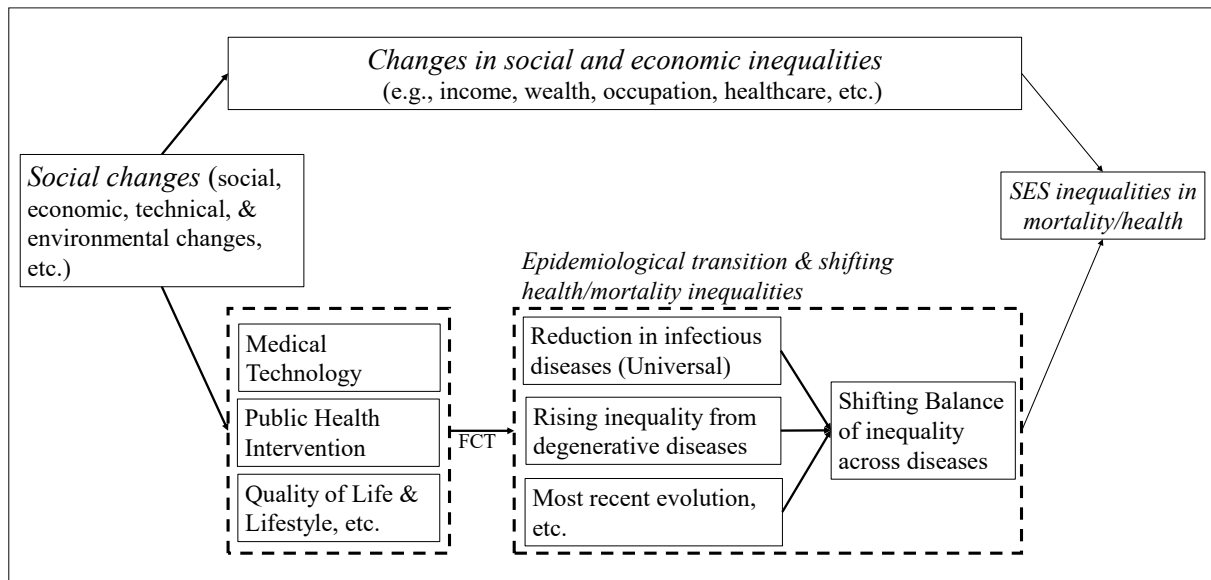
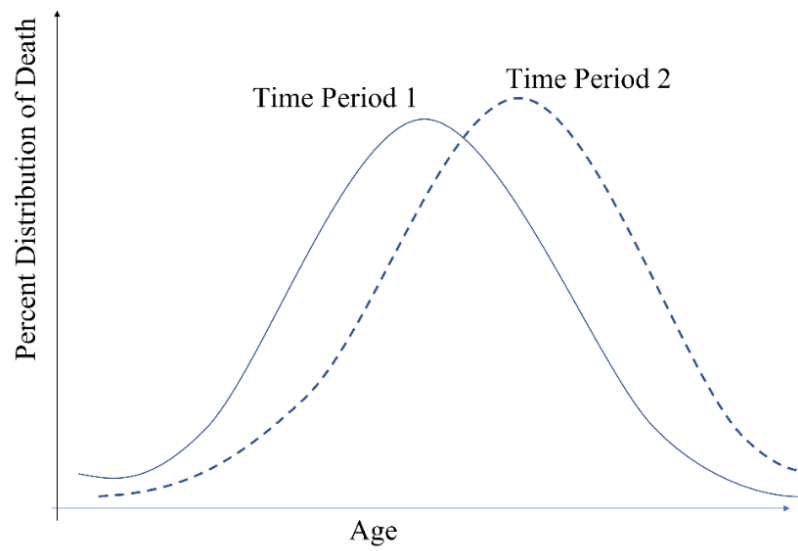


Figure 3. 2 The Percent Distribution of Death Under the Age of Delayed Degenerative Diseases.



Notes: adapted from (Olshansky & Ault, 1986).

Chapter Four: Trends in Mortality Inequalities and Age Group Patterns

Introduction and Background

In the previous chapter, I introduced the theoretical framework for explaining the changes in mortality inequality over time. Beginning in this chapter, I will introduce the empirical data and method in testing the hypotheses proposed in the last chapter. Following Currie and Schwandt (2016), I use county-level US mortality rates (1969-2020) from NCHS (National Center for Health Statistics) to examine the trends of county-level SES inequality in mortality in the United States. The county-level mortality data from NCHS has several advantages.

Firstly, previous studies tracking health inequality trends at the individual level always encountered a methodological concern related to compositional changes in socioeconomic status (SES). For instance, studies have revealed that educational disparities in health have been widening. However, the expansion of education might introduce bias into these findings as the less educated individuals are becoming more and more negatively selected (Bound et al., 2015). To address these potential issues and ensure a more dependable comparison over time, the use of county-level data becomes a promising approach (Baker et al., 2019; Banks et al., 2021; J. Currie & Schwandt, 2016a, 2016b; Schwandt et al., 2021).

Secondly, this data set spans a comprehensive fifty-year period with very little missing information, and it provides a clear picture of local trends across the entire country (much more accurate than state-level data). No other survey data or administrative records currently available offer a comparable extensive temporal scope as this dataset does. Further, a key aspect emphasized in this study is the diverging mortality inequality trends along with

the evolving disease patterns over time. The comprehensive cause-of-death information available in the death records from the National Center for Health Statistics (NCHS) enables the analysis of mortality trends based on specific causes of death, facilitating easy comparisons across time periods.

Using county-level mortality data also has some disadvantages. The first risk is a potential bias of “ecological fallacy”. The ecological fallacy, first proposed by sociologist Robinson in 1950s (Robinson, 2009), suggests that the association between mortality and SES at the county-level may not be equal to the association at the individual level death risk and socioeconomic status. There are two sources of bias. First, the county-level variables measure the different constructs with the individual level variable (Diez Roux, 2015). For example, the individual level SES and neighborhood’s level poverty might have separate and independent influence on people’s health and mortality hazards (Neumark, 2017). Second, in ecological data, the issue of confounding may be more complicated than for individual level data (Diez Roux, 2015; Neumark, 2017). For instance, the variations in geographic environment across US counties, such as the distribution of disease-carrying vectors like mosquitoes and ticks, differences in altitude and oxygen levels, water quality, and food types, may confound the relationship between socioeconomic status (SES) and mortality.

These risk biases are difficult to avoid and challenging to address through statistical tools. However, there is some good news. Recent empirical studies indicate that while the SES inequalities in mortality at the aggregate level and individual level are not identical, they still exhibit a striking similarity (Lokar et al., 2019). This suggests that using aggregated data in health inequality research is acceptable and has been widely employed in this field.

Another potential limitation of using aggregated county-level data is that migration across counties could bias the association between SES and mortality (J. Currie & Schwandt,

2016a, 2016b). To mitigate the bias from migration across counties in this study, I rank all US counties into 100 county bins with equal population size based on their relative SES position (e.g., percent in poverty, education, average income, occupation structure, etc.), with each county bin representing 1% of the whole population. Then, SES inequalities in mortality are measured by comparing the difference in mortality rates between the county bins from the highest and lowest SES ranked counties. I do this ranking separately for each year and always estimate county-level mortality inequality across the 100 county bins. This approach effectively accounts for migration between counties and minimizes any potential bias in the results³ (Baker et al., 2019; J. Currie & Schwandt, 2016a).

In this chapter, I focus on trends over time in SES inequality in all-cause mortality and consider diverging patterns by age groups. Then in the next chapter, I will split the analyses by cause of death to present how trends in mortality inequality vary by different types of diseases. I also link these trends to key historical changes in medical knowledge and lifestyles affecting different diseases.

Data and Variables

Data

Mortality data

Vital Statistics data from the Multiple Cause-of-Death files from the CDC (1969-2020) are used in this paper. The data include all death certificates in the United States, which provides the demographic variables including sex, race, age at death, underlying and multiple

³ Certainly, this approach does not completely eradicate the potential for bias. For instance, if wealthier individuals tend to relocate from lower percentile counties to higher percentile counties, the outcomes could also carry bias: health inequality might appear more pronounced than the actual estimate in the absence of any migration. However, the diverging pattern across diseases and age groups should be less impacted.

causes of death (coded by ICD8, ICD 9, or ICD 10). The 1968-1988 mortality file with geographic variables is obtained from the *Compressed Mortality File* (1968-1988) (National Center for Health Statistics, 2000). Detailed US mortality (all counties) from the years 1989 through 2020 were obtained through the National Center for Health Statistics (Hyattsville, MD) under a data use agreement (National Center for Health Statistics, 1989-2020). The data are secured in the University of Maryland's controlled unclassified information (CUI) environment and accessed from Maryland Population Research Center, University of Maryland.

Population Data

The Multiple-Cause-of-Death file only provides the counts of deaths. To calculate the mortality rate, I use the estimation of the county-level population from National Cancer Institute (Surveillance, Epidemiology, and End Results (SEER) Program Populations, 1969-2020). The SEER database provides the county population size by gender, race, and age groups for each year since 1969⁴. The SEER population estimates use data from Bridged-race Intercensal estimates of 1990-1999 (National Center for Health Statistics, 2003), bridged-race postcensal Vintage 2010 population estimates (2000-2010) (National Center for Health Statistics, 2012), bridged-race postcensal Vintage 2020 population estimates (2010-2020) (National Center for Health Statistics, 2021b), and National Cancer Institute (NCI) standard 200 format files (for population in 1969 to 1989). A modification that SEER database makes is an adjustment for population of Hawaii because Hawaii's population has been thought to be undercounted in previous Censuses. For the details of modifications that

⁴ <https://seer.cancer.gov/popdata/>

NCI has made, see SEER website documentation⁵. All county-level mortality rates are age standardized with 2000 US standard population⁶.

Changes in County Borders and Missing Data

There are 3155 Counties and County Equivalent Entities (FIPS codes) in the 1969-2020 mortality file. The 139 counties with changes to their borders during this period were deleted. For the list of FIPS county codes that changed over time, see Census Bureau website⁷, CDC Compressed Mortality file⁸, SEER website documentation⁹, and some summaries from previous study (David Dorn, 2013)¹⁰. Another 10 counties are excluded because they did not have mortality data in some specific years. Finally, 3006 counties are used, with 149 counties (4.7%) deleted. The data from our study revealed a nearly identical association between SES and mortality compared to Currie and Schwandt's study (2016a, 2016b)—they also used county-level mortality data in 1990 and 2010 but included all counties. This indicates that the missing counties had little impact on our research.

County-level Social Economic Status (SES) Data

Five variables are used to measure county-level SES: (1) county poverty rate, (2) average family income, (3) proportion of population with less than a high school degree, (4) the proportion of population with a bachelor's degree, (5) and occupational structure (the proportion of population with professional and managerial jobs). These county level SES variables come from 1970, 1980, 1990 2000 decennial census (U.S. Census Bureau, 1970-

⁵ <https://seer.cancer.gov/popdata/modifications.html>

⁶ <https://seer.cancer.gov/stdpopulations/stdpop.19ages.html>

⁷ <https://www.census.gov/programs-surveys/geography/technical-documentation/county-changes.html>

⁸ https://www.cdc.gov/nchs/data_access/cmf.htm

⁹ <https://seer.cancer.gov/popdata/modifications.html>

¹⁰ https://www.ddorn.net/data/FIPS_County_Code_Changes.pdf

2000), and American Community Survey (ACS) data (5-year estimates, 2006-2010, 2010-2015, and 2016-2020)(U.S. Census Bureau, 2006-2020). The 2006-2010, 2010-2015, and 2016-2020 American Community Survey (ACS) data are used to estimate the counties' socioeconomic status in 2010, 2015, and 2020, separately. Following previous studies (Clouston et al., 2016), intercensal years are interpolated for each county. All these data could be accessed from the Social Explorer website¹¹.

Variables

Mortality rate

The Mortality rate equals the number of deaths divided by the size of population of each county. Table 4.2 shows that the age standardized mortality rate has declined from about 1580 per 100,000 in 1970 to about 973 per 100,000 in 2010. But the mortality rate increased to about 1125 per 100,000 in 2020 because of the Covid-19 pandemic.

County level SES Index (Percentiles)

To measure county-level SES, I use principal components analysis to construct a county-level SES index(G. K. Singh, 2003). Following previous studies (J. Currie & Schwandt, 2016a), five county-level variables are used: (1) county poverty rate, (2) proportion with more than 12 years of education, (3) proportion with less than 9 years of education, (4) proportion with professional and managerial occupation, (5) the average family income. Table 4.1 provides the descriptive analysis of all five SES variables. Figure 4.1 further presents the association between the SES variables and the constructed SES index

¹¹ <https://www.socialexplorer.com/>

in data for 2020. Not surprisingly, the constructed SES index is highly correlated with all five SES variables.

[--Table 4.1 the descriptive table of SES variables--]

[--Figure 4.1 the scatter plot of SES index and five SES variables in 2020--]

Year. The year ranges from 1969 to 2020. The first available SEER county-level population data is in 1969. 2020 is the most recent mortality data currently available.

Age. The SEER county-level population data provides population data by 5-year grouping of age: 0, 1-4, 5-9, 10-14, ..., 80-84, and 85+. However, the compressed mortality data 1969-1988 provides 5-year grouping of age for those less than 20 but 10-year grouping of age to those 25 and older: 0, 1-4, 5-9, 10-14, 15-19, 20-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85+. I follow this latter grouping of age in mortality data to make the age group consistent across all data sources.

Control Variables.

Following (Caudillo et al., 2022), I also include a series of time-varying demographic variables as additional controls for the changes in basic demographic structure and population density of each county. These variables are: (1) percent of White population, (2) percent of Black population, (3) percent of Hispanic population, (4) percent of population in other race, (5) percent of foreign-born population, (6) population per square mile. Table 4.2 provides the basic descriptives of these control variables.

[--Table 4.2 descriptive table of death/pop, control variables, 1970-2020--]

Analytical Strategy

Descriptive Analysis

Mortality rate and Age standardization

The all-cause mortality rates are age standardized using the US 2000 standard population structure (19 age groups – census P25-1130¹²) (Day, 1996). The annual life expectancy for each county group (SES percentile) is calculated using life table methods based on the age specific mortality rates for each county group (Rowland, 2003).

SES inequalities in Mortality

Following previous studies (Baker et al., 2021; J. M. Currie, 2018; J. Currie & Schwandt, 2016a, 2016b), I rank the 3006 US counties into 100 county groups (i.e., percentiles) based on county-level SES index, making each county group have almost equal population size (around 1% of the US population). This approach rescales the counties into 100 groups (percentiles) based on their relative SES position in the population (ranked from the lowest to the highest), with the 1st percentile (P0-1) representing the poorest county group and the 100th percentile (P99-100) representing the richest county group. I do this separately for each year.

Following Currie and Schwandt (2016a, 2016b), I measure SES inequalities in mortality by comparing the absolute difference in mortality rates between the lowest SES county group and highest SES group. To be specific, this approach fits a linear relationship between mortality and SES rank, net of the control variables, and then compares the predicted

¹² <https://seer.cancer.gov/stdpopulations/stdpop.19ages.html>

mortality for the lowest and highest SES groups on the fitted line (see Figure 4.3 for an illustration). This approach is also known as the slope index of inequality (SII) (Kunst & Mackenbach, 1994), which measures the average predicted decline in absolute mortality rate as one move from the lowest SES rank to the highest (Pamuk, 1985; Renard et al., 2019). The changes in mortality inequality over time would increase if the mortality rates in rich county groups decreased more steeply in absolute terms than poor county groups, or if the mortality rates in rich county groups increase less steeply in absolute terms than the poor county groups (J. Currie & Schwandt, 2016a).

[--Figure 4.2. population size by 100 SES percentiles]

[--Figure 4.3 illustrating graph of SES inequality in mortality--]

The absolute measure of mortality inequality (rather than relative measure like the ratios of mortality rates) is used in this study. Measuring mortality inequality as the absolute difference between the lowest and highest SES groups has been used frequently in this area (Baker et al., 2021; J. M. Currie, 2018; J. Currie & Schwandt, 2016a, 2016b). Further, an absolute measure has some advantages for this study. Firstly, the relationship between mortality and SES rank appears to be linear each year (refer to Figure 4.4a & 4.4b). Therefore, the extreme values are not expected to have a significant impact on the comparison between the highest and lowest SES ranks. As is discussed by Currie and Schwandt (2016a, 2016b), the absolute-level measure of mortality inequality goes right to the core problems people care about: how many people die at different SES levels? Relative-level measurements are sometimes misleading. For example, if the mortality rate is about 2000 (per 100000) for poor people and then the mortality rate increases by 200 over a specific time. For rich people, the mortality rate is about 1000 (per 100000) and increases by 110. If we calculate the relative increase, mortality increased by 10% for the poor people but

increased by 11% for the rich people, suggesting rich people worsen more. The relative inequality between the poor and rich counties even declined a little bit. However, the absolute level inequality indicates that poor people suffer more from the mortality increase and the mortality inequality increases¹³.

Regression Analysis

First, to model the trends in mortality inequality by SES index (percentiles of county groups ranked from the 1st percentile to 100th percentile), an OLS regression model with year, SES, interaction of year and SES, plus controls is used.

$$Mortality_i = \beta_0 + \beta_1 Year_i + \beta_2 SES_i + \beta_3 Year_i \cdot SES_i + \pi_4 Controls + e_i$$

where i represents the county, year ranges from 1969 to 2020 (51 years of data) and enters the model as 50 dummies. The model predicts the mortality rates at the county level as a function of county-level SES and year, net of controls. The SES index (percentiles) enters the model as a continuous variable ranging from 1st to 100th percentile (the higher the richer), making the coefficient capture the absolute level of difference in mortality as one moves from the lowest SES rank (the 1st percentile, referred to as P0-1) to the highest (the 100th percentile, referred to as P99-100). In some of Tables (see Table 4.3-Table 4.6), the SES index is recoded to range from 0 to 1—which equals $(SES \text{ percentiles} - 1)/99$ —to make the coefficients represent the predicted mortality inequality between the lowest percentile (P0-1) and the highest percentile (P99-100) county group.

Robust standard errors are use in all OLS regression analyses to account for potential heteroscedasticity and correlated errors in the longitudinal data (Frees, 2004).

¹³ See Currie and Schwandt (2016), appendix p.4.

Results

Descriptive Analysis

Figure 4.4 presents the age standardized mortality rates by SES index (percentiles) for males and females, separately for time intervals between 1969 and 2020. The results show that the mortality rates decrease with SES (i.e., moving from the lowest SES percentile to the highest percentile of county group). Further, the relationship between SES percentiles and mortality rate is relatively linear. However, the slope has become steeper over time, suggesting that mortality inequality has been rising over time.

[---Figure 4.4 about here---]

Figures 4.5 presents the life expectancy by SES percentiles. Life expectancy presents similar patterns as the mortality rates: life expectancy increases as the counties move from the lower SES percentile to higher SES percentile. The relationships are quite linear, and the slopes become steeper over time.

[---Figure 4.5 about here---]

To better summarize the changes in mortality inequality over time, Figure 4.6 presents the age standardized mortality rates for the poorest county group (P0-1) and the richest county group (P99-100), for both males and females, across the whole period from 1969 to 2020¹⁴. The results show that for both males and females, the mortality rates decline for both high and low SES county groups from 1970 to the early 1980s. The SES disparities also remain relatively stable during this time period, though it is noteworthy that disparities were much narrower for women than for men.

¹⁴ I also used the comparison between bottom 20% percentile (P0-20) vs. top 20% percentiles (P80-100) and got very similar results.

Then since the mid-1980s, the higher SES group decreases much faster in mortality rates than the lower SES group, leading to widening disparities over time, for both males and females. The mortality rates for females of low SES county groups remained relatively flat since the 1980s. Further, the mortality rates increase dramatically during 2020 (the Covid-19 period) for both males and females, thereby exacerbating the SES disparities in mortality.

[----Figure 4.6 about here ----]

Figure 4.7 further shows the trends in life expectancy for the poorest county group (P0-1) and the richest county group (P99-100), for both males and females. The patterns are quite similar to the mortality rates presented above.

[----Figure 4.7a & Figure 4.7b about LE----]

Regression Analysis

Trends in Mortality Inequality

Figures 4.8 presents the predicted mortality rate of the 1st percentile (noted as P0-1) and 100th percentile (noted as P99-100) based on the OLS regression models controlling for race structure of county population, the percent of foreign-born, and the population density¹⁵. Year (1969-2020) enters the OLS models as 51 dummies. The regression models are not presented here but in appendix to save the space (see Appendix table S4.1). The predicted disparities between the P0-1 and P99-100 for males and females are also presented in Figure 4.9.

[--Figures 4.8 about here--]

¹⁵ Figure 4.8 shows that the confidence intervals are larger among the high SES county group. One possible reason is that the population size of the richer counties is larger, making the sample size (number of counties) in richer county bins are smaller.

[--Figures 4.9 about here--]

The predicted values from the OLS regression models display similar patterns of mortality inequality as the descriptive analysis above, but with more details. From Figure 4.9, we can see that during the 1970s and mid-1980s, mortality inequality remains relatively flat for both males and females (with some fluctuations: a short increase in the early 1970s and then a short decrease in late 1970s to early 1980s). Since the mid-1980s, mortality inequality increases fast for both males and females. The widening inequalities are due to faster decline of mortality for the higher SES group than the lower SES group. The low SES county groups mortality among females even stagnated since the 1980s.

Mortality inequality remains at high levels with slower increases since the mid-1990s for males and since the mid-2000s for females. But mortality inequality increases faster again since the mid-2010s, for both males and females. Mortality Inequality increased dramatically during Covid-19 (2020).

These results show that the trends in mortality inequality change nonlinearly over time. So, in Table 4.3 & 4.4, I split the trends from 1969 to 2020 into five separate observation periods: (1) 1969-1980, (2) 1981-1990, (3) 1991-2010, (4) 2011-2018, (5) 2019-2020. Then I fit a linear model of year within each of five observation periods to see the average increase in mortality inequality by SES. The year is re-coded to start from 0 to the max number of years in each period (e.g., in 1969-1980, year is recoded to 0-11; in 1981-1990, the year is recoded to 0-9). SES percentile is recoded to a continuous variable—which equals $(\text{SES Index}-1)/99$ —ranging from 0 to 1 (with 0 representing the lowest SES percentile and 1 representing highest SES percentile). The recoded SES makes the coefficients capture the average increase or decrease in mortality per year by SES at each observation period. The results in Table 4.3 show that for the lowest SES group (P0-1) males, the mortality rates

decrease during the first three observation periods but increase in 2010s and dramatically increase during 2019-2020 (see the coefficient of year). For the highest SES (P99-100) county group, the annual change in mortality rates would be the coefficient of year plus coefficient of year*SES interaction, leading to a stronger decline in mortality or a smaller increase in mortality during Covid. The coefficients of SES indicate the SES inequalities of mortality at the beginning of each observation period (e.g., 1969, 1981, 1991, 2011, and 2019). It shows the SES inequalities increase over time.

[--Table 4.3 about here--]

Then for the females, the mortality rates for the lowest SES group stagnated from 1980s-2018 (with only minor decline in 1991-2010). The mortality rates for the highest SES group (the coefficient of year plus year*SES) decreases with a larger magnitude since 1980s.

[--Table 4.4 about here--]

Diverging Mortality Inequality Trends Across Age Groups

Figures 4.10 present the predicted mortality difference between the poorest (P0-1) and richest (P99-100) county group, by age groups. These are based on predicted mortality values for the poorest and richest county group by age groups (see appendix Figure S4.1 for males and Figure S4.2 (for females). As is shown in Figure 4.10, the trends in mortality inequality vary systematically by age groups. In general, for the 1970s and early 1980s, mortality inequality remains relatively flat with even some decrease in some age groups. Then since the mid-1980s, mortality inequality increases, but the increase is much faster among older age groups than younger age groups, for both males and females. To be specific:

Mortality inequalities among age 0, 1-4, 5-9, 10-14, 15-19, and 20-24 decline from the 1970s to 1980s and then after the 1980s, the inequalities remain relatively flat for age groups 0-14, and fluctuate a little bit for age groups 15-19 and 20-24 (with a minor increase

in 1980s, a decrease in 1990s and 2000s, and an increase again since recent year). Similar patterns are found among age groups 25-34 and 35-44: there is a decline in mortality inequality from 1970 to the early 1980s, then an increase after the mid-1980s, then a minor decrease in inequality in 2000s and an increase again in the most recent year.

Among middle-aged adults (45-64) or older adults (65-84), the mortality inequalities remain relatively flat during 1970s and 1980s (with a short declined at the beginning of 1970s). Then the inequalities increase dramatically after the mid-1980s. The mortality inequality among the population aged 85+ presents unique trends, for both males and females: it remained flat until the 1990s, then increased fast from 1990 to mid-2000s, then it started to decline steeply since the mid-2000s.

[--Figure 4.10--]

Tables 4.5 and 4.6 further show the SES inequalities of mortality in 1970, 1980, 2000, 2010, and 2020 for each age group. The tables show that the mortality inequality decreased or remained stable among infants and children. For example, the mortality inequality decreased from 547 per 100000 to 371 for male aged 0, and from 184 to 98 for male aged 20-24. However, the mortality inequality increased among middle-aged adults and increased extremely among older adults. For example, increased from 152 to 315 for male aged 35-44, increased from 291 to 1171 for male aged 55-64, and from 35 to 2568 for male aged 75-84.

[---Table 4.5 and 4.6 here---]

The Age Distribution of Mortality Inequality

The changes in mortality inequality mentioned above indicate that the mortality inequality during infancy and early-life declined or remained relatively flat since the mid-1980s, but the inequality among middle-aged and later life increased dramatically over the

past several decades. That leads to the changes in the distribution of mortality inequality across different age groups or life stages, as shown in Figure 4.11 & 4.12.

While the inequalities from early life declined or remained relatively flat, the inequalities of middle-aged and especially older people, increased dramatically. As a result, the mortality inequalities are more and more concentrated on later life. Further, the peak of mortality inequality moves towards older age groups over time. For example, in 1970, the highest mortality inequality was in age 55-64; in 1980, the highest mortality inequality was in 65-74; in 2000, the highest mortality inequality was in 75-84; in 2010, the highest mortality inequality was in 85+. Then in 2020, because of the decline in mortality inequality among the 85+, the highest mortality inequality was in 75-84.

[--Figures 4.11 & 4.12--]

Conclusion and Discussion

Using county-level US mortality data by age and sex for the period (1969-2020) from NCHS (National Center for Health Statistics), this chapter examines the trends in SES inequality in all-cause mortality and further investigates how the trends in mortality inequality vary by age groups. The results show that the mortality inequality experienced several different stages of evolution: (1) mortality inequality experienced a minor increase during the 1970s and mid-1980s; (2) from the mid-1980s to mid-1990s for males and to the mid-2000s for females, mortality inequality increased dramatically due to faster decline of mortality for the higher SES group than for the lower SES group (the low SES county group's mortality among females even stagnated since the 1980s); (3) since then, mortality inequality remains at high levels with slower increases until the mid-2010s; (4) after the mid-2010s, the increase in mortality inequality speeds up for both males and females; and lastly, (5) the mortality inequality increases dramatically during Covid-19 (2020). Previous studies

have also shown that the SES inequalities in health and mortality have been widening since the latter half of the last century (Ezzati et al., 2008; G. K. Singh & Siahpush, 2006)., but the detailed trends over time—the acceleration or slowdown in the increase rate of mortality inequality, are first presented in this chapter.

Further, the results show that the trends in mortality inequality vary largely by age groups. To be specific, mortality inequalities among those age 0-24 declined from the 1970s to the 1980s and then remain relatively after the 1980s. The increase in mortality inequality since the mid-1980s was mainly concentrated on middle-aged adults (45-64) or older adults (65-84) and was much faster among older age groups than younger age groups, for both males and females. The diverging increase rate of mortality inequality across age groups leads to the changes in the distribution of mortality inequality across different life stages: mortality inequalities are more and more concentrated on later life. Several recent studies showed that mortality inequality had declined for infants, children, and young adults in the US from 1990 to 2010s, with stable or minor change for those in middle age, but it increased dramatically for older adults (Baker et al., 2021; J. M. Currie, 2018; J. Currie & Schwandt, 2016a, 2016b). Findings in this chapter are consistent with these studies. However, this study provides much more details about the diverging mortality inequality trends at each age group over the long-term period since the late 1960s. Further, this study is the first to show that the age distribution of mortality inequality has been progressively delayed to later life.

These results provide support for the theoretical framework I proposed in Chapter three. To be specific, the following hypotheses are supported by empirical evidence in this chapter:

- *Hypothesis 1.* SES disparities in all-cause mortality have increased over time.

However, the increase in mortality inequality mainly happened since the mid-1980s,

not since 1960s—which is lagged behind the Stage four of the Age of Delayed Degenerative diseases (Olshansky & Ault, 1986).

- *Hypothesis 3.* Mortality inequalities have widened more among later life stages than earlier life stages.
- *Hypothesis 4a.* The age distribution of mortality inequality has also shifted progressively toward older ages. Specifically, for successive years, the percent distribution of SES inequalities in mortality has been more and more concentrated toward older ages of later life.
- *Hypothesis 5.* SES inequalities in Covid-19 increase dramatically in 2020, especially among the later life.

Tables and Figures

Table 4.1 The Average or Percent of (County Level) SES Variables Across Counties.

	Year					
	1970	1980	1990	2000	2010	2020
% Bachelor's degree	7.18	11.27	13.27	16.26	18.72	22.29
% Less than high school degree	55.47	40.81	30.54	22.71	16.95	12.44
Unemployment rate	4.55	6.82	6.69	5.76	7.53	5.16
% Prof. and managerial occupation	18.99	17.58	19.66	28.29	29.87	32.89
% of population under poverty line	17.28	15.86	16.80	14.21	15.57	14.66
Average family income	8533.05 (1860.75)	19238.97 (3494.24)	33565.47 (7724.08)	51085.18 (11444.78)	65598.12 (15168.80)	84638.70 (20091.37)
N of Counties	3006	3006	3006	3006	3006	3006

Notes: In 1970, the percentage of families under the poverty rate, rather than the population under poverty rate, is used because the latter one is not provided. The average family income is not adjusted for inflation. The values of all variables (e.g., % bachelor's degree) are the arithmetic mean of 3006 counties, which would be different from the mean values for the US population

Table 4.2 Descriptive Trends in Age Adjusted Mortality Rates and Control Variables at the County Level, 1970-2020

	Year					
	1970	1980	1990	2000	2010	2020
Age adjusted mortality rate (per 100,000)	1580.01	1339.81	1218.49	1113.19	973.07	1125.05
Percent White	89.94	86.26	85.15	81.93	79.44	76.46
Percent Black	9.01	8.36	8.38	8.53	8.70	8.75
Percent Hispanic	3.37	3.76	4.44	6.15	7.78	9.54
Percent Other Race	1.05	1.62	2.03	3.40	4.08	5.25
Percent foreign-born	1.65	2.00	2.14	3.33	4.19	4.55
Population per square mile	133.19 (693.70)	139.59 (627.63)	150.39 (638.44)	164.95 (659.17)	174.94 (669.19)	186.22 (725.02)
N of Counties	3006	3006	3006	3006	3006	3006

Notes: The mortality rate is age standardized with US 2000 standard population. The values of all control variables (e.g., percent White) are the arithmetic mean of 3006 counties, which would be different from the mean values of US population

Table 4.3 The OLS Regression of County Level Mortality Rates on Year and SES Index (Percentiles), Male, US 1969-2020.

	1969-1980 Male	1981-1990 Male	1991-2010 Male	2011-2018 Male	2019-2020 Male
Year	-26.080*** [0.628]	-5.823*** [0.253]	-13.881*** [0.537]	2.710*** [0.614]	201.974*** [5.212]
SES Index (0-1)	-154.639*** [17.666]	-170.909*** [10.076]	-368.825*** [12.057]	-352.480*** [11.090]	-382.884*** [14.436]
Year * SES	-2.927* [1.968]	-11.892*** [0.586]	-0.587 [1.269]	-6.863*** [1.555]	-147.183*** [12.735]
Population per square mile	0.024*** [0.003]	0.034*** [0.004]	0.025*** [0.005]	0.024*** [0.005]	0.030*** [0.006]
Percent Black	3.891*** [0.197]	4.279*** [0.163]	3.421*** [0.176]	2.109*** [0.186]	3.428*** [0.250]
Percent Hispanic	-0.940*** [0.282]	-1.428*** [0.216]	-1.125*** [0.261]	-0.257* [0.235]	0.620** [0.304]
Percent foreign-born	-3.183** [1.253]	-2.971*** [0.667]	-4.935*** [0.651]	-8.033*** [0.663]	-8.638*** [0.868]
Intercept	1636.072*** [6.240]	1323.827*** [4.440]	1203.651*** [5.379]	1088.844*** [5.376]	1069.444*** [6.590]
N (County-Year Obs.)	36072	60120	30060	24048	6012
N (Counties)	3006	3006	3006	3006	3006
R-squared	0.194	0.287	0.362	0.341	0.436
Adjusted R-squared	0.194	0.287	0.362	0.341	0.436

Notes: † p<0.1, * p<0.05, ** p<0.01, *** p<0.01

The Year is recoded to start from 0 of each time-period.

Table 4.4 The OLS Regression of County Level Mortality Rates on Year and SES Index (Percentiles), Female, US 1969-2020.

	1969-1980 Female	1981-1990 Female	1991-2010 Female	2011-2018 Female	2019-2020 Female
Year	-22.051*** [0.428]	1.255*** [0.181]	-4.850*** [0.374]	0.860** [0.475]	133.500*** [3.929]
SES Index (0-1)	-108.138*** [12.790]	-44.309*** [6.747]	-207.252*** [9.183]	-259.956*** [8.587]	-271.259*** [11.295]
Year * SES	2.867** [1.436]	-7.524*** [0.427]	-5.867*** [1.005]	-4.822*** [1.171]	-110.565*** [9.707]
Population per square mile	0.013*** [0.002]	0.016*** [0.003]	0.011*** [0.003]	0.012*** [0.003]	0.017*** [0.005]
Percent Black	2.196*** [0.114]	2.383*** [0.105]	1.975*** [0.120]	0.822*** [0.130]	1.862*** [0.175]
Percent Hispanic	-0.410** [0.214]	-0.680*** [0.129]	-0.851*** [0.165]	-0.701*** [0.185]	-0.299* [0.289]
Percent foreign- born	1.478** [0.885]	-0.109 [0.425]	-1.792*** [0.450]	-3.881*** [0.523]	-4.751*** [0.745]
Intercept	1004.547*** [4.387]	751.298*** [2.936]	812.972*** [3.728]	794.165*** [4.176]	765.069*** [5.058]
N (County-Year Obs.)	36072	60120	30060	24048	6012
N (Counties)	3006	3006	3006	3006	3006
R-squared	0.194	0.132	0.266	0.295	0.394
Adjusted R- squared	0.193	0.132	0.266	0.295	0.394

Notes: † p<0.1, * p<0.05, ** p<0. 01, *** p< 0.01

The Year is recoded to start from 0 of each time period.

Table 4.5 The Regression of County Level Mortality Rates On SES Index(Percentiles) by Age Groups, Males, US 1970-2020

	1970 Male	1980 Male	2000 Male	2010 Male	2020 Male
The coef. Of SES (0-1)					
0	-547.831*** [130.096]	-264.735*** [79.071]	-302.648*** [74.542]	-332.853*** [80.120]	-371.135*** [82.863]
1-4	-37.089** [15.515]	-2.338 [18.521]	-16.662* [10.874]	-20.728** [8.396]	-21.882*** [6.813]
5-9	-29.862*** [8.976]	-14.901** [5.897]	-9.746* [6.834]	-9.984** [4.355]	-6.768** [3.604]
10-14	-20.742** [8.452]	-2.472 [9.559]	-22.007*** [6.014]	-12.211* [8.180]	-10.749** [6.434]
15-19	-120.524*** [17.353]	-100.251*** [14.003]	-62.522*** [13.515]	-27.344** [14.961]	-26.424* [19.122]
20-24	-183.664*** [35.808]	-92.017*** [21.874]	-91.365*** [29.634]	-86.446*** [22.220]	-97.717*** [19.498]
25-34	-122.528*** [20.785]	-104.368*** [14.167]	-115.258*** [14.901]	-131.232*** [14.920]	-134.224*** [25.286]
35-44	-151.962*** [26.957]	-155.861*** [19.550]	-227.700*** [13.413]	-191.457*** [18.984]	-315.058*** [24.959]
45-54	-254.446*** [33.328]	-304.837*** [31.908]	-390.713*** [22.454]	-389.257*** [23.272]	-554.718*** [34.003]
55-64	-291.229*** [54.039]	-436.081*** [44.561]	-742.410*** [40.308]	-753.549*** [32.311]	-1171.277*** [42.087]
65-74	-29.793 [107.439]	-455.141*** [86.741]	-1206.885*** [70.314]	-1235.775*** [61.337]	-1865.701*** [66.513]
75-84	-35.801 [279.733]	-244.889* [188.417]	-1664.640*** [151.946]	-1759.347*** [127.483]	-2568.683*** [134.684]
85+	-158.752 [904.129]	-354.624 [762.729]	-1875.684*** [416.781]	-2438.812*** [417.156]	-577.581* [507.297]

Notes: † p<0.1, * p<0.05, ** p<0.01, *** p<0.01

The SES percentile is recoded to range from 0 to 1, which equals (SES index-1)/99.

The coefficients of SES for each age group are presented, with standard errors in parentheses

Table 4.6 The Regression of County Level Mortality Rates on SES Index(Percentiles) by Age Groups, Females, US 1970-2020

	1970 Female	1980 Female	2000 Female	2010 Female	2020 Female
The coef. Of SES (0-1)					
0	-547.831*** [130.096]	-264.735*** [79.071]	-302.648*** [74.542]	-332.853*** [80.120]	-371.135*** [82.863]
1-4	-37.089** [15.515]	-2.338 [18.521]	-16.662* [10.874]	-20.728** [8.396]	-21.882*** [6.813]
5-9	-29.862*** [8.976]	-14.901** [5.897]	-9.746* [6.834]	-9.984** [4.355]	-6.768** [3.604]
10-14	-20.742** [8.452]	-2.472 [9.559]	-22.007*** [6.014]	-12.211* [8.180]	-10.749** [6.434]
15-19	-120.524*** [17.353]	-100.251*** [14.003]	-62.522*** [13.515]	-27.344** [14.961]	-26.424* [19.122]
20-24	-183.664*** [35.808]	-92.017*** [21.874]	-91.365*** [29.634]	-86.446*** [22.220]	-97.717*** [19.498]
25-34	-122.528*** [20.785]	-104.368*** [14.167]	-115.258*** [14.901]	-131.232*** [14.920]	-134.224*** [25.286]
35-44	-151.962*** [26.957]	-155.861*** [19.550]	-227.700*** [13.413]	-191.457*** [18.984]	-315.058*** [24.959]
45-54	-254.446*** [33.328]	-304.837*** [31.908]	-390.713*** [22.454]	-389.257*** [23.272]	-554.718*** [34.003]
55-64	-291.229*** [54.039]	-436.081*** [44.561]	-742.410*** [40.308]	-753.549*** [32.311]	-1171.277*** [42.087]
65-74	-29.793 [107.439]	-455.141*** [86.741]	-1206.885*** [70.314]	-1235.775*** [61.337]	-1865.701*** [66.513]
75-84	-35.801 [279.733]	-244.889* [188.417]	-1664.640*** [151.946]	-1759.347*** [127.483]	-2568.683*** [134.684]
85+	-158.752 [904.129]	-354.624 [762.729]	-1875.684*** [416.781]	-2438.812*** [417.156]	-577.581* [507.297]

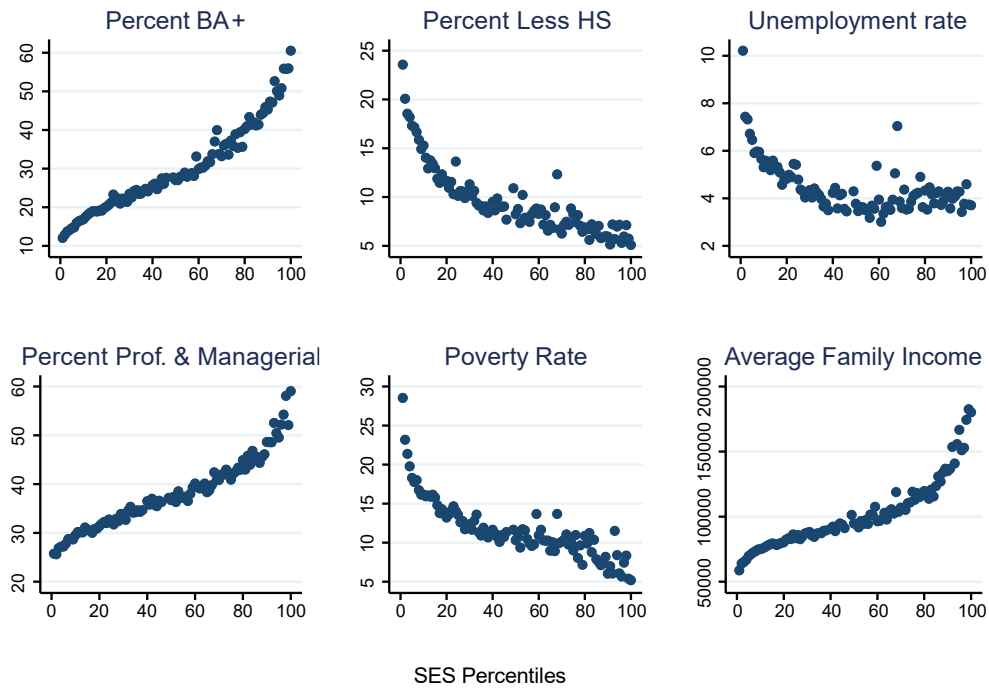
Notes: † p<0.1, * p<0.05, ** p<0.01, *** p<0.01

The SES percentile is recoded to range from 0 to 1, which equals (SES index – 1)/99.

The coefficients of SES for each age group are presented, with standard errors in parentheses

Figures

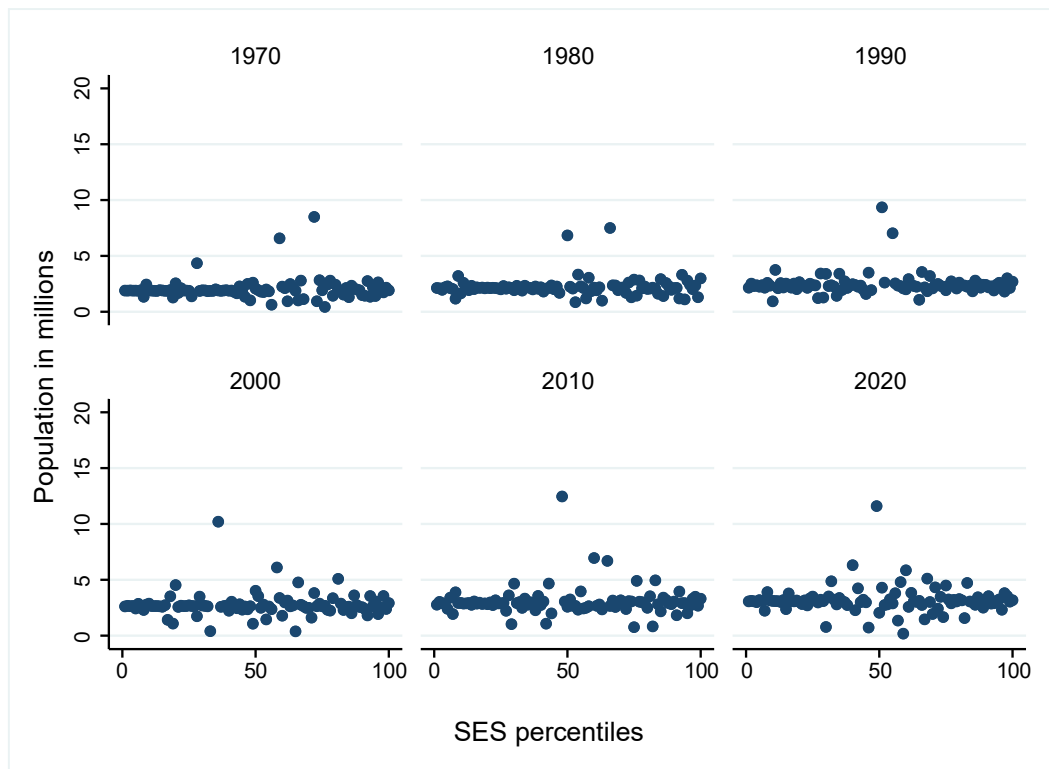
Figure 4.1 The Average Values of Each SES Variable (at County Level) by SES Index (Percentiles) In 2020.



Note: County level data at 2020

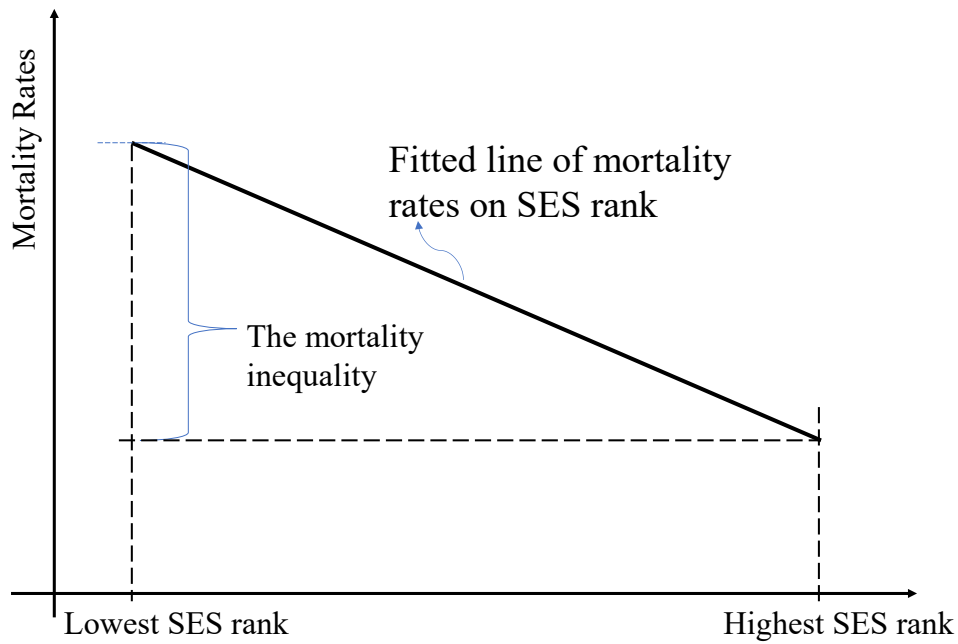
Notes: SES index (percentiles) are constructed by ranking counties by a SES index (the SES index is a constructed by proportion of education, occupation structure, poverty rate and average family income) and dividing the counties into 1% bins of the overall population.

Figure 4.2 Population Size of 100 County Groups Ordered By SES Index (Percentiles), 1970-2020.



Notes: SES percentiles are constructed by ranking counties by a SES index, which is a constructed by proportion of education, occupation structure, poverty rate and average family income) and dividing the counties into 1% bins of the overall population. Overall, the variation of population size at each bin is relatively small (except two to three bins with larger population size) and is not related to the SES variables.

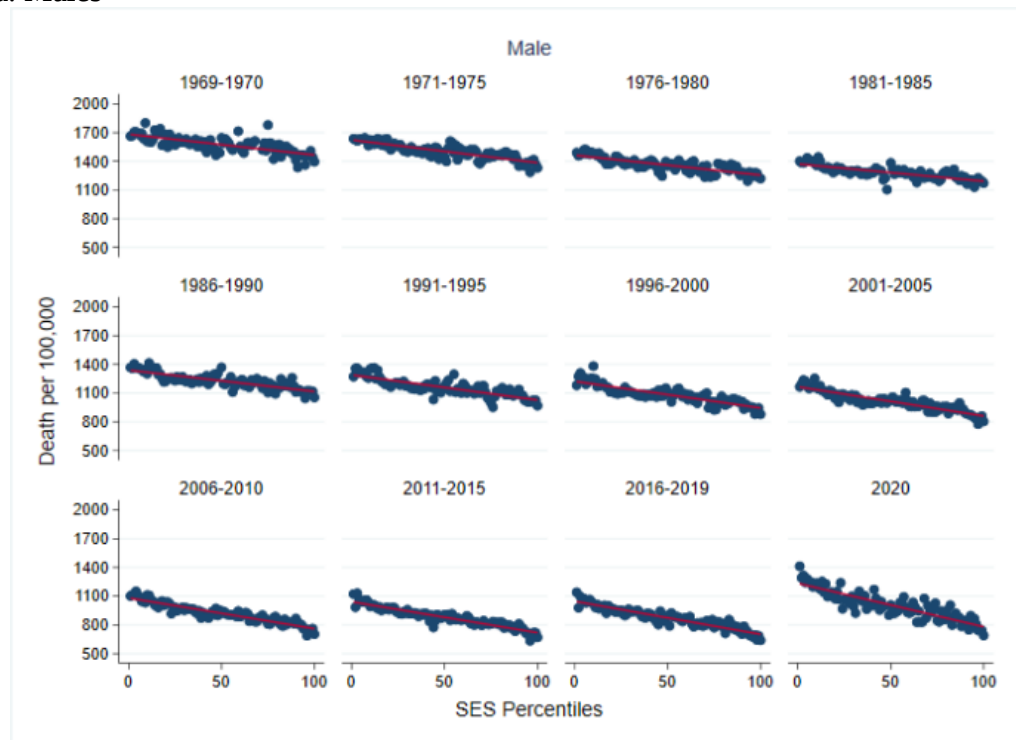
Figure 4.3 The Measurement of Absolute Mortality Inequality By SES Rank.



Notes: The mortality inequality measures the changes in mortality when moving from the lowest SES rank to highest SES rank. If the lowest SES rank is rescaled to 0 and the highest SES rank is rescaled to 1, then the slope of the fitted line represents the magnitude of mortality inequality when moving from the lowest SES rank to highest SES rank.

Figure 4.4 The Association of Age-adjusted Mortality Rates and SES Percentiles in the US. (1969-2020)

a. Males



b. Females

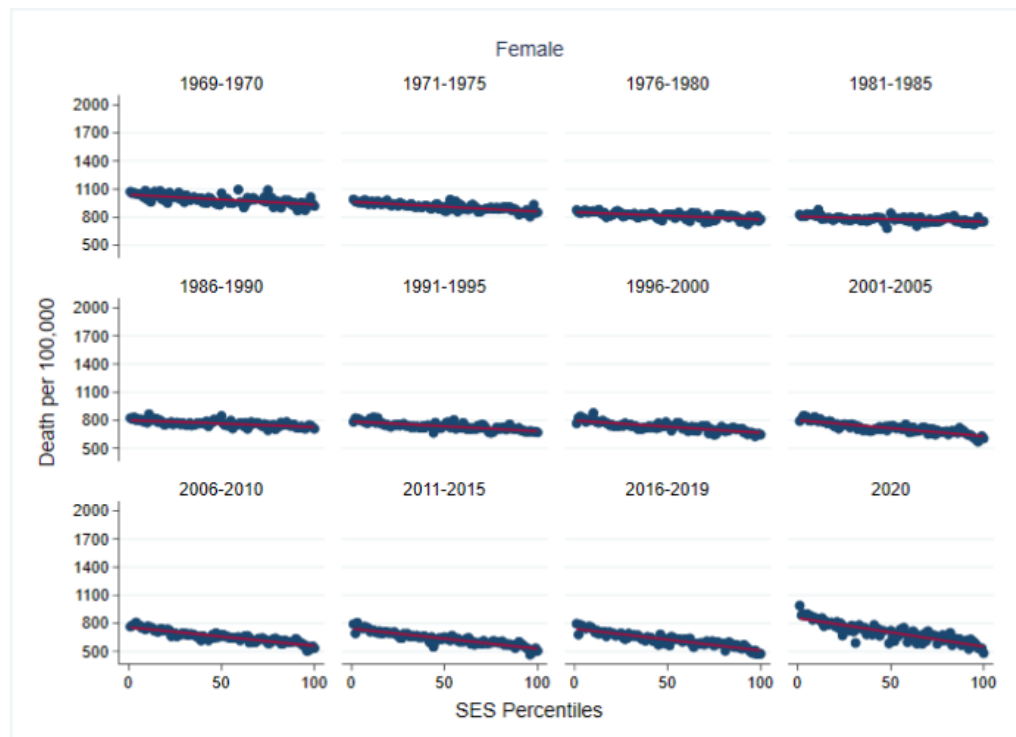
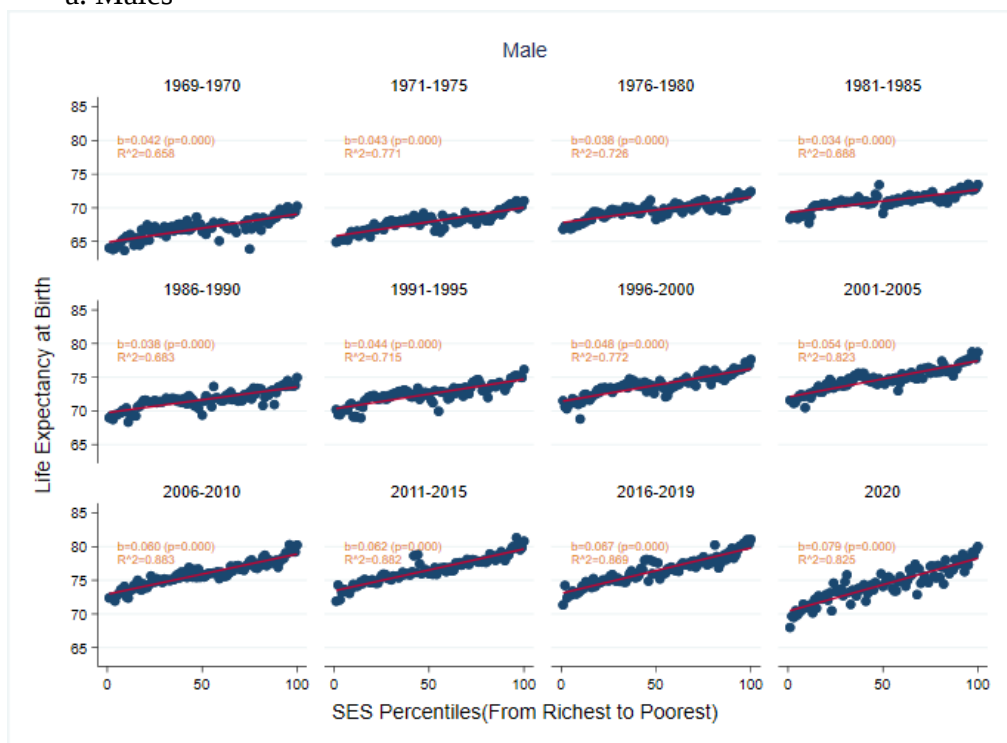


Figure 4.5 The Association of Life Expectancy and SES Index (Percentiles) in the US. (1969-2020)

a. Males



b. Females



Figure 4.6 The Trends in All-cause Mortality for the Highest SES Percentile (P99-100) and Lowest SES Percentile (P0-1)

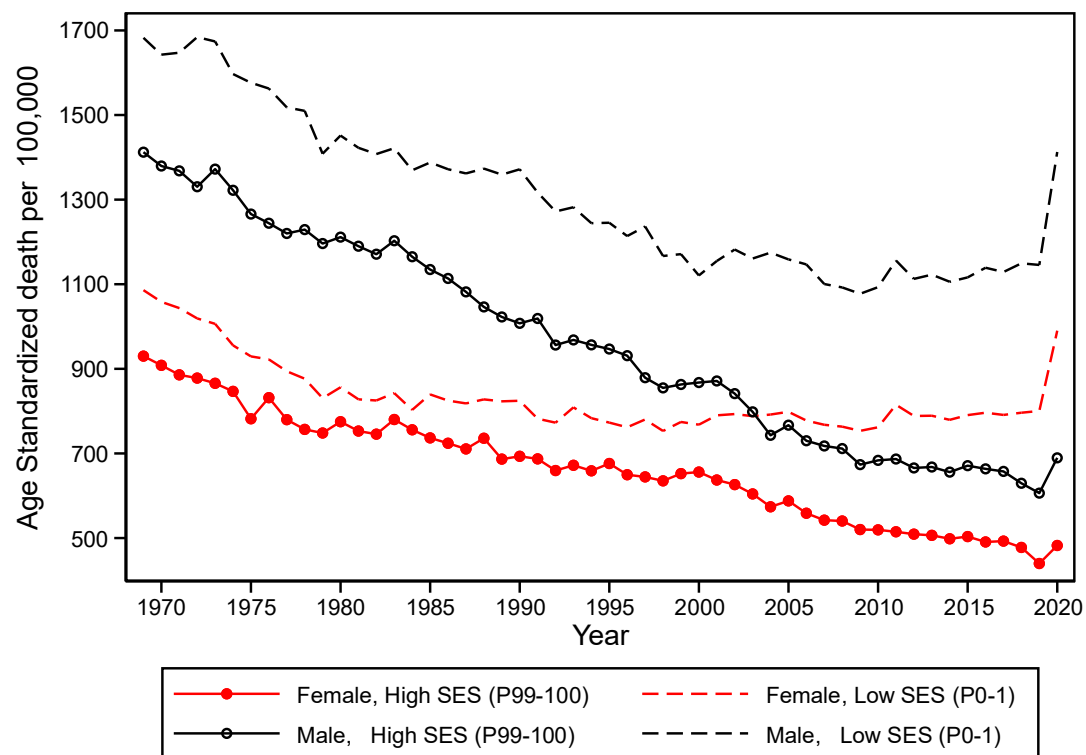
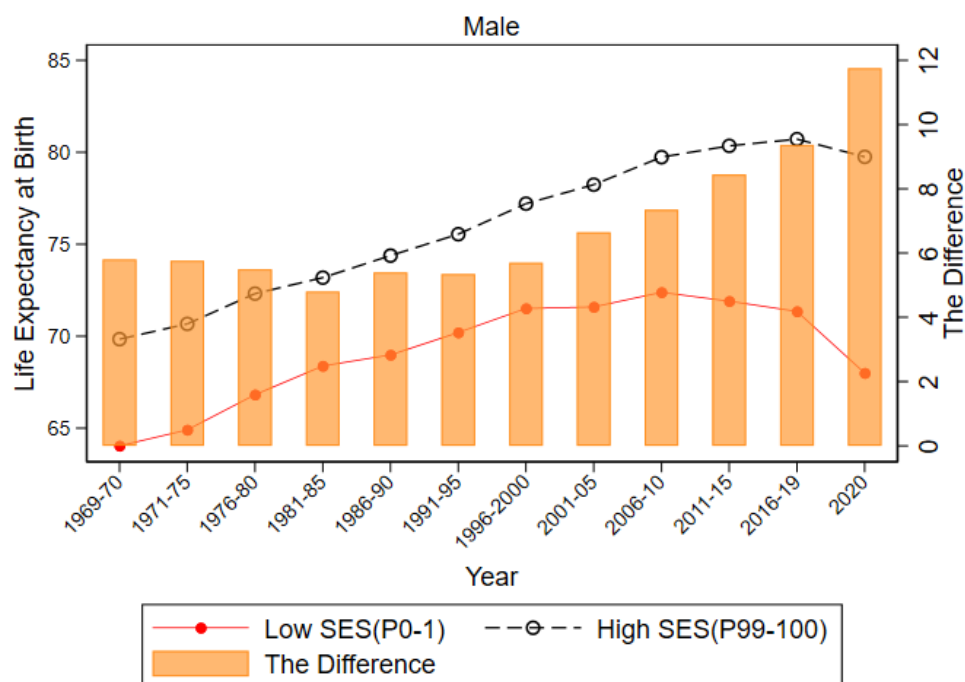


Figure 4.7 Trends in Life Expectancy by Highest SES Percentile (P99-100) vs. Lowest SES Percentile (P0-1), and the SES Difference in Life Expectancy.

a. Males.



b. Females

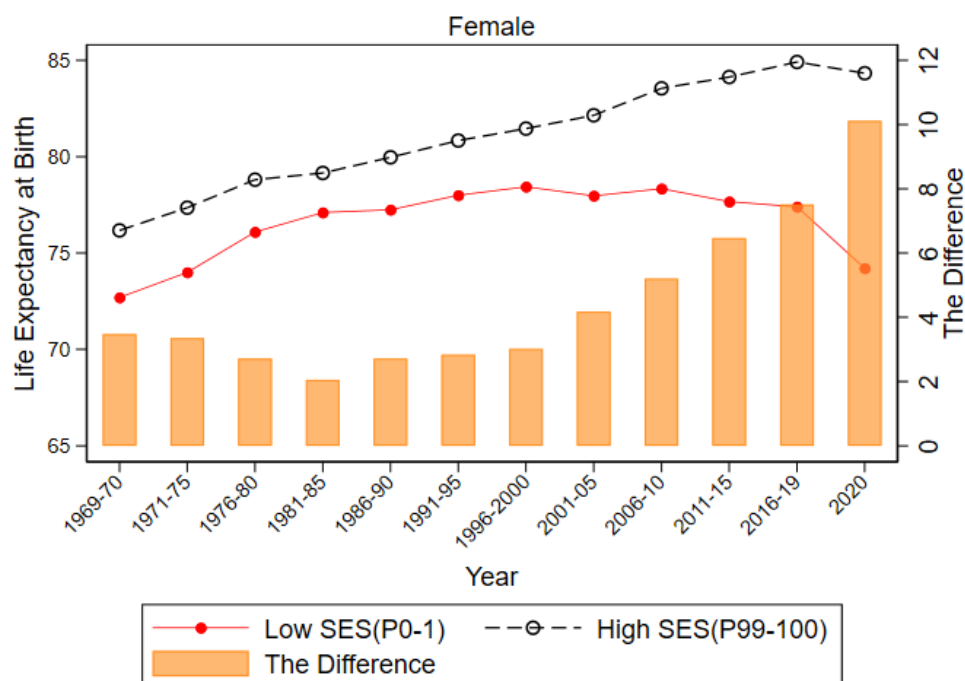
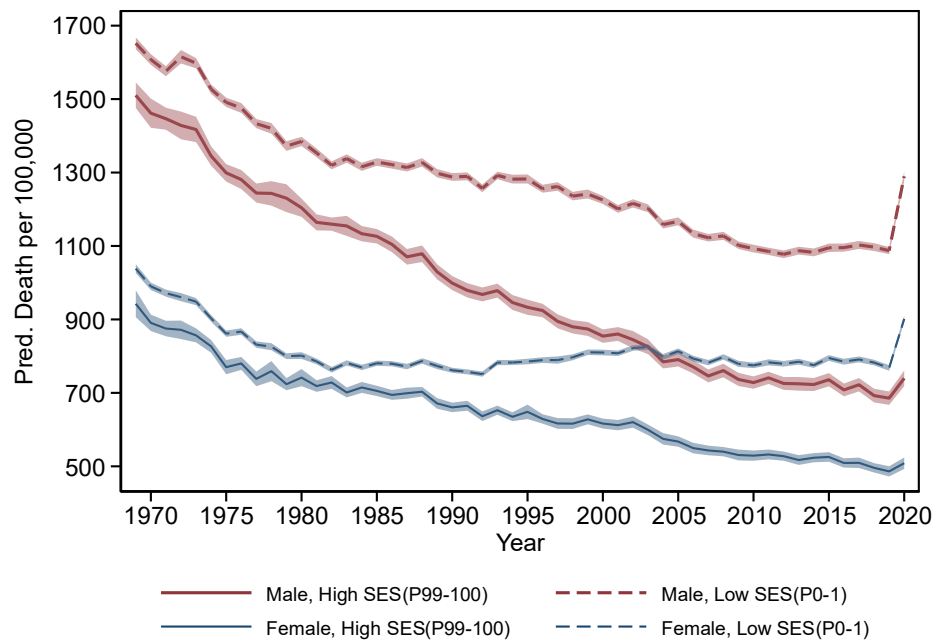
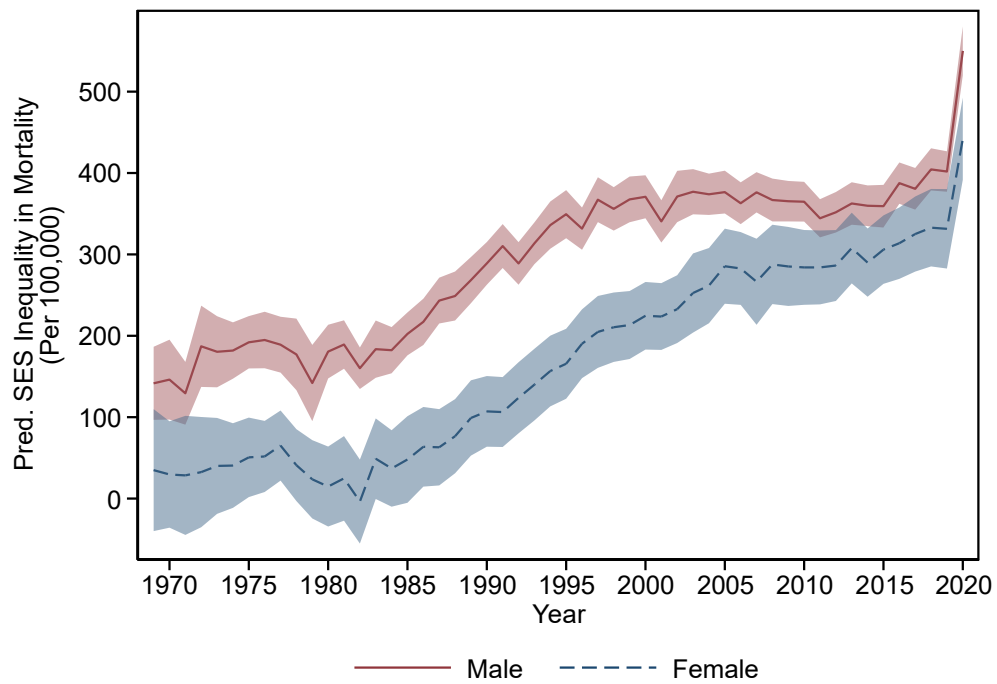


Figure 4.8 Predicted Mortality Rates for Highest SES Percentile (P99-100) and Lowest SES Percentile (P0-1), 1969-2020, Males and Females.



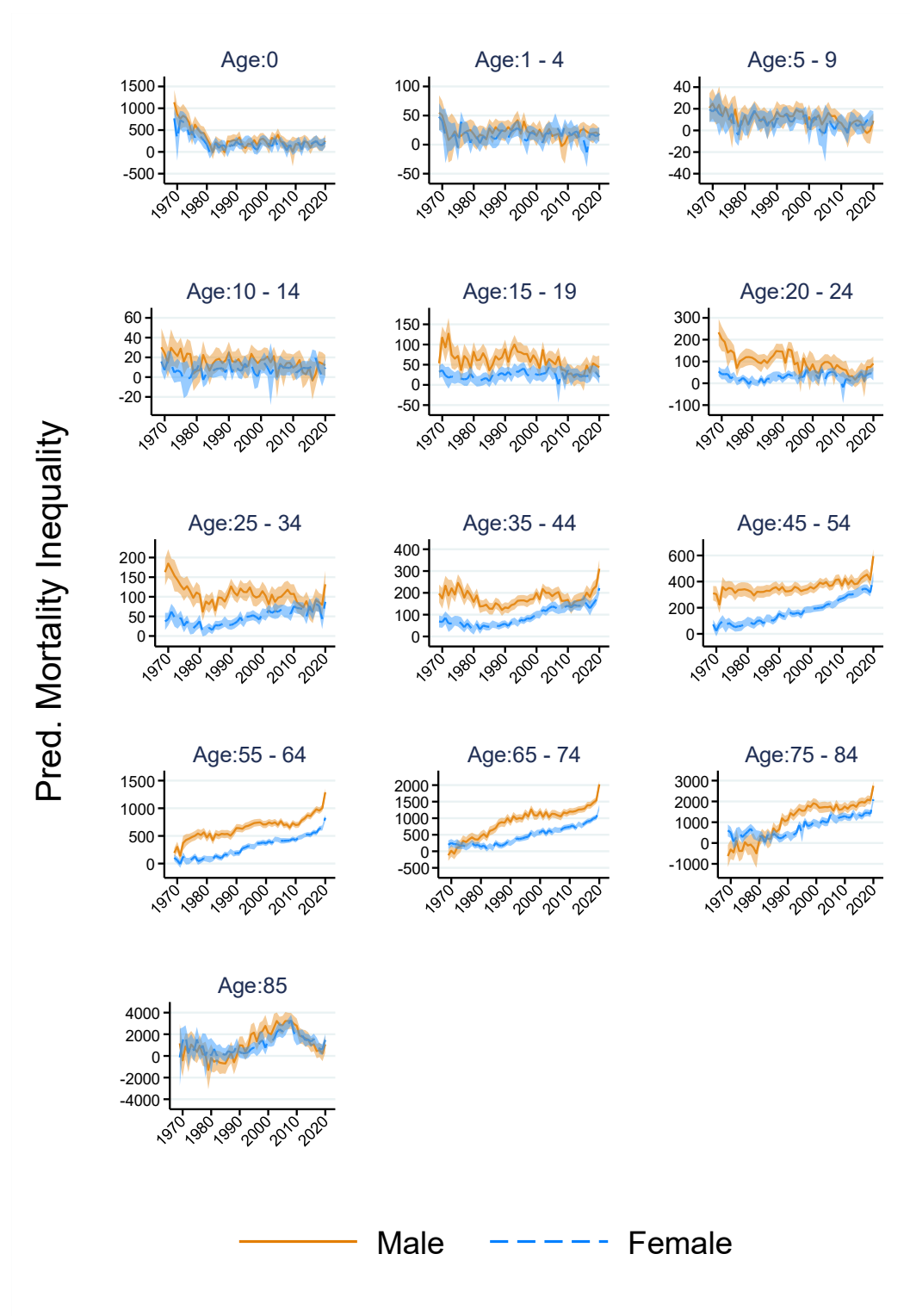
Notes: Shaded areas represent 95% confidence intervals. Confidence intervals the confidence intervals are larger among the high SES county bins. One possible reason is that the population size of the richer counties is larger, making the sample size (number of counties) in richer county bins smaller.

Figure 4.9 Predicted Mortality Inequality Between the Highest SES Percentile (P99-100) and Lowest SES Percentile (P0-1), 1969-2020, Males and Females.



Note: Shaded areas represent 95% confidence intervals.

Figure 4.10 Predicted Mortality Inequality Between the Highest SES Percentile (P99-100) and Lowest SES Percentile (P0-1), By Age Groups and Sex, 1969-2020.



Note: Shaded areas represent 95% confidence intervals.

Figure 4.11 Trends in Male Mortality Inequality Between the Highest SES Percentile (P99-100) and Lowest SES Percentile (P0-1), by Age Group, 1970-2020 (with 95% confidence intervals.)

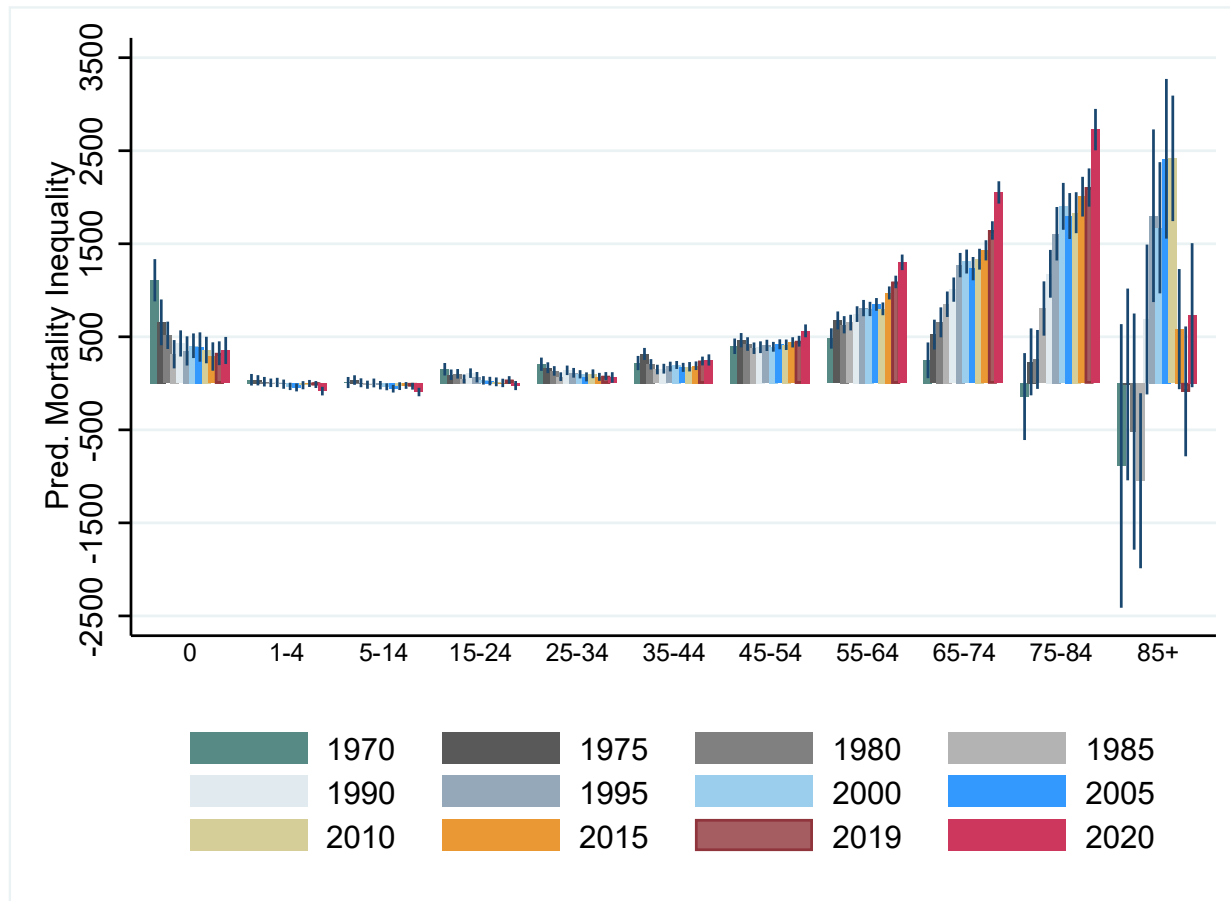
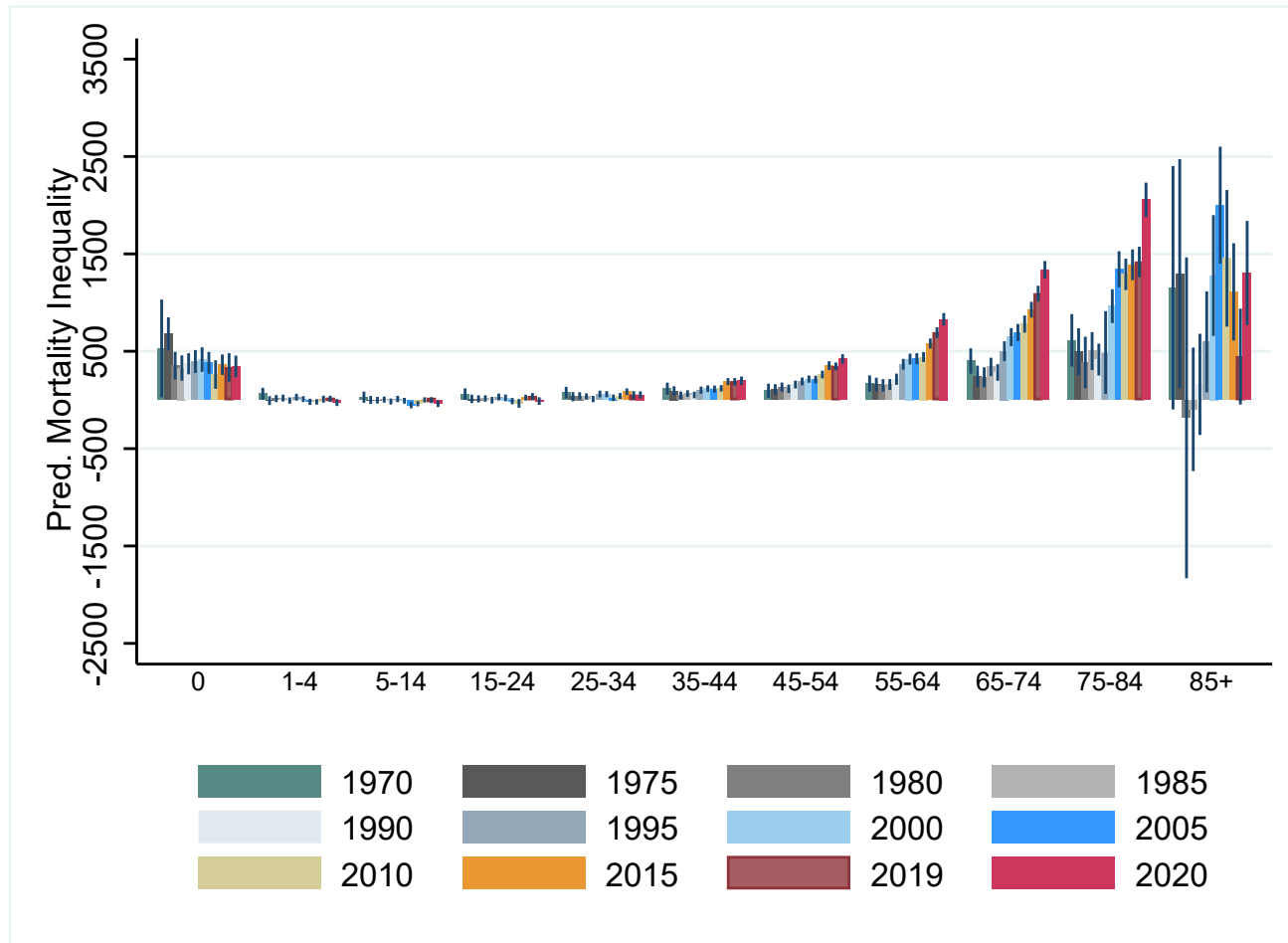


Figure 4.12 Trends in Female Mortality Inequality Between the Highest SES Percentile (P99-100) and Lowest SES Percentile (P0-1), by Age Group, 1970-2020 (with 95% confidence intervals).



Appendix Tables and Figures

Table S4.1 OLS Regression of County Level Mortality Rates on Year and SES Index(Percentiles)

	Male			Female		
	M1:OLS	M2:+Controls	M3:Robust	M4:OLS	M5:+Controls	M6:Robust
1970	-50.83** [23.33]	-51.41** [22.48]	-51.41** [22.19]	-46.89** [20.07]	-47.46** [19.58]	-47.46** [20.49]
1971	-79.08*** [23.33]	-80.36*** [22.48]	-80.36*** [20.63]	-59.16*** [20.07]	-60.34*** [19.58]	-60.34*** [19.58]
1972	-109.32*** [23.31]	-111.04*** [22.46]	-111.04*** [24.79]	-73.13*** [20.06]	-74.70*** [19.57]	-74.70*** [18.91]
1973	-127.73*** [23.30]	-130.03*** [22.45]	-130.03*** [24.69]	-93.21*** [20.05]	-95.26*** [19.56]	-95.26*** [20.53]
1974	-197.20*** [23.28]	-200.29*** [22.43]	-200.29*** [24.26]	-135.11*** [20.03]	-137.75*** [19.54]	-137.75*** [21.40]
1975	-254.20*** [23.23]	-257.79*** [22.38]	-257.79*** [25.72]	-188.73*** [19.99]	-191.73*** [19.50]	-191.73*** [21.94]
1976	-289.65*** [23.18]	-293.87*** [22.34]	-293.87*** [29.20]	-184.92*** [19.94]	-188.35*** [19.46]	-188.35*** [22.34]
1977	-325.35*** [23.14]	-330.32*** [22.30]	-330.32*** [30.68]	-231.70*** [19.91]	-235.61*** [19.42]	-235.61*** [25.19]
1978	-327.93*** [23.11]	-333.60*** [22.27]	-333.60*** [31.65]	-212.28*** [19.88]	-216.68*** [19.40]	-216.68*** [24.81]
1979	-339.51*** [23.07]	-345.89*** [22.23]	-345.89*** [34.28]	-225.88*** [19.85]	-230.74*** [19.36]	-230.74*** [27.17]
1980	-366.62*** [23.05]	-373.77*** [22.22]	-373.77*** [33.75]	-213.38*** [19.84]	-218.76*** [19.35]	-218.76*** [28.34]
1981	-397.22*** [23.19]	-406.49*** [22.34]	-406.49*** [40.37]	-236.89*** [19.95]	-244.21*** [19.46]	-244.21*** [25.29]
1982	-416.95*** [23.32]	-428.39*** [22.47]	-428.39*** [40.92]	-229.48*** [20.07]	-238.74*** [19.57]	-238.74*** [26.79]
1983	-439.49*** [23.43]	-453.34*** [22.58]	-453.34*** [42.12]	-259.34*** [20.16]	-270.66*** [19.67]	-270.66*** [28.18]
1984	-450.90*** [23.52]	-466.92*** [22.67]	-466.92*** [44.32]	-256.32*** [20.24]	-269.45*** [19.75]	-269.45*** [28.70]
1985	-446.43*** [23.61]	-464.70*** [22.76]	-464.70*** [44.61]	-255.09*** [20.32]	-270.06*** [19.82]	-270.06*** [31.00]
1986	-470.57*** [23.72]	-491.15*** [22.86]	-491.15*** [45.06]	-268.82*** [20.41]	-285.67*** [19.91]	-285.67*** [32.52]
1987	-510.12*** [23.80]	-532.87*** [22.93]	-532.87*** [46.09]	-272.89*** [20.48]	-291.47*** [19.98]	-291.47*** [35.59]
1988	-507.85*** [23.87]	-532.66*** [23.00]	-532.66*** [45.50]	-269.20*** [20.54]	-289.38*** [20.04]	-289.38*** [36.00]
1989	-556.09*** [23.93]	-582.91*** [23.06]	-582.91*** [47.13]	-300.75*** [20.59]	-322.49*** [20.09]	-322.49*** [36.36]
1990	-588.25*** [23.98]	-617.05*** [23.11]	-617.05*** [47.57]	-317.53*** [20.63]	-340.72*** [20.13]	-340.72*** [36.61]
1991	-614.42*** [23.93]	-642.97*** [23.06]	-642.97*** [47.93]	-320.96*** [20.59]	-344.73*** [20.09]	-344.73*** [37.16]
1992	-619.45***	-647.66***	-647.66***	-344.73***	-368.94***	-368.94***

	[23.89]	[23.02]	[51.76]	[20.55]	[20.05]	[36.97]
1993	-609.16***	-636.94***	-636.94***	-329.73***	-354.22***	-354.22***
	[23.82]	[22.96]	[51.91]	[20.50]	[20.00]	[37.63]
1994	-641.73***	-668.87***	-668.87***	-347.08***	-371.67***	-371.67***
	[23.76]	[22.90]	[50.60]	[20.44]	[19.95]	[37.85]
1995	-666.06***	-692.55***	-692.55***	-351.40***	-376.03***	-376.03***
	[23.69]	[22.83]	[50.32]	[20.38]	[19.89]	[37.75]
1996	-677.34***	-703.08***	-703.08***	-372.06***	-396.58***	-396.58***
	[23.62]	[22.76]	[50.75]	[20.32]	[19.83]	[38.72]
1997	-712.95***	-738.05***	-738.05***	-387.41***	-411.84***	-411.84***
	[23.54]	[22.69]	[50.93]	[20.26]	[19.76]	[39.06]
1998	-730.58***	-755.06***	-755.06***	-387.72***	-412.06***	-412.06***
	[23.47]	[22.63]	[51.22]	[20.20]	[19.71]	[39.63]
1999	-739.28***	-763.17***	-763.17***	-376.77***	-400.97***	-400.97***
	[23.40]	[22.55]	[51.61]	[20.13]	[19.65]	[39.48]
2000	-763.52***	-786.78***	-786.78***	-390.96***	-414.90***	-414.90***
	[23.32]	[22.47]	[51.19]	[20.06]	[19.58]	[39.88]
2001	-756.62***	-779.86***	-779.86***	-392.81***	-417.25***	-417.25***
	[23.28]	[22.44]	[51.33]	[20.03]	[19.55]	[40.01]
2002	-784.68***	-807.93***	-807.93***	-387.21***	-412.16***	-412.16***
	[23.24]	[22.40]	[51.45]	[20.00]	[19.52]	[40.64]
2003	-803.20***	-826.52***	-826.52***	-404.47***	-429.88***	-429.88***
	[23.19]	[22.36]	[51.50]	[19.96]	[19.48]	[42.24]
2004	-842.74***	-865.99***	-865.99***	-438.50***	-464.18***	-464.18***
	[23.13]	[22.29]	[51.75]	[19.90]	[19.42]	[42.20]
2005	-841.04***	-864.27***	-864.27***	-446.63***	-472.63***	-472.63***
	[23.08]	[22.25]	[51.97]	[19.86]	[19.38]	[42.36]
2006	-859.11***	-882.26***	-882.26***	-465.20***	-491.47***	-491.47***
	[23.01]	[22.18]	[52.07]	[19.80]	[19.32]	[42.21]
2007	-885.28***	-908.24***	-908.24***	-461.73***	-488.09***	-488.09***
	[22.93]	[22.11]	[51.95]	[19.73]	[19.26]	[44.00]
2008	-877.11***	-900.00***	-900.00***	-466.71***	-493.25***	-493.25***
	[22.87]	[22.04]	[52.01]	[19.68]	[19.20]	[42.95]
2009	-901.09***	-923.99***	-923.99***	-482.05***	-508.80***	-508.80***
	[22.79]	[21.98]	[51.41]	[19.61]	[19.14]	[43.13]
2010	-909.73***	-932.47***	-932.47***	-486.26***	-513.09***	-513.09***
	[22.73]	[21.91]	[52.04]	[19.55]	[19.09]	[42.68]
2011	-897.49***	-919.93***	-919.93***	-478.86***	-505.67***	-505.67***
	[22.72]	[21.90]	[52.10]	[19.55]	[19.08]	[42.83]
2012	-914.92***	-937.16***	-937.16***	-485.07***	-511.81***	-511.81***
	[22.69]	[21.88]	[52.18]	[19.53]	[19.06]	[42.63]
2013	-916.80***	-938.82***	-938.82***	-500.05***	-526.69***	-526.69***
	[22.66]	[21.85]	[52.20]	[19.50]	[19.03]	[42.52]
2014	-918.82***	-940.75***	-940.75***	-492.31***	-518.81***	-518.81***
	[22.61]	[21.80]	[52.42]	[19.45]	[18.99]	[42.39]
2015	-905.80***	-927.47***	-927.47***	-488.49***	-514.66***	-514.66***
	[22.53]	[21.72]	[52.34]	[19.39]	[18.92]	[42.32]
2016	-933.02***	-958.78***	-958.78***	-502.19***	-532.55***	-532.55***
	[22.77]	[21.95]	[52.65]	[19.59]	[19.12]	[43.02]
2017	-918.98***	-948.96***	-948.96***	-501.67***	-536.21***	-536.21***
	[22.97]	[22.15]	[53.84]	[19.77]	[19.30]	[43.68]
2018	-943.72***	-978.18***	-978.18***	-513.17***	-551.99***	-551.99***
	[23.16]	[22.33]	[54.83]	[19.92]	[19.45]	[44.52]
2019	-945.03***	-983.88***	-983.88***	-521.15***	-564.04***	-564.04***

	[23.30]	[22.47]	[54.40]	[20.05]	[19.58]	[44.93]
2020	-881.26***	-924.49***	-924.49***	-491.90***	-538.73***	-538.73***
	[23.43]	[22.60]	[53.69]	[20.16]	[19.68]	[44.63]
SES						
Quantile	50.72**	21.90*	21.90	31.02**	34.93**	34.93*
	[21.61]	[20.89]	[54.34]	[18.60]	[18.19]	[38.21]
1970#SES						
Quantile	5.56	6.55	6.55	-6.08	-5.35	-5.35
	[30.58]	[29.47]	[28.46]	[26.32]	[25.67]	[25.39]
1971#SES						
Quantile	4.74	6.89	6.89	-7.95	-6.45	-6.45
	[30.61]	[29.49]	[27.01]	[26.34]	[25.69]	[24.36]
1972#SES						
Quantile	77.07**	80.02***	80.02**	-4.42	-2.45	-2.45
	[30.61]	[29.49]	[32.99]	[26.34]	[25.69]	[24.04]
1973#SES						
Quantile	77.67**	81.60***	81.60***	2.60	5.18	5.18
	[30.61]	[29.50]	[31.61]	[26.34]	[25.70]	[25.84]
1974#SES						
Quantile	75.58**	80.81***	80.81***	2.27	5.60	5.60
	[30.60]	[29.49]	[30.92]	[26.33]	[25.69]	[26.34]
1975#SES						
Quantile	98.75***	104.85***	104.85***	11.91	15.70	15.70
	[30.58]	[29.47]	[32.62]	[26.31]	[25.67]	[26.89]
1976#SES						
Quantile	121.31***	128.44***	128.44***	12.46	16.79	16.79
	[30.55]	[29.44]	[36.47]	[26.29]	[25.65]	[27.62]
1977#SES						
Quantile	114.89***	123.26***	123.26***	25.22*	30.18*	30.18*
	[30.53]	[29.42]	[38.05]	[26.27]	[25.63]	[30.65]
1978#SES						
Quantile	102.18***	111.71***	111.71***	0.50	6.09	6.09
	[30.52]	[29.41]	[39.58]	[26.26]	[25.62]	[30.34]
1979#SES						
Quantile	64.15**	74.88**	74.88**	-17.48	-11.28	-11.28
	[30.50]	[29.39]	[41.92]	[26.24]	[25.60]	[33.17]
1980#SES						
Quantile	103.45***	115.47***	115.47***	-27.05*	-20.17*	-20.17
	[30.50]	[29.39]	[41.62]	[26.25]	[25.60]	[34.46]
1981#SES						
Quantile	101.08***	116.14***	116.14**	-19.63*	-10.17	-10.17
	[30.63]	[29.52]	[49.42]	[26.35]	[25.71]	[30.78]
1982#SES						
Quantile	89.27***	107.43***	107.43**	-50.68**	-38.63*	-38.63*
	[30.75]	[29.64]	[50.21]	[26.46]	[25.82]	[32.41]
1983#SES						
Quantile	135.08***	156.63***	156.63***	-0.78	14.02	14.02
	[30.87]	[29.74]	[51.59]	[26.56]	[25.91]	[33.60]
1984#SES						
Quantile	120.80***	145.37***	145.37***	-15.16	2.03	2.03
	[30.95]	[29.83]	[54.06]	[26.63]	[25.98]	[34.26]
1985#SES						
Quantile	128.42***	156.10***	156.10***	-6.56	13.05	13.05
	[31.04]	[29.91]	[54.31]	[26.71]	[26.05]	[37.03]

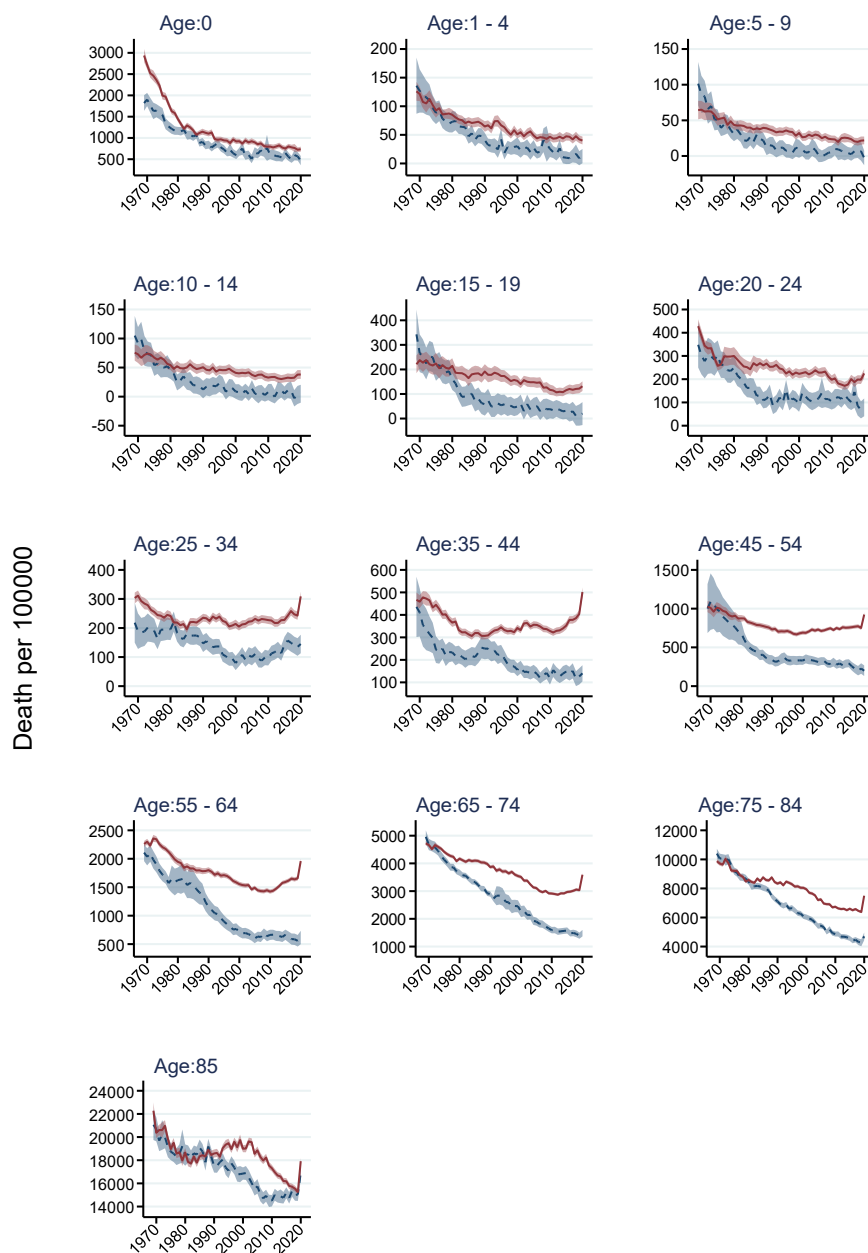
1986#SES						
Quantile	144.57*** [31.13]	175.43*** [30.00]	175.43*** [54.89]	6.61 [26.79]	28.66* [26.13]	28.66* [39.06]
1987#SES						
Quantile	177.75*** [31.20]	211.57*** [30.07]	211.57*** [56.02]	3.78 [26.85]	28.06* [26.19]	28.06 [42.73]
1988#SES						
Quantile	190.64*** [31.25]	227.25*** [30.12]	227.25*** [55.37]	15.35 [26.89]	41.69* [26.24]	41.69* [43.26]
1989#SES						
Quantile	210.20*** [31.30]	249.53*** [30.17]	249.53*** [57.51]	35.77* [26.93]	64.09** [26.28]	64.09* [43.62]
1990#SES						
Quantile	231.73*** [31.33]	273.72*** [30.20]	273.72*** [57.84]	42.01* [26.96]	72.18*** [26.30]	72.18** [43.82]
1991#SES						
Quantile	260.99*** [31.32]	302.40*** [30.18]	302.40*** [58.33]	41.09* [26.95]	71.35*** [26.29]	71.35* [44.50]
1992#SES						
Quantile	230.73*** [31.31]	271.43*** [30.18]	271.43*** [62.24]	58.69** [26.94]	88.88*** [26.29]	88.88** [44.03]
1993#SES						
Quantile	255.25*** [31.29]	295.12*** [30.16]	295.12*** [62.49]	75.14*** [26.92]	105.06*** [26.27]	105.06** [44.68]
1994#SES						
Quantile	278.12*** [31.27]	316.88*** [30.13]	316.88*** [61.01]	92.35*** [26.91]	121.75*** [26.25]	121.75*** [44.88]
1995#SES						
Quantile	304.03*** [31.24]	341.66*** [30.11]	341.66*** [60.72]	101.88*** [26.88]	130.69*** [26.22]	130.69*** [44.40]
1996#SES						
Quantile	287.02*** [31.22]	323.35*** [30.08]	323.35*** [61.08]	127.29*** [26.86]	155.28*** [26.21]	155.28*** [45.47]
1997#SES						
Quantile	328.42*** [31.17]	363.59*** [30.04]	363.59*** [61.17]	142.64*** [26.82]	169.83*** [26.17]	169.83*** [46.07]
1998#SES						
Quantile	320.25*** [31.14]	354.29*** [30.01]	354.29*** [61.39]	149.23*** [26.80]	175.60*** [26.14]	175.60*** [46.34]
1999#SES						
Quantile	334.69*** [31.10]	367.65*** [29.98]	367.65*** [61.84]	152.81*** [26.76]	178.29*** [26.11]	178.29*** [45.87]
2000#SES						
Quantile	342.74*** [31.05]	374.51*** [29.93]	374.51*** [61.20]	165.24*** [26.72]	189.65*** [26.07]	189.65*** [46.14]
2001#SES						
Quantile	310.23*** [30.99]	341.83*** [29.87]	341.83*** [61.22]	164.15*** [26.67]	188.73*** [26.02]	188.73*** [46.21]
2002#SES						
Quantile	355.50*** [30.95]	386.97*** [29.82]	386.97*** [61.08]	173.02*** [26.63]	197.83*** [25.98]	197.83*** [46.53]
2003#SES						
Quantile	361.74*** [30.89]	393.18*** [29.77]	393.18*** [61.24]	192.69*** [26.58]	217.64*** [25.93]	217.64*** [48.62]

2004#SES						
Quantile	355.07*** [30.81]	386.26*** [29.69]	386.26*** [61.31]	201.87*** [26.51]	226.71*** [25.86]	226.71*** [48.23]
2005#SES						
Quantile	364.41*** [30.76]	395.43*** [29.64]	395.43*** [61.49]	225.73*** [26.47]	250.55*** [25.82]	250.55*** [48.35]
2006#SES						
Quantile	346.52*** [30.68]	377.28*** [29.57]	377.28*** [61.58]	223.06*** [26.40]	247.76*** [25.76]	247.76*** [48.06]
2007#SES						
Quantile	361.79*** [30.59]	392.13*** [29.48]	392.13*** [61.33]	207.10*** [26.32]	231.46*** [25.68]	231.46*** [50.15]
2008#SES						
Quantile	359.14*** [30.52]	389.23*** [29.41]	389.23*** [61.44]	228.64*** [26.26]	252.76*** [25.62]	252.76*** [48.65]
2009#SES						
Quantile	357.84*** [30.44]	387.82*** [29.34]	387.82*** [60.36]	226.29*** [26.19]	250.26*** [25.55]	250.26*** [48.65]
2010#SES						
Quantile	356.26*** [30.38]	385.87*** [29.28]	385.87*** [60.95]	225.48*** [26.14]	249.09*** [25.50]	249.09*** [48.07]
2011#SES						
Quantile	335.72*** [30.38]	364.94*** [29.28]	364.94*** [60.75]	225.62*** [26.14]	249.07*** [25.50]	249.07*** [48.13]
2012#SES						
Quantile	344.54*** [30.37]	373.52*** [29.27]	373.52*** [60.83]	228.16*** [26.13]	251.40*** [25.49]	251.40*** [47.92]
2013#SES						
Quantile	357.29*** [30.34]	386.03*** [29.24]	386.03*** [61.10]	249.88*** [26.11]	272.85*** [25.47]	272.85*** [47.76]
2014#SES						
Quantile	354.07*** [30.30]	382.73*** [29.20]	382.73*** [61.37]	232.32*** [26.07]	254.99*** [25.43]	254.99*** [47.52]
2015#SES						
Quantile	353.59*** [30.23]	381.93*** [29.13]	381.93*** [61.21]	248.82*** [26.01]	270.94*** [25.38]	270.94*** [47.50]
2016#SES						
Quantile	380.00*** [30.43]	414.43*** [29.33]	414.43*** [61.36]	250.93*** [26.18]	278.84*** [25.54]	278.84*** [48.21]
2017#SES						
Quantile	372.69*** [30.60]	413.29*** [29.49]	413.29*** [62.77]	256.47*** [26.33]	290.08*** [25.69]	290.08*** [49.02]
2018#SES						
Quantile	390.77*** [30.73]	437.78*** [29.62]	437.78*** [63.88]	258.51*** [26.44]	297.89*** [25.80]	297.89*** [49.87]
2019#SES						
Quantile	381.37*** [30.83]	434.58*** [29.72]	434.58*** [63.46]	251.72*** [26.53]	296.50*** [25.89]	296.50*** [50.43]
2020#SES						
Quantile	521.71*** [30.89]	581.02*** [29.78]	581.02*** [63.12]	357.26*** [26.58]	407.20*** [25.94]	407.20*** [50.28]
Population per square mile		0.07***	0.07**		0.05***	0.05**

		[0.00]	[0.03]		[0.00]	[0.03]
Percent Black		3.67***	3.67***		2.05***	2.05***
		[0.05]	[0.21]		[0.05]	[0.17]
Percent Hispanic		-1.48***	-1.48***		-1.29***	-1.29***
		[0.09]	[0.46]		[0.08]	[0.43]
Percent foreign-born		2.43***	2.43*		5.83***	5.83**
		[0.27]	[3.06]		[0.24]	[3.23]
Intercept	1612.69***	1591.20***	1591.20***	1013.74***	980.04***	980.04***
	[16.50]	[15.93]	[43.25]	[14.20]	[13.87]	[30.32]
R-squared	0.33	0.38	0.38	0.11	0.15	0.15
Adjusted R-squared	0.33	0.38	0.38	0.11	0.15	0.15

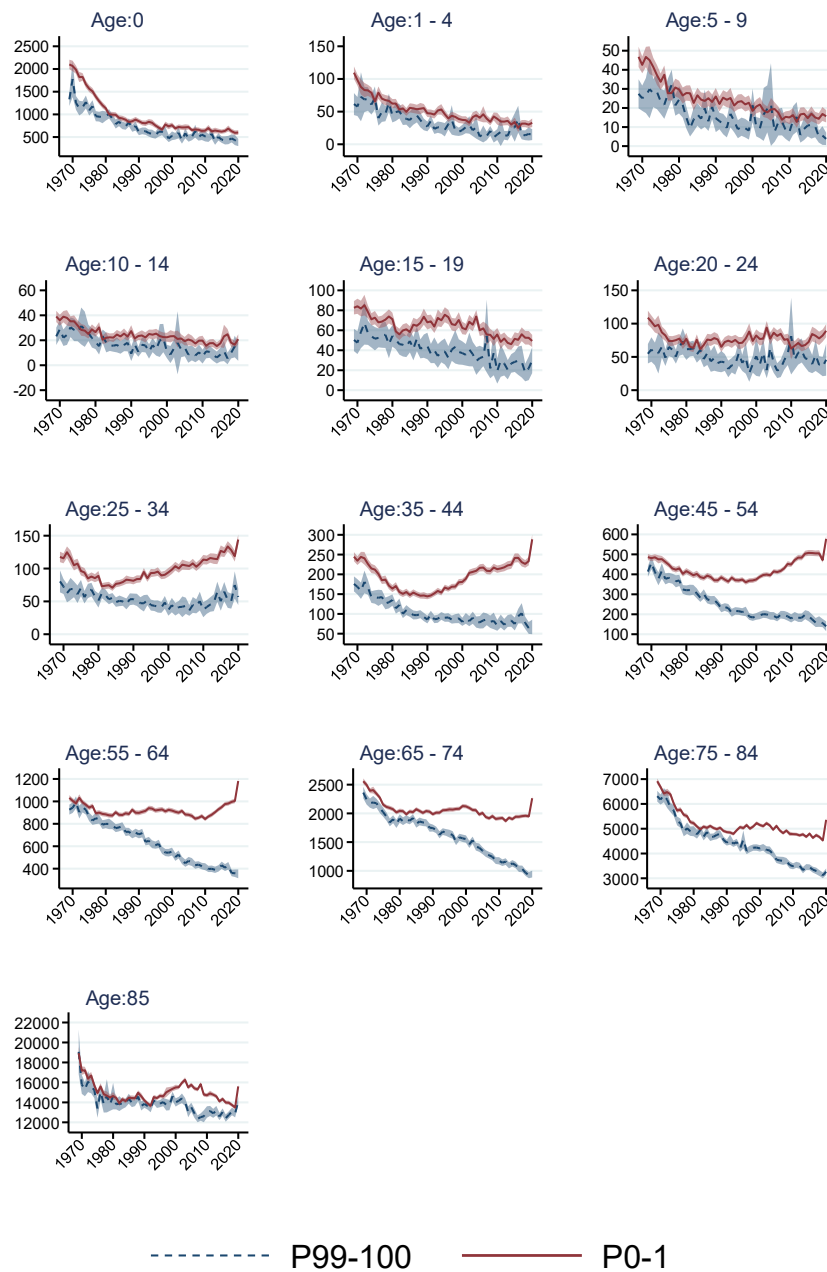
Notes: † p<0.1, * p<0.05, ** p<0.01, *** p<0.001

Figure S4.1 The Predicted Mortality Rates for the Highest SES Percentile (P99-100) and Lowest SES Percentile (P0-1), by Age Groups, 1969-2020, Males



Note: Shaded areas represent 95% confidence intervals. The predicted mortality is calculated from OLS regression models in Table 4.6. With red line representing the Low SES percentile (P0-1) and blue line representing high SES percentile (P99-100)

Figure S4.2 The Predicted Mortality Rates for The Highest SES Percentile (P99-100) and Lowest SES Percentile (P0-1), by Age Groups, 1969-2020, Females



Note: Shaded areas represent 95% confidence intervals. The predicted mortality is calculated from OLS regression models in Table 4.6. With red line representing the Low SES percentile (P0-1) and blue line representing high SES percentile (P99-100)

Chapter Five: Changes in Disease Patterns and Mortality Inequality

Introduction

The preceding chapter focused on temporal changes in socioeconomic (SES) inequalities in all-cause mortality, as well as the diverging trends observed across different age groups. The findings indicated a rise in all-cause mortality inequality from the late 1960s to 2020. However, this overarching trend conceals divergent patterns across specific causes of death. In this chapter, I will delve deeper into the trends in mortality inequalities categorized by cause of death. The objectives of this chapter are as follows:

Firstly, this chapter aims to illustrate that different diseases (causes of death) display varying levels and trends in mortality inequality. During any given time period, socioeconomic (SES) inequalities in mortality do not change uniformly for all diseases, but rather they may narrow for some diseases while widening for others. This chapter seeks to provide evidence on how the evolution of medical knowledge and changing disease patterns helped to shape trends in cause-specific mortality inequality. The disease burdens evolve over time under complex social, economic, medical, and environmental changes. Specifically, this chapter aims to demonstrate that mortality inequality from infectious diseases has considerably reduced due to widespread access to technologies such as vaccines, sanitation, and improved standards of living. Conversely, since the 1960s, mortality inequality stemming from degenerative diseases (especially cardiovascular diseases, cancer, and other major non-communicable diseases like COPD and diabetes) has widened, becoming the dominant contributor to overall inequality. Degenerative diseases have surpassed infectious diseases in their impact on overall patterns of inequality.

This chapter also intends to present how the widening inequalities resulting from degenerative diseases primarily affect later stages of life, with minimal influence on early life. This pattern across age groups may help explain the divergent mortality trends by age groups observed in the previous chapter. Additionally, this chapter examines how recent developments in the epidemiological transition, including the reemergence of infectious diseases (including HIV and Covid-19), the deceleration of decline in heart disease, and the rise of conditions like diabetes and behavioral related death (e.g., violence, suicide, drug-overdose and alcoholism), contribute to understanding the most recent trends of mortality inequalities in the United States.

Cause-of-Death Data

In this chapter, I utilize the same sources of mortality data, population data, and socioeconomic data as introduced in the previous chapter. I calculate county-level US mortality rates (1969-2020) based on age, sex, and cause of death, and merge them with county-level socioeconomic variables.

Over time, the coding of causes of death has changed as the ICD has been updated to reflect the evolution of disease patterns and updates in medical knowledge. For the mortality records in the United States for the period 1969-1978, causes-of-death were coded in accordance with the Eighth International Classification of Diseases, Adapted for Use in the United States (ICDA-8). For the data years 1979-1999, cause-of-death data were coded in accordance with the ninth International Classification of Diseases (ICD-9). After 1999, the cause-of-death data were coded in accordance with tenth International Classification of

Disease(ICD-10). For details in the changes of coding from ICD-8 to ICD-10, see the CDC website¹⁶.

Based on the Global Burden of Disease (GBD) program and Epidemiological Transition Theory (Mathers et al., 2006), diseases and causes of death can be classified into three broad groups: 1) Group I, which includes communicable, maternal, neonatal, and nutritional disorders (referred to here as infectious diseases); 2) Group II, consisting of non-communicable diseases (or degenerative diseases, referred to as non-communicable diseases); and 3) Group III, encompassing injuries.

For this analysis, I calculate cause-specific mortality rates for the 3 broad groups (infectious diseases, non-communicable diseases, and injuries), and then conduct separate analyses for several major diseases within each of these three groups. For instance, within non-communicable diseases, I analyze the mortality rates for: 1) cardiovascular diseases (e.g., heart disease, stroke), 2) cancer (e.g., lung cancer, colon cancer, etc.), 3) chronic obstructive pulmonary disease (COPD), diabetes, chronic liver disease and cirrhosis, and other chronic diseases. This analysis allows us to examine the divergent trends in mortality inequality alongside the unique trajectory of innovations in diagnostics and treatment for each disease. Similarly, I explore several major infectious diseases (e.g., influenza and pneumonia, HIV) and injuries (e.g., unintentional injuries, suicide, violence). Additional details regarding the grouping of diseases can be found in Table 5.1.

[-Table 5.1, Global burden of disease, group I, II, III—ICD8-10 codes--]

The specific diseases chosen for analysis are based on three important criteria. Firstly, these diseases are among the leading causes of death in recent decades. Focusing on these

¹⁶ https://www.cdc.gov/nchs/data_access/cmf.htm

diseases could capture the key transitions in mortality trends over the past few decades. Secondly, these diseases have witnessed notable advancements in medical prevention and treatment, which (based on our theoretical framework) would show widening or narrowing of inequalities associated with these specific diseases. Thirdly, the ICD-8, ICD-9, and ICD-10 codes for the disease are provided by previous studies (Centers for Disease Control and Prevention, 2007; Lopez et al., 2006; National Center for Health Statistics, 2021a) to make the cross-time comparison possible. All the ICD codes by cause-of-death are in appendix Table S5.1 and S5.2.

Analytical Strategy

OLS regression models predicting cause-specific mortality with year, SES, and interaction of year and SES are used. I run the OLS models by each cause-of-death to see how the cause-specific trends of mortality inequality.

Cause specific Mortality_i

$$= \beta_0 + \beta_1 Year_i + \beta_2 SES_i + \beta_3 Year_i \cdot SES_i + \pi_4 Controls + e_i$$

where i represents the county, year ranges from 1969 to 2020. The model predicts the mortality rates at the county level as a function of county-level SES and year. Year enters the model as dummies. SES enters the model as a continuous variable. The predicted mortality of the richest county-group (the 100th percentile, referred to as P99-100) and poorest county group (the 1st percentile, referred to as P0-1) are calculated based on the OLS models to capture the absolute level of difference in mortality as one moves from the lowest SES rank to the highest.

Then I stratify the above analyses by age groups to examine how trends in mortality inequality for each cause of death varies across age groups or life course stages.

Results

Trends in Mortality Inequality by Cause of Death

In the previous chapter, I have shown that the overall mortality inequality has been rising since the 1960s. But these overall trends conceal the diverging trends by cause of death. In this section, I present how different diseases have unique trends in mortality inequality. I will examine the trends of the broad groups of diseases (e.g., infectious diseases or non-communicable diseases). Within each of these broader categories, I will select several major causes of death to investigate specific mortality trends. This examination will also involve assessing how these trends align with the timeline of medical advancements for each disease. The predicted mortality rates by high/low SES county groups based on Ordinary Least Squares (OLS) models will be presented, although the detailed regression tables will not be presented here to conserve space.

Infectious diseases

Figure 5.1 illustrates the predicted mortality inequality pertaining to infectious diseases from regression models, with separate panels for males (panel a) and females (panel b). Major medical innovation events are annotated within the figure, providing contextual reference for the improvements in medical innovation.

[--Figure 5.1 about here--]

In general, the male mortality rates are higher than female rates, but the male and female rates have very similar trends in inequality. The mortality inequality in infectious diseases decreases in the early 1970s because the lower SES groups declined faster than the higher SES group. The declining overall trends and narrowing disparities are consistent with the long-term improvement in sanitation and hygiene and the implement of vaccination in the

19th century to and early 20th century. During the 1980s and early 1990s, mortality from infectious diseases increased for both high and low SES groups, which is consistent with the emergence of HIV and drug-resistant strains of some old infectious diseases during this period (CDC, 1999). However, the mortality inequality at this period remains stable and at a low level (close to zero).

Since approximately 1995, there has been a decline in infectious disease mortality rates observed among both high and low socioeconomic status (SES) groups, regardless of gender. However, mortality inequality increases because high SES males and females saw steeper declines. The mortality rates from influenza and pneumonia decline steeply after 2000, with a deeper decline for high SES group (see appendix Figure S5.1). This pattern is consistent with the use of pneumococcal conjugate vaccine (PCV7) since 2000 (which may have been adopted sooner by higher SES individuals). The emergence of inequalities from HIV since 1995 (when HAART for HIV is introduced) also contributes to the increase in mortality inequality. Mortality rates for all other infectious diseases except influenza/pneumonia and HIV also experienced an increase during the 1980s and 1990s, followed by widening inequalities after the mid-1990s (refer to Appendix Figure S5.1). The mortality inequality increases dramatically during the COVID-19 period (2020). These trends warrant further investigation in future research.

Non-communicable diseases

Figure 5.2 illustrates the predicted mortality inequality associated with non-communicable diseases, presented separately for males (panel a) and females (panel b). In general, males have higher mortality rates than females. At the beginning of the 1970s, the mortality rates from non-communicable diseases remained high for both genders. However, the level of inequality during this period was relatively low. Since the early 1970s, both

males and females demonstrate a decline in mortality rates, while the decline is more salient for high SES group (the low SES female even present a stagnant or even worsening mortality level since the 1980s). As a result, the mortality inequality increased over time.

[--Figure 5.2 about here--]

While both males and females experienced an increase in inequality, the reasons and timing of this increase differ between the two genders. For males, the steeper declines in mortality are observed among those in the high socioeconomic status (SES) group compared to the low SES group. However, among females, a stagnant or even worsening trend since 1980 are observed among those in the low SES group, while those in the high SES group maintain steady declines. In terms of timing, the increase in inequality occurs earlier for males and then the trend levels off, while it seems to still be increasing for women.

To be specific, the increase in male mortality inequality occurs since the 1970s, accelerating in the 1980s and 1990s. Since around 2000, the increase in male mortality inequality has slowed down due to a deceleration in the decline of mortality among the high socioeconomic status (SES) group, coupled with an acceleration in the decline among the low SES group. The mortality inequality for females remained relatively stable during this period during the 1970s and 1980s because high and low SES females experience similar declines in mortality rates. However, between 1985 and 2005, there has been a significant acceleration in female mortality inequality due to the stagnation of gains or an actual worsening for low SES women. . After 2005, the increase in female mortality inequality has also slowed down, primarily because the decline of mortality among the high SES group has stagnated. These gender differences in mortality inequality trends raise concern about the status of women's health, especially for those with limited resources (cause-specific analyses below provides more details).

However, mortality inequality has once again increased in recent decades (since 2010 for males and 2015 for females) due to an accelerated decline in mortality among the high socioeconomic status (SES) group. Additionally, the COVID-19 pandemic (in 2020) has contributed to an additional increase in mortality inequality, as lower SES county groups have experienced higher mortality increases.

Cardiovascular Diseases

The major medical innovation events annotated in Figure 5.2 present the contextual background for the decline in non-communicable disease mortality and the rise of inequality. Based on Fundamental Cause Theory, mortality inequality increases as the emergence of these new medical tools. General discussions about non-communicable could obscure contrasting patterns among different diseases. In this section, I will delve into the specifics of major non-communicable diseases to illustrate the distinct inequality trends. Figure 5.3, Figure 5.4, and Figure 5.5 further show cause-specific mortality inequality trends in several major non-communicable diseases, shedding lights on the medical innovations in contributing to widening inequalities.

Cardiovascular diseases (CVD), which encompass heart diseases, strokes, and other conditions like peripheral arterial disease, stand as the leading cause of death in the US. In Figure 5.3, I present the trends over time in mortality inequality for CVD. CVD mortality has been declining over time, and the widening inequality is clearly due to the faster decline in mortality among high SES county groups. Within the realm of CVD, heart diseases play a pivotal role in the surge of inequality, and even dominate the rising inequality in overall non-communicable diseases. This is in parallel with the emergence of treatment for heart diseases and medical knowledge about the risk factors like smoking, diet, lifestyle, blood pressure, and cholesterol during 1970s, 1980s, and 1990s (see annotations in Figure 5.2). Again, the

increase in inequality occurs earlier for males and then levels off, while it seems to still be increasing for women. But inequality has increased again in recent years.

[-Figure 5.3 about here--]

Stroke mortality has consistently decreased over time. Previous studies showed that the decline in stroke mortality benefits from control of hypertension, hyperlipidemia, and tobacco use. The inequality in stroke mortality has diminished as the lower socioeconomic (SES) county group catches up, with an even more rapid decline in mortality. The decline slowed down significantly after 2000, as both low and high SES county groups showed a plateau in mortality rates. But the reason for narrowing stroke mortality inequality has been not well understood. Further, the patterns of inequality differ dramatically between HD and stroke, which is puzzling because both share many of the same risk factors.

Cancer

The inequality in cancer mortality has witnessed a substantial increase over time (see Figure 5.4). Lung cancer and colon cancer play a pivotal role in the surge of inequality. In the early 1970s, lung cancer mortality shows an increase for both high and low socioeconomic status (SES) groups, with the inequality being relatively low. However, for males, the inequality in lung cancer mortality increases after the 1970s due to a more significant decline in the high SES group. The peak of male lung cancer mortality inequality occurs around 2000, followed by a decline, as the lower SES group exhibits a faster decline compared to the high SES group (refer to Figure 5.4). The female lung cancer shows an increase for both high and low socioeconomic status (SES) groups from 1970 to 1995, with the inequality being relatively low. However, since 1995, a decline in lung cancer mortality has been observed among high SES females, while low SES females continue to experience an increase (until 2010). Consequently, the mortality inequality for female lung cancer has dramatically

increased from 1990 to 2020. This is consistent with the public report on smoking and changes in smoke behavior in the United States. The decline in lung cancer mortality among females occurred much later compared to males, aligning with the delay in the changes in smoking behaviors among females in the United States.

[-Figure 5.4 here--]

The mortality inequality of colorectal cancer has been widening over time. During the early 1970s and 1980s, colorectal cancer mortality rates were higher among the high socioeconomic status (SES) county group. This could be due to a higher likelihood of contracting the cancer or a greater probability of receiving a diagnosis. However, mortality inequality from colorectal cancer has increased from 1970 to 2020. Around 1990, a crossover point was observed between the high and low SES groups, as shown in Figure 5.4. The widening mortality inequality coincides with the introduction of colorectal cancer screening recommendations in the 1990s, as well as the public awareness of diet and lifestyle in reducing cancer risks. These screening techniques and lifestyle changes were probably adopted first by high SES adults, thereby leading to widening inequality in mortality. The shifts in smoking behavior, lifestyle choices, diet, and advancements in cancer screening technology have reduced risks in multiple other kinds of cancer (e.g., breast cancer or other kinds of cancer, see Appendix S5.2 for more details), leading to an increase in mortality inequality from cancer over time.

COPD, Diabetes, and other non-communicable diseases

Mortality inequality from other major non-communicable diseases (e.g., COPD, diabetes, chronic liver conditions) has also increased since the 1970s and 80s as mortality declines have been greater for high SES. The timing of these shifts reflects differences in

when interventions and treatments became available and more widespread, and as risk factors changed (e.g., smoking, obesity).

The mortality inequality from chronic obstructive pulmonary disease (COPD) follows a similar pattern to lung cancer mortality, with very low inequality in 1970s and a crossover in mortality in the mid-1980s for males and 1990 for females (see Figure 5.5). The male COPD mortality inequality increases dramatically since 1980; the female COPD mortality inequality remains relatively stable from 1970 to 1990 and increases since the 1990s, albeit later than in males. These gender differences in COPD and lung cancer mortality patterns—both related to the tobacco use—contribute to the diverging overall mortality trends observed between males and females.

[-Figure 5.5 here-]

The mortality inequality from diabetes remains relatively low until the mid-1980s. From the mid-1980s to 2005, there is an increase in diabetes mortality rates for both high and low socioeconomic status (SES) groups, with a faster increase observed in the low SES group, leading to a widening mortality inequality. Since 2005, there has been a significant decrease in diabetes mortality rates overall. While low SES group has experienced only minor decreases or even increases in mortality rates after 2010. The increasing inequality in diabetes mortality aligns with the improvement in medical knowledge regarding intensive control of glucose levels, blood pressure, and lipid levels, which has led to a reduction in diabetes mellitus complications since the 1990s. Additionally, public guidelines and recommendations for lifestyle interventions in diabetes prevention and management have been in place since the late 1990s, contributing to this trend. To the extent that higher SES individuals are more likely to utilize newer techniques for controlling their glucose levels and to alter their lifestyle, this could account for the widening mortality inequality in diabetes.

However, it is important to note that many diabetes patients pass away from complications related to diabetes, such as vascular disease, rather than directly from diabetes (Gregg et al., 2018; McEwen et al., 2011; Raghavan et al., 2019). As a result, the death certificates use in this study may not fully capture the complete impact of diabetes on mortality rates and disparities.

The mortality inequality from chronic liver conditions also increases from 1980s to mid-1990s, then levels off in 2000s, but increases again in recent years. This might be related to rising alcohol use among those with lower socioeconomic status (Dawson et al., 2015; Mellinger, 2019).

To sum up, the mortality inequality from heart diseases, cancer (especially lung cancer and colorectal cancer), and COPD are the main contributors to the increase in non-communicable diseases mortality inequality during 1970-1990. The increase in mortality inequality from heart diseases aligns with the temporal change in treatment for heart diseases and medical knowledge about the risk factors like smoking, diet, lifestyle, blood pressure, and cholesterol during 1970s. The increase in mortality inequality from COPD and lung cancer are especially consistent with the public awareness on smoking and changes in smoke behavior in the United States. Since 1990, mortality inequalities have leveled off or even narrowed for diseases like CVD and male lung cancer, but they have widened for others such as COPD, diabetes, and liver conditions. The widening mortality inequalities from these conditions coincided with the delayed female smoking quit behavior, lifestyle choices, diet, and behavioral risks like control of glucose levels and alcohol use.

Injuries

During the 1970s and 1980s, there was a notable decrease in mortality resulting from injuries, particularly unintentional injuries, among both males and females. This decline

coincided with significant public health advancements in motor vehicle safety, including the implementation of the road safety legislation, as well as enhancements in vehicle safety design such as headrests, safety belts, and improvements in road environments during the 1960s and 1970s (refer to the key events highlighted in Figure 5.6). Injuries mortality (especially unintentional injury) exhibited a rapid decline in both low and high socioeconomic (SES) county groups, with a particularly steep decrease among the low SES group from 1970 to 1985. This trend suggests that public health advancements in motor vehicle safety quickly benefited both high and low SES groups, leading to a reduction in mortality inequality. However, due to a lack of data in the 1960s, it remains unclear whether mortality inequality increased during the early years of the motor vehicle safety program. It is possible that improvements in motor vehicle safety and road environments might not be closely linked to SES status, as these enhancements (safer road and vehicle designs, or road safety legislation) benefit everyone.

[--Figure 5.6 here--]

Between 1985 and 1990, the high SES group experienced a much swifter decline in both unintentional injury and violence mortality, leading to a significant surge in mortality inequality during this period. From 1990 to the early 2010s, both male and female injury-related mortality increased, mainly due to unintentional injuries and suicide (see appendix Figure S5.4). During this time, male inequality decreased as higher SES groups had more injuries, while lower SES groups remained stable. But since 2015, male inequality rose again, mainly due to unintentional injuries, suicide, and violent deaths. The inequalities exacerbated especially in 2020. For females, injury-related mortality inequality consistently rose from 1990 to 2020, driven by unintentional injuries and suicides (appendix Figure S5.3), albeit with a modest slope.

These fluctuations in mortality inequality don't correlate well with health innovations. The rise in inequality between 1985 and 1990, the decline in male injury mortality from 1990 to 2015, and the recent increase for both genders aren't easily explained by medical advancements. It seems that violent deaths (e.g., accidents, homicides) and behavior-related deaths (e.g., suicides) are more linked to "social pathology" (R. G. Rogers & Hackenberg, 1987). The worsening firearm injuries, which disproportionately affect populations with fewer resources, might contribute to an increase in levels of death from suicide, homicide, and unintentional injuries (Degli Esposti et al., 2022; Fowler et al., 2015; Garen, 2015; Kena & Truman, 2022).

Summary of Mortality Inequality by Cause of Death

Figures 5.6 and 5.7 present a summary of mortality inequality across different causes of death in a stacked graph, separately for males and females. The overall mortality inequality is divided among various causes of death, and the stacked graph effectively illustrates the changing distribution of mortality inequality by cause of death, spanning from 1970 to 2020.

[--Figure 5.7 here--]

[--Figure 5.8 here--]

Notably, mortality inequalities stemming from infectious diseases decreased in the early 1970s for both genders, experienced a slight resurgence in the 1990s (with a notable spike in 2020). However, during 1970 to 2020, infectious diseases only contributed to a minor portion of the overall mortality inequality.

Then as the improvement in prevention/treatment knowledge for cardiovascular diseases and cancer (especially lung cancer) improved during the first half of the 20th century, heart diseases dominated the increase in mortality inequality. Between 1970 and

1990, the increase in mortality inequality among males is primarily attributed to disparities in heart disease and lung cancer (refer to Figure 5.7). Similarly, the rise in mortality inequality among females is driven by heart disease, though with a slight delay compared to males, extending from 1980 to the early 2000s (as depicted in Figure 5.8). This delay could potentially be linked to the lower prevalence of female tobacco use during the 1970s and 1980s, resulting in a slower rise in mortality inequality related to lung cancer and heart disease.

Post-1990, male mortality inequality originating from heart disease remains relatively stable. As the improvements in knowledge about lifestyle, diet, exercise, cancer screening technology, and blood glucose/blood lipids managements, mortality inequalities related to colorectal cancer and various other cancers, COPD, diabetes, and other chronic diseases, emerge as the main driving factors. Similar patterns obtain for females: inequalities in cancer, COPD, diabetes, and other chronic conditions contribute to an increase in female mortality inequalities after the year 2000.

Mortality inequalities from injuries do not present a clear pattern linked to medical innovation history but might be more related to “social pathology” or firearm crisis, especially in recent decades. However, deaths from injuries exhibit minimal impact on the general trends in mortality inequality distribution. Another issue worth noting is that during the early 1970s and 1980s, unspecified deaths—cases where the actual cause of death is unknown or missing in death records, typically categorized under the "garbage box" in ICD coding—comprise a significant portion of the overall pattern. The shifts in unspecified deaths mainly concentrated among infants aged 0 and oldest old aged 85+ (see Figure 5.9-5.10 for more details) and are not widely understood and could be influenced by alterations in ICD coding regulations.

Diverging Mortality Inequality Trends Across Age Groups

The trends in mortality inequalities from infectious diseases, non-communicable diseases, and injuries vary largely across age groups (see Figure 5.9 and Figure 5.10). At the younger age groups (age 0-25), the inequality story is driven by injuries, except for infants who had higher infections in the 1970s. The inequality in mortality due to infectious diseases is concentrated mainly among infants and children, experiencing a significant decline throughout the 1970s and 1980s. Overall inequality at younger ages shows clear declines over time for males—mainly caused by declines in deaths from injuries. The trends of mortality inequality for young females fluctuated.

[--Figure 5.9 about here--]

[--Figure 5.10 about here--]

As individuals enter middle age, overall mortality inequalities witness an increase, particularly among females aged over 25 and males over 45. Non-communicable diseases play an increasingly significant role in these inequalities, with the importance of injuries declining (especially after age 35). While injury inequalities decrease for young and middle-aged males, they rise over time for middle-aged females, contributing significantly to the overall mortality inequality, especially within the 25-44 age group.

For women aged 45 and over, and males aged 55 and over, non-communicable diseases account for virtually all mortality inequalities, and these inequalities have skyrocketed during this time period. This surge arises from increased mortality inequalities in heart disease, cancer, COPD, diabetes, and chronic liver diseases; the mortality inequality in these diseases increased dramatically among middle-aged and late life, see appendix Figure S5.6 for examples of male heart disease and lung cancer mortality inequalities).

Consequently, the age distribution of mortality inequality shifts progressively toward older age groups. For instance, in 1969-1970 and 1971-1975, the inequality in heart disease mortality peaks between ages 55-64, followed by a decline (resulting in negative inequality among very old ages). However, after 1996-2000, the mortality inequality peaks at the oldest age groups (85+). Similar patterns are evident for cancer, COPD, diabetes, chronic liver diseases, and numerous other non-communicable diseases (graphs omitted for brevity).

Conclusion

In this chapter, I examine trends in mortality inequalities based on three categories of causes, namely infectious diseases, non-communicable diseases, and injuries. The results indicate that all three categories of causes have contributed to the overall increase in inequality, particularly after 1990. However, various diseases demonstrate distinct patterns of inequality growth, with inequalities becoming more pronounced at different points in time.

Inequality in infectious diseases decreased during the 1970s, and experienced a slight rise during the 1990s. Then the inequality has remained relatively stable in recent decades for both males and females, except for the period of the Covid pandemic. On the other hand, non-communicable diseases have witnessed significant inequality growth since the 1980s, primarily due to steeper declines in high socioeconomic status (SES) counties compared to low SES counties. Inequality related to injuries has been on the rise since 1985 for females (especially among middle-life females aged 25-44). While for males, there was a decrease from 1985 to 2010 followed by an increase after 2010. The significant increase in inequalities from injury-related deaths among middle-aged females since 1985, as well as among both genders after 2010, is quite striking and calls for further investigation in the future.

Detailed analysis of specific diseases shows great variability across diseases in the temporal patterns of inequality, often reflecting historical changes in the risk factors, medical

knowledge, and diagnostic tools and treatments that have become available over time. For instance, during the period from the 1970s to the 1990s, alongside the emergence of treatments for heart diseases and changes in behaviors such as smoking, we found that heart disease, lung cancer, and COPD stood out as the primary contributors to the observed increase in inequality. Since the 1990s, there has been a noticeable slowdown in the pace of growth in heart diseases. However, as our knowledge of lifestyle, diet, and cancer screening technologies improved, COPD, diabetes, cancers (except lung), and liver conditions have essentially taken over as the main contributors to inequality, as if passing the baton in a relay race.

Detailed analysis of mortality inequality by age group also shows great variability in the sources of inequality, largely reflecting the differing causes of death by age. Among individuals under the age of 25, inequalities in injuries play a pivotal role in shaping overall mortality inequality. However, as age advances, non-communicable diseases have progressively emerged as increasingly significant contributors to this inequality.

Over time, non-communicable diseases have taken center stage as the primary catalyst for the growth in mortality inequality. This increase has been particularly pronounced among those in middle-age and late life. Consequently, the distribution of mortality inequality has shifted progressively towards older age groups.

These results are generally consistent with my hypothesized expectations based on the theoretical framework. To be specific, the following hypotheses are supported by empirical evidence in this chapter:

- *Hypothesis 1.* Different diseases (causes of death) show divergent magnitude and direction in mortality inequality trends.

- *Hypothesis 2.* The mortality inequality from infectious diseases has been largely reduced.
- *Hypothesis 3.* Mortality inequality from degenerative diseases has widened and now dominates all-cause mortality inequality.
- *Hypothesis 4a-4c.* The widening mortality inequality from degenerative disease is mainly attributed to widening mortality inequalities from cardiovascular diseases (CVD), cancer, other degenerative diseases (e.g., COPD and diabetes, etc.).
- *Hypothesis 5.* The increase of mortality inequalities is more salient among later life stages than earlier life stages. This is because widening inequalities from degenerative diseases affect later life mainly, with little influence on early life.
- *Hypothesis 6.* Age distribution of mortality inequality is also shifted progressively toward older ages.
- *Hypothesis 8b.* The mortality inequality from diabetes and liver diseases has been rising and plays a more and more important role in affecting overall mortality inequality.
- *Hypothesis 9.* The mortality inequality from infectious diseases experienced a reemergence with a minor increase during the 1980s and 1990s, followed by a significant surge during the Covid-19 pandemic of 2020.

Meanwhile, there are two hypotheses that are only partially supported and need to be further discussed:

- *Part of Hypothesis 7.* Mortality inequality from accidents, violent deaths, and suicide dominates the overall inequality among children and young

adolescents. But the inequality becomes more important only for female young and middle-aged adults (age 25-44), but not for males.

This corresponds to the prime working and parenting ages for females. Over this time period, increasing numbers of women were employed, but we also saw increases in divorce and nonmarital childbearing. Perhaps the increasing inequalities in injuries reflect women's greater independence, yet also their greater vulnerabilities, especially among low SES women.

- *Part of Hypothesis 8a.* The mortality inequality in heart diseases has stagnated in the last two decades. However, the mortality inequality from stroke declined steadily to a relatively lower level.

The stroke mortality stands as a rare example where inequality has seen a steep decrease—it can be considered a success story, as all county groups, regardless of wealth, have benefited from treatments and, to a lesser extent, shifts in risk factors. The heart disease mortality also declined over this period, but the pace slowed after 1990. Inequality peaked in the 1990s (1995 for males, 2000 for females). Unfortunately, instead of continuous decline, the trend plateaued, with minor reduction between 2000 and 2010, followed by an increase after 2010. If recalling Clouston's four stage ideal type, the transition mortality inequality in heart diseases stopped in stage 2 and could not move to stage 3, with stagnated but persistent inequality over the past 20 years. The inequality even increased after 2015. This stagnation in inequality is quite striking. Reasons for differing inequality trends between heart diseases and stroke remain unclear and warrant further research. One potential explanation is linked to differences in their treatments. Strokes are considered crisis events, and the ability to respond effectively has significantly improved, ostensibly for anyone who arrives at an emergency

room with symptoms. While heart diseases also involve crisis events such as heart attacks, it encompasses chronic and progressive conditions that develop over an extended period.

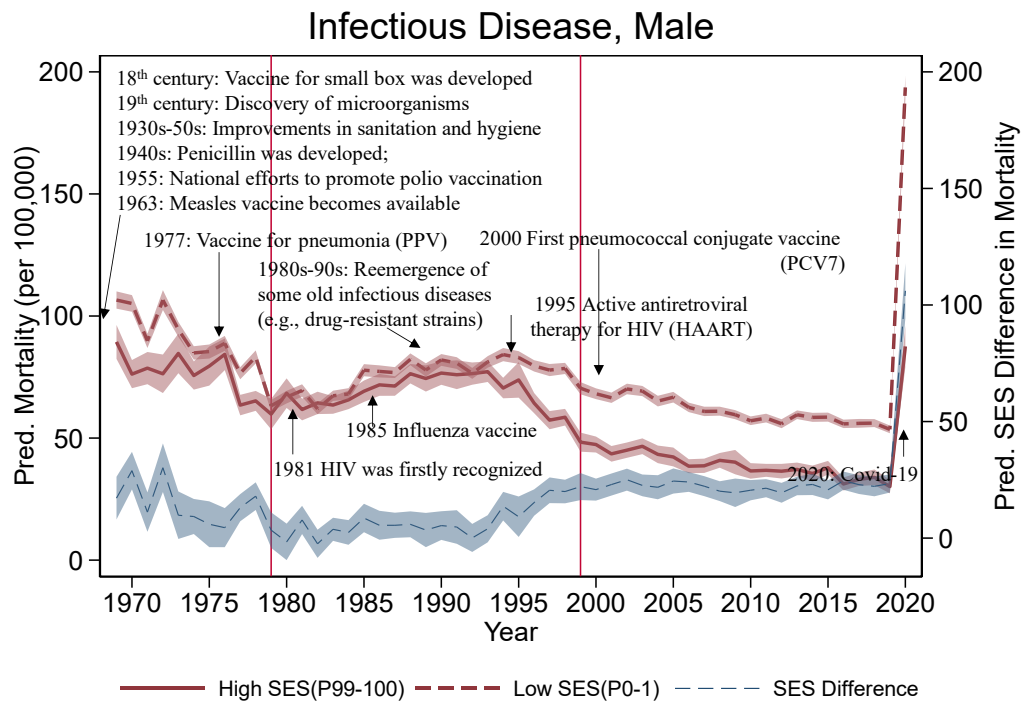
Tables and Figures

Table 5.1 Global Burden of Disease Study Classification Group I, II, and III of Cause-Of-Death, and the Major Diseases Analyzed in This Study.

Three groups of diseases	Detailed Group of diseases
I. Communicable, maternal, perinatal, and nutritional conditions	(1) Influenza and pneumonia (2) HIV (3) other infectious diseases
II. Non-communicable diseases	(1) heart disease (2) stroke (3) other CVD (4) lung cancer (5) other cancer (except lung) (6) COPD (7) diabetes (8) chronic liver disease and cirrhosis (9) Other chronic diseases.
III. Injuries	(10) unintentional injuries (11) suicide (12) violence (13) other injuries
Unspecified death	Insufficiently specific or implausible cause of death codes

Figure 5.1 The predicted mortality by SES percentile (left Y axis) and the predicted SES difference in mortality (right Y axis) for infectious diseases (shaded areas represent 95% confidence intervals).

a. Male



b. female

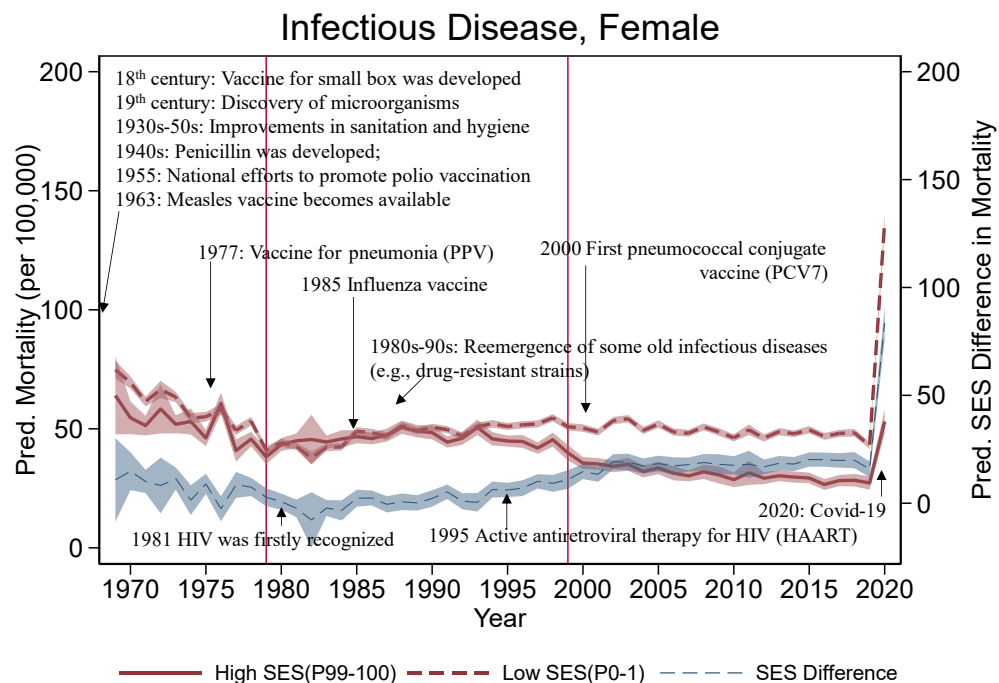
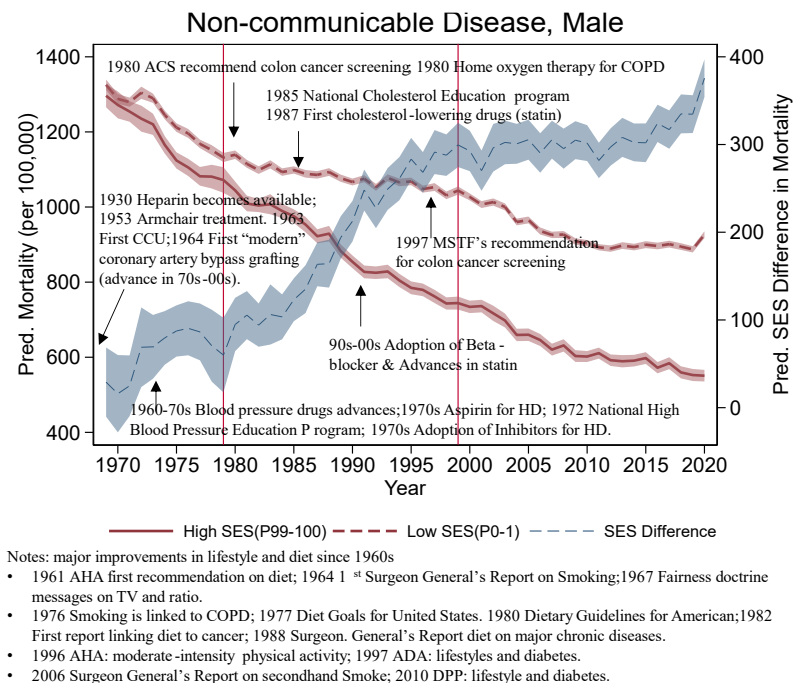


Figure 5.2 The predicted mortality by SES percentile (left Y axis) and the predicted difference in mortality (right Y axis) for noncommunicable diseases (shaded areas represent 95% confidence intervals).

a. Male



b. Female

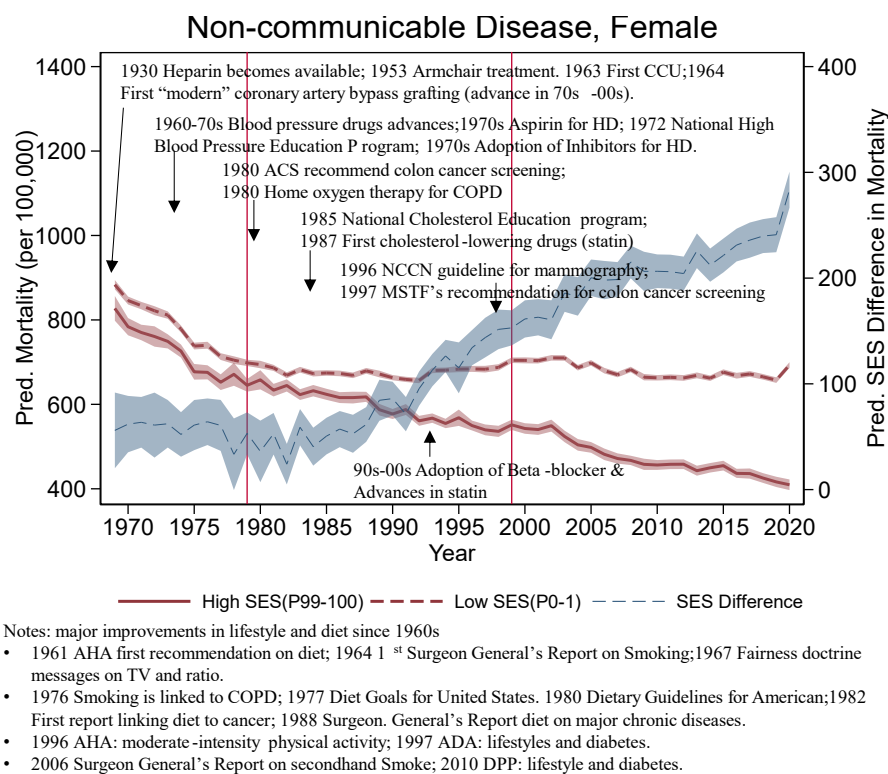


Figure 5.3 The predicted mortality by SES percentiles (left Y axis) and the predicted SES difference in mortality (right Y axis) for cardiovascular diseases (shaded areas represent 95% confidence intervals).

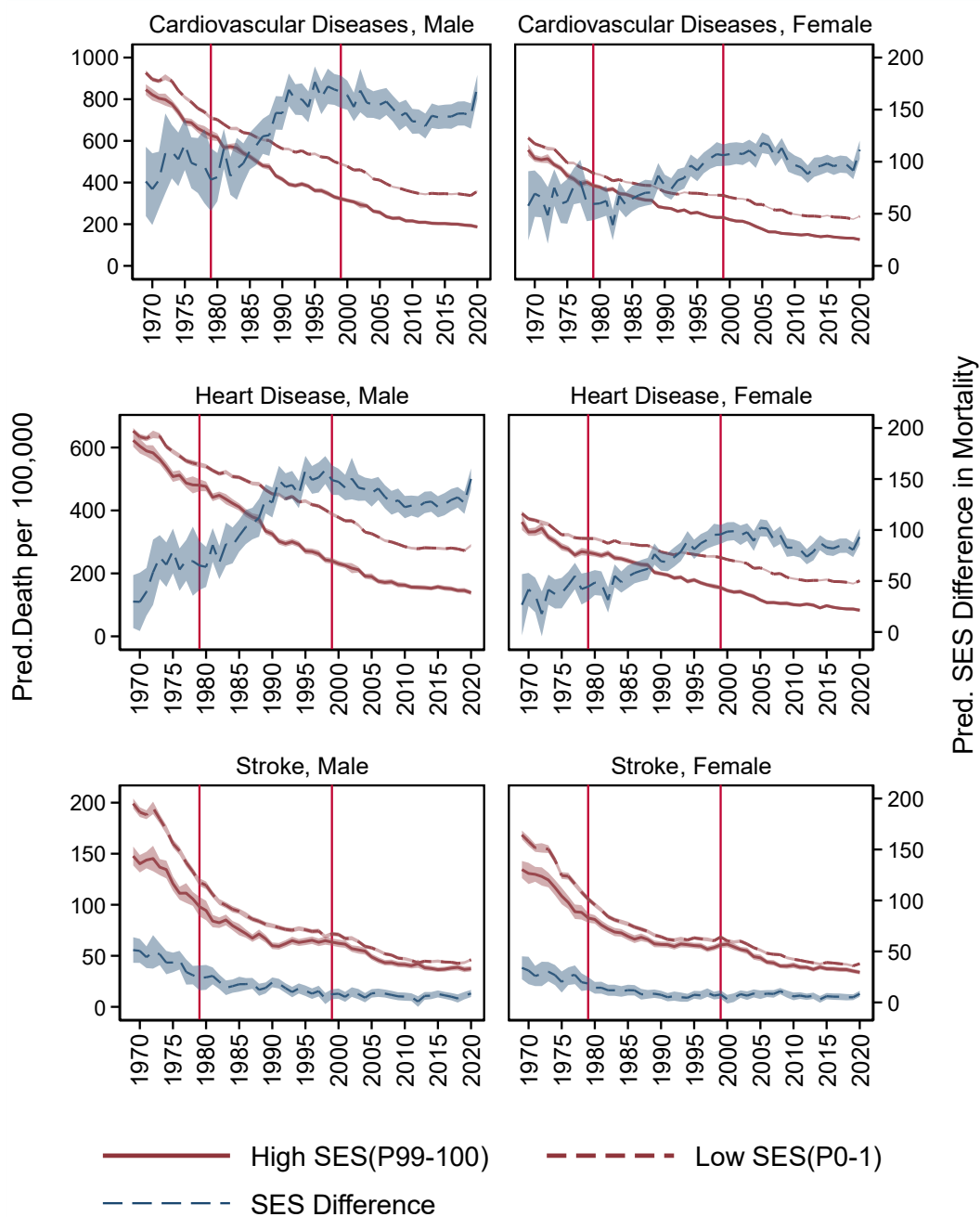


Figure 5.4 Trends in mortality inequality cancer (shaded areas represent 95% confidence intervals).

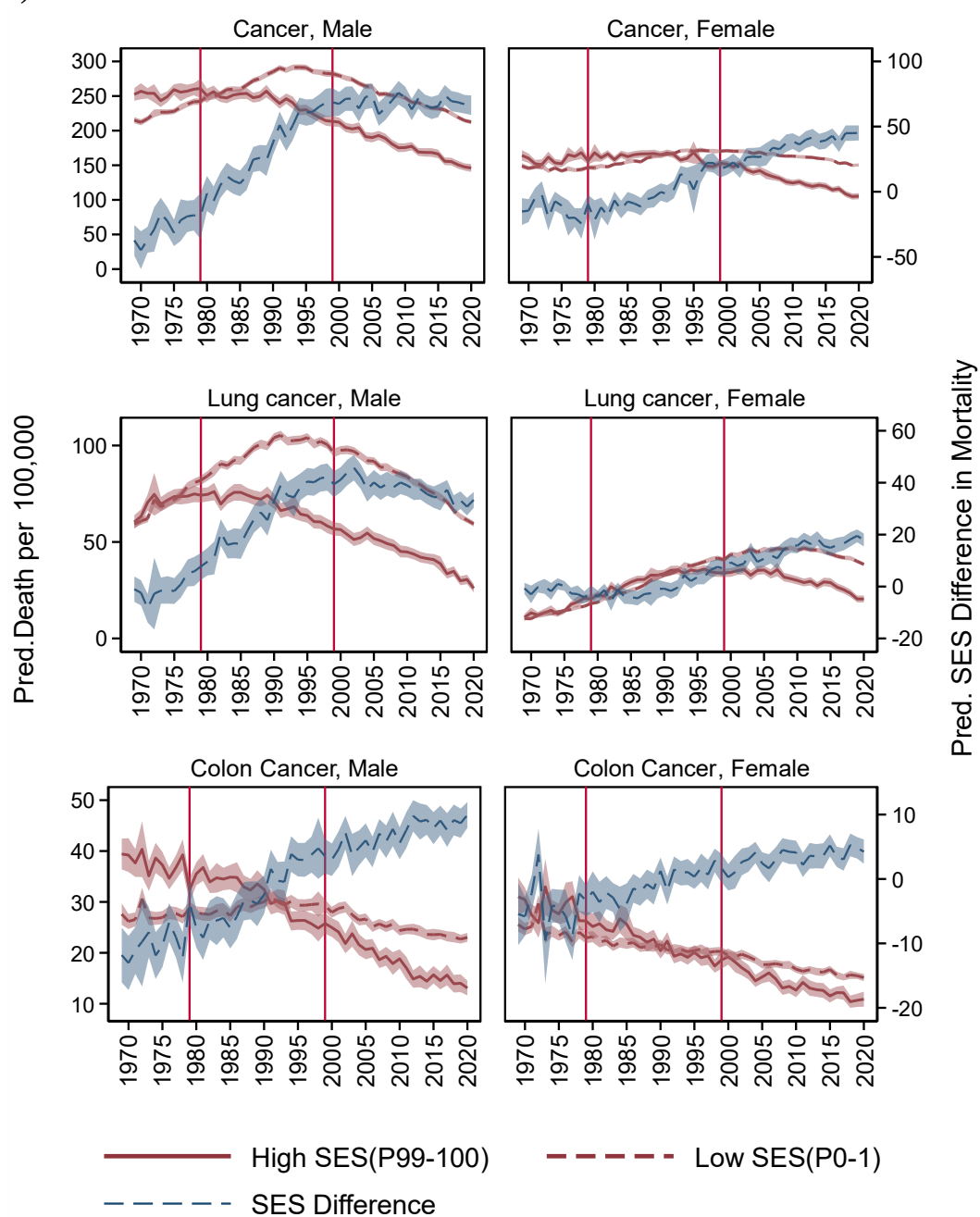


Figure 5.5 Trends in mortality inequality of COPD, diabetes, and chronic liver diseases (shaded areas represent 95% confidence intervals).

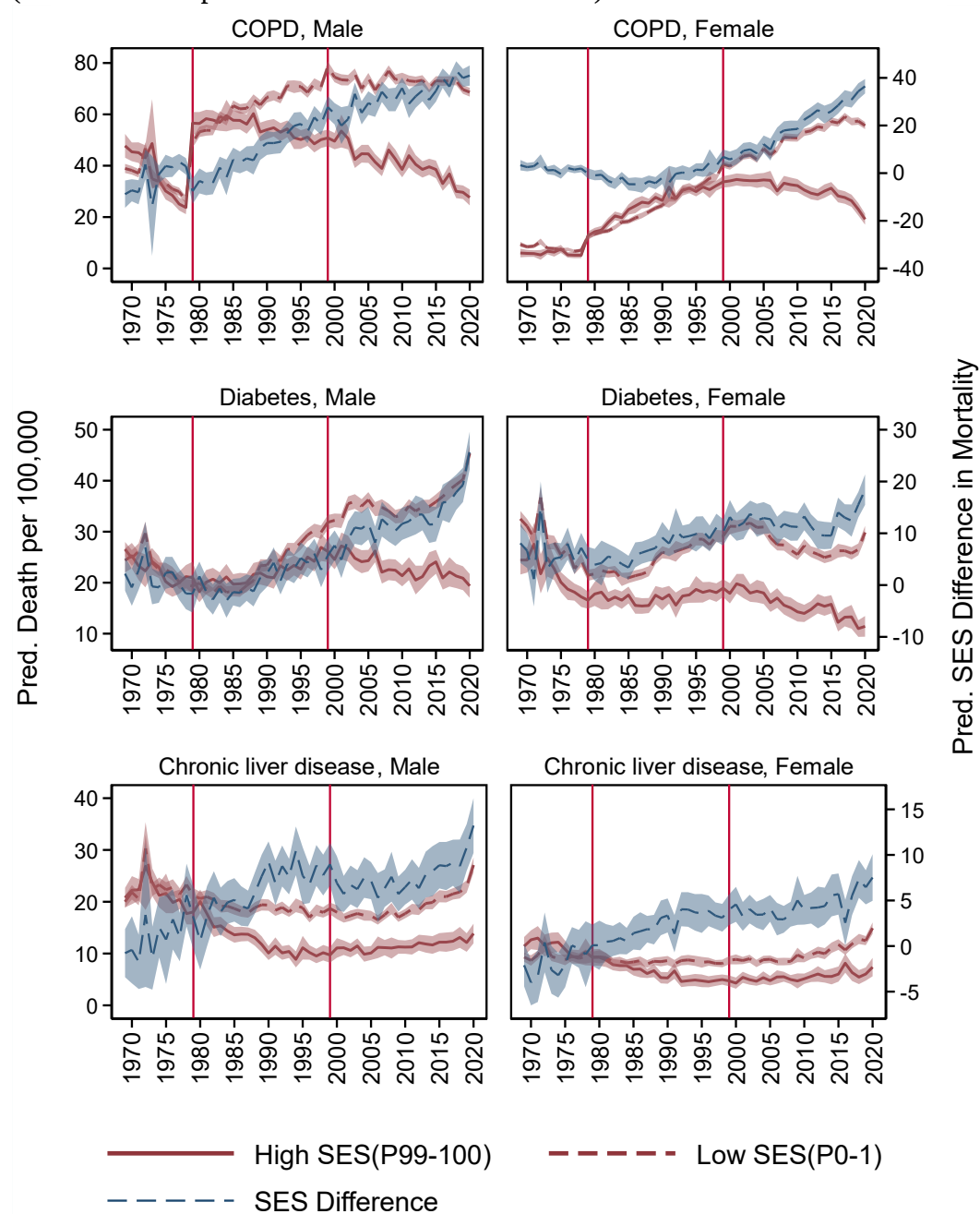
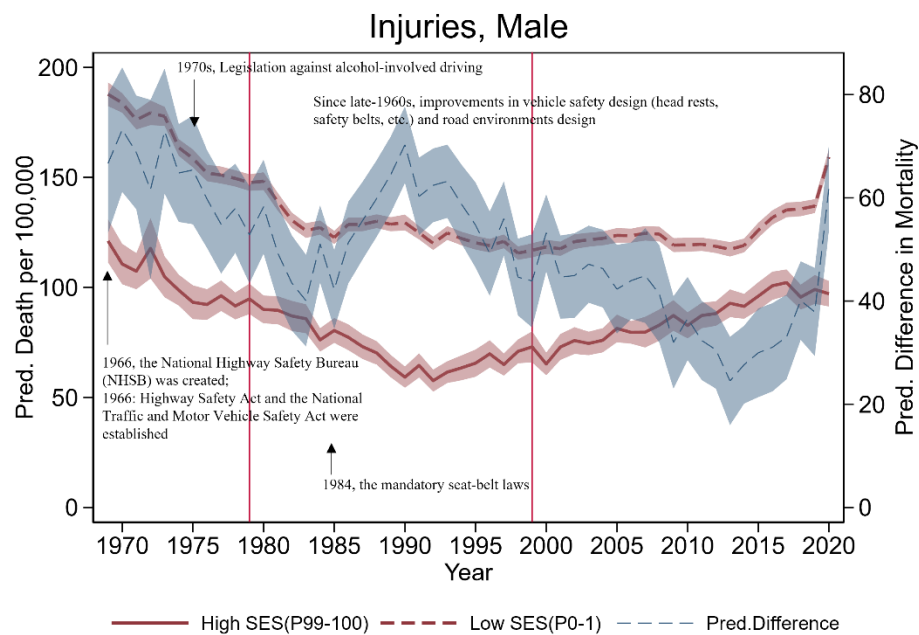


Figure 5.6 The predicted mortality by SES percentile (left Y axis) and the predicted difference in mortality (right Y axis) for injuries (shaded areas represent 95% confidence intervals).

a. Male



b. Female

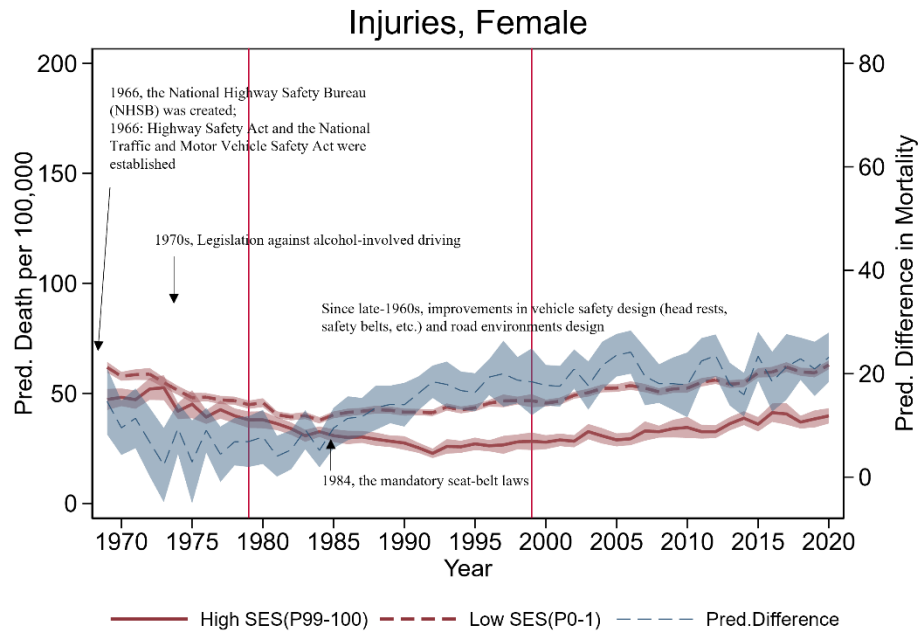
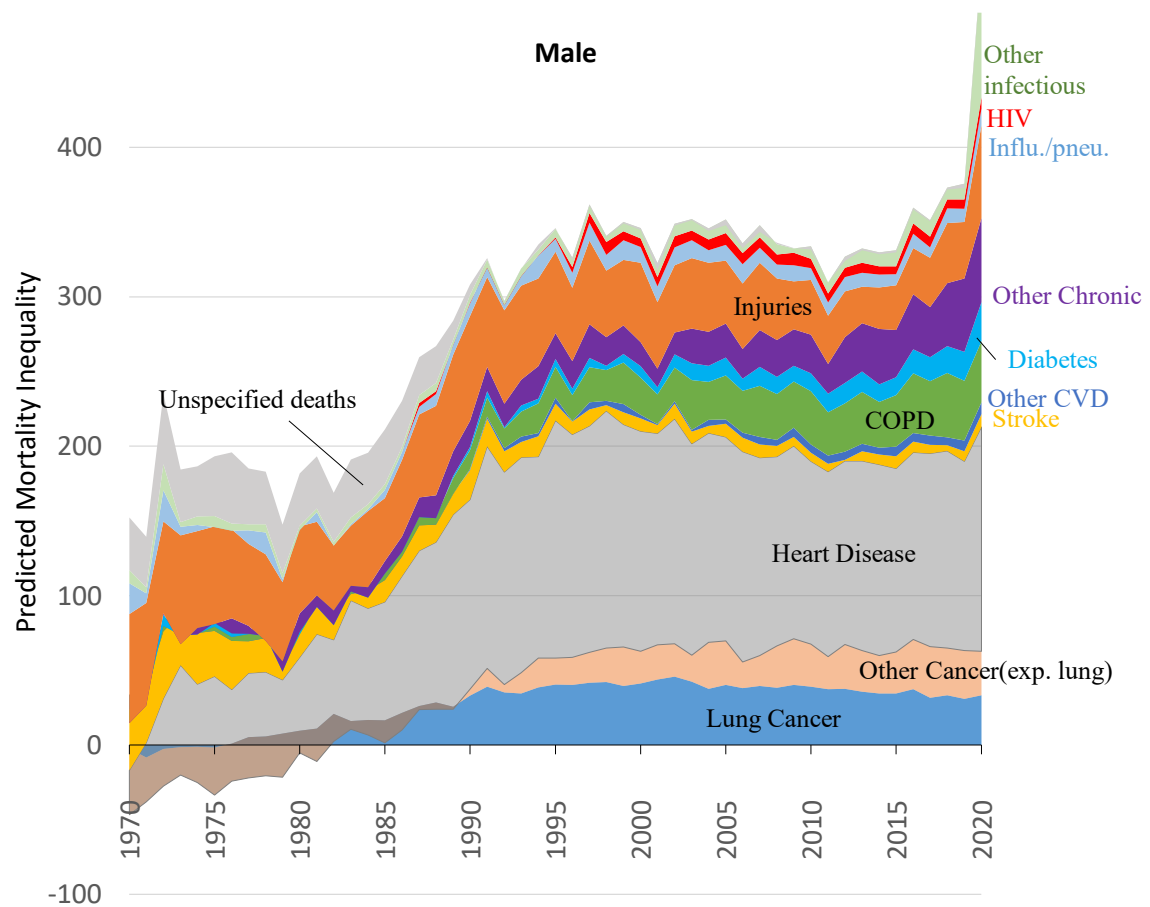
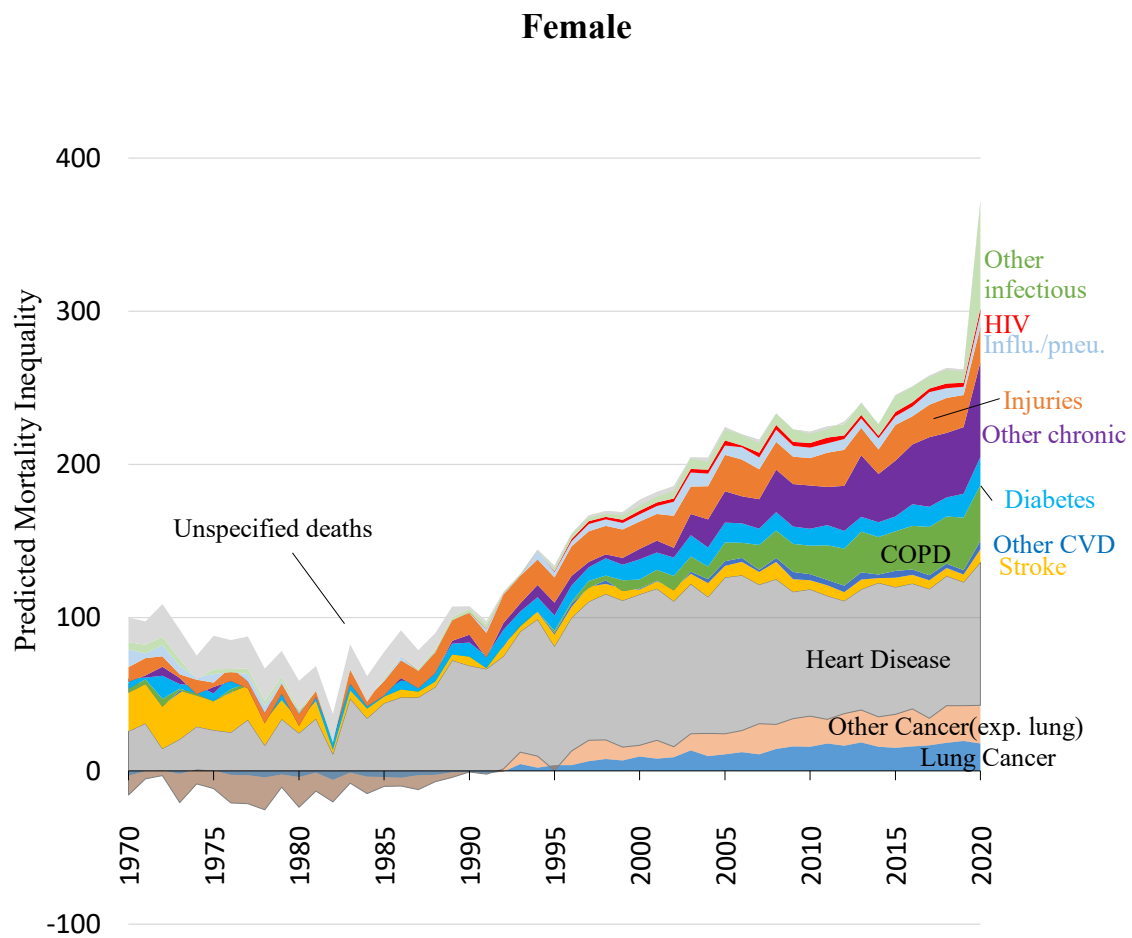


Figure 5.7 The area graph of mortality across cause of death, males.



Notes: the mortality inequality across cause of death summed together equals the all-cause mortality inequality.

Figure 5.8 The patterns of mortality inequality across cause of death, females.



Notes: the mortality inequality by cause of death summed together equals the all-cause mortality inequality.

Figure 5.9 The patterns of inequality in mortality rates, by cause of death and age, males.

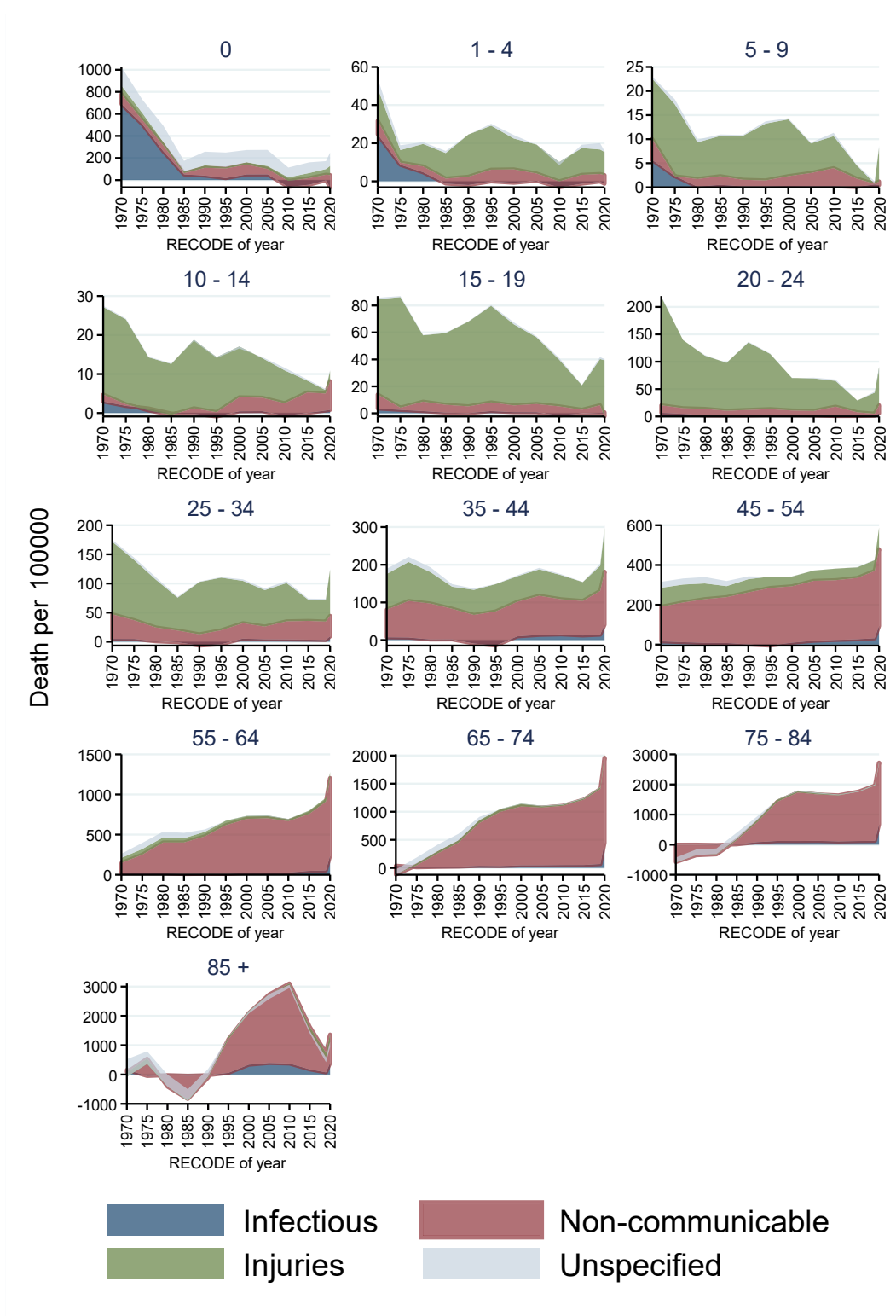
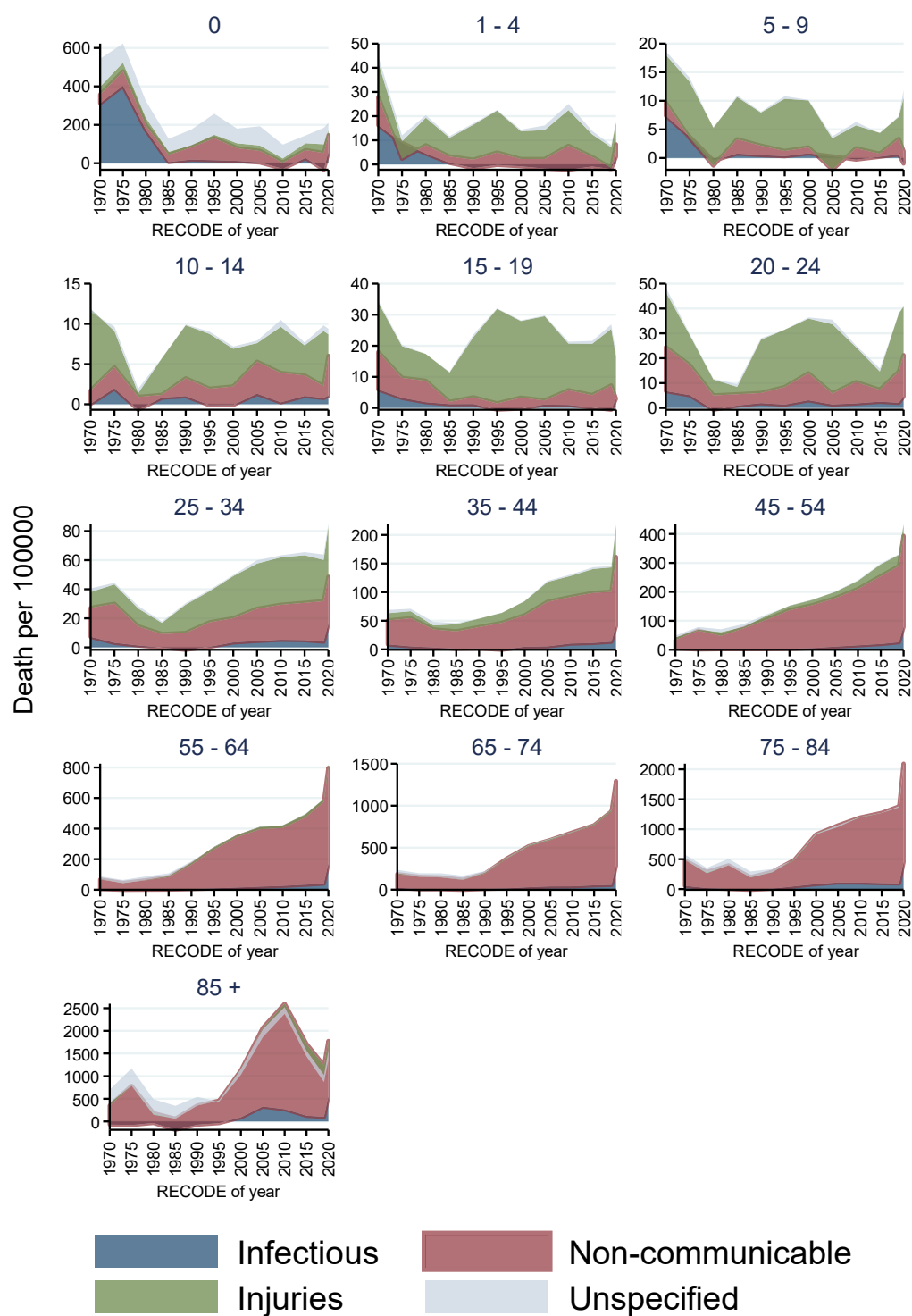


Figure 5.10 The patterns of inequality in mortality rates, by cause of death and age, females.



Appendix Tables and Figures

Table S5.1 The ICD-8, ICD-9 And ICD-10 codes for the global burden of disease study classification Group I , II , And III.

Group of diseases	ICD-8 A-list	ICD-9 code	ICD-10 code
I. Communicable, maternal, perinatal, and nutritional conditions	A1-A44, A65, A67, A72, A78, A89-A92, A112-A118, A131, A135	001–139, 243, 260–269, 279.5, 280–281, 285.9, 320–323, 381–382, 460–465, 466, 480–487, 614–616, 630–676, 760–779	A00–B99, G00–G04, N70–N73, J00–J06, J10–J18, J20–J22, H65–H66, O00–O99, P00–P96, E00–E02, E40–E46, E50, D50–D53, D64.9, E51–64
II. Noncommunicable diseases	A45-A64, A88, A68-A71, A730A77, A79-A88, A93-A111, A119-A130	140–242, 244–259, 270–279 (minus 279.5), 282–285 (minus 285.9), 286–319, 324–380, 383–459, 470–478, 490–611, 617–629, 680–759	C00–C97, D00–D48, D55–D64 (minus D 64.9) D65–D89, E03–E07, E10–E16, E20–E34, E65–E88, F01–F99, G06–G98, H00–H61, H68–H93, I00–I99, J30–J98, K00–K92, N00–N64, N75–N98, L00–L98, M00–M99, Q00–Q99
III. Injuries	AE138-AE150	E800–999	V01–Y89

Source: Global Burden of Disease Study. The ICD-8 codes come from (Mathers et al. 2006); ICD-9 and ICD-10 comes from (Lopez et al. 2006)

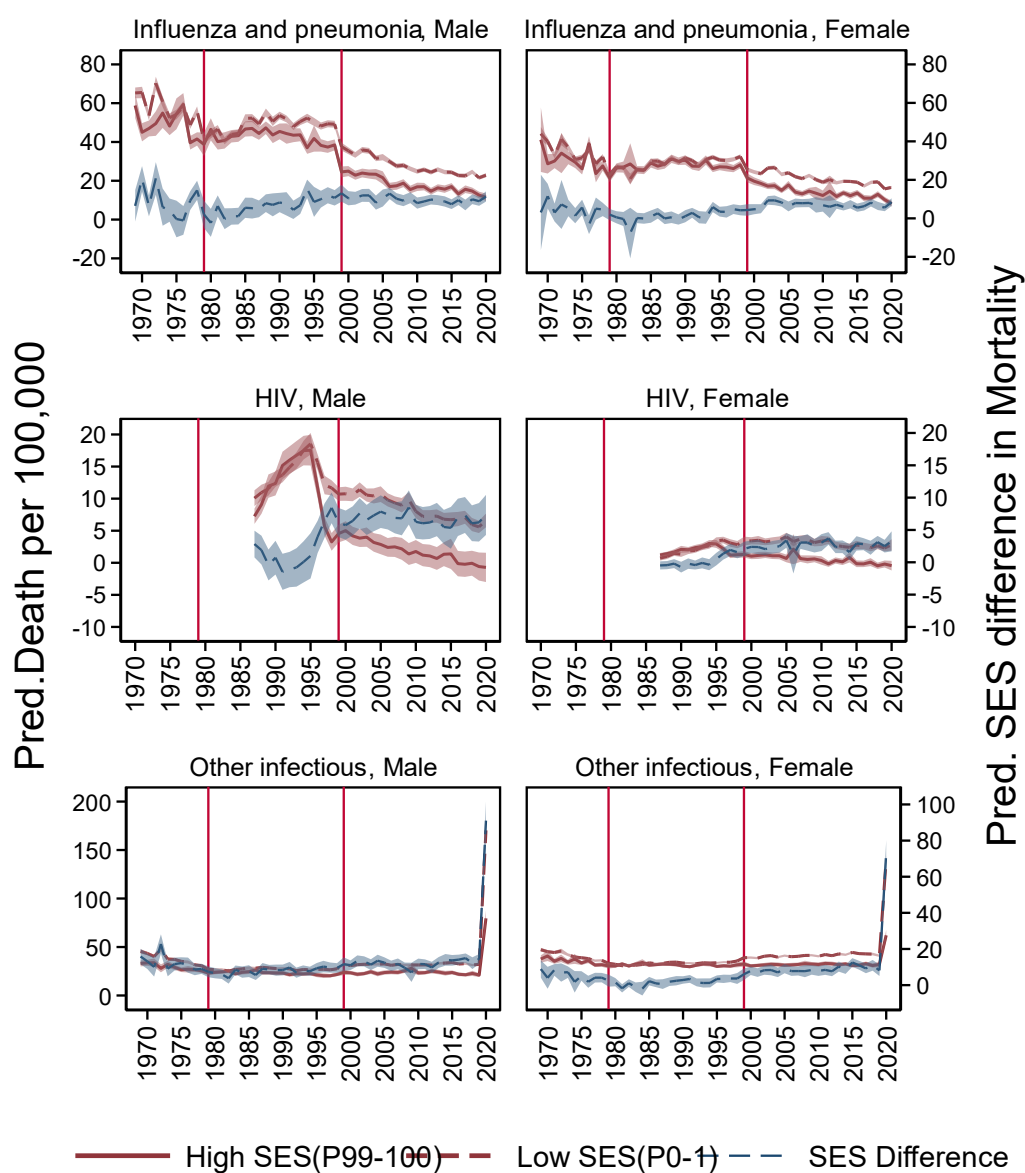
Table S5.2 The Cause-of-death Codes, by ICD-8, ICD-9, and ICD-10.

Group of diseases	ICD-8 A-list	ICD-9 code	ICD-10 code
(1) Influenza and pneumonia	470-474, 480-486	480-487	J10-J18
(2) maternal and prenatal diseases	630-678, 764-779	630-676, 760-779	A34, O00-O95, O98-O99, P00-P96
(3) HIV	...	042-044	B20-B24
(4) other infectious diseases	All other Group I diseases		
(5) heart disease	390, 398, 402, 404, 410-429	390-398, 402, 404, 410-429	I00-I09, I11, I13, I20-I51
(6) stroke	430-438	430-434, 436-438	I60-I69
(7) other CVD	390-458 (except heart disease and stroke)	390-459 (except heart disease and stroke)	I00-I99 (except heart disease and stroke)
(8) lung cancer	162	162	C33-C34
(9) other cancer (except lung cancer)	140-209(except)	140-208(except lung)	C00-C97 (except lung)
(10) chronic lower respiratory diseases (CLRD)	490-493, 519.3	490-494, 496	J40-J47
(11) diabetes	250	250	E10-E14
(12) chronic liver disease and cirrhosis	571	571	K70, K73-K74
(13) Alzheimer's disease	...	331.0	G30
(14) Other chronic diseases.	All other Group II diseases		
(15) unintentional injuries	E800-E929, E940/E946	E800-E869, E880-E929	V01-X59, Y85-Y86
(16) suicide	E940-E959	E940-E959	X60-X84, Y87.0
(17) violence	E960-E969	E960-E969	X85-Y09.9 Y87.1
(18) other injuries	All other Group III diseases		

Source: Historical Leading cause of death 1900-1998(Centers for Disease Control and Prevention, 2007);United States, 2019, p.25; Global Burden of Disease Study (Lopez et al. 2006).

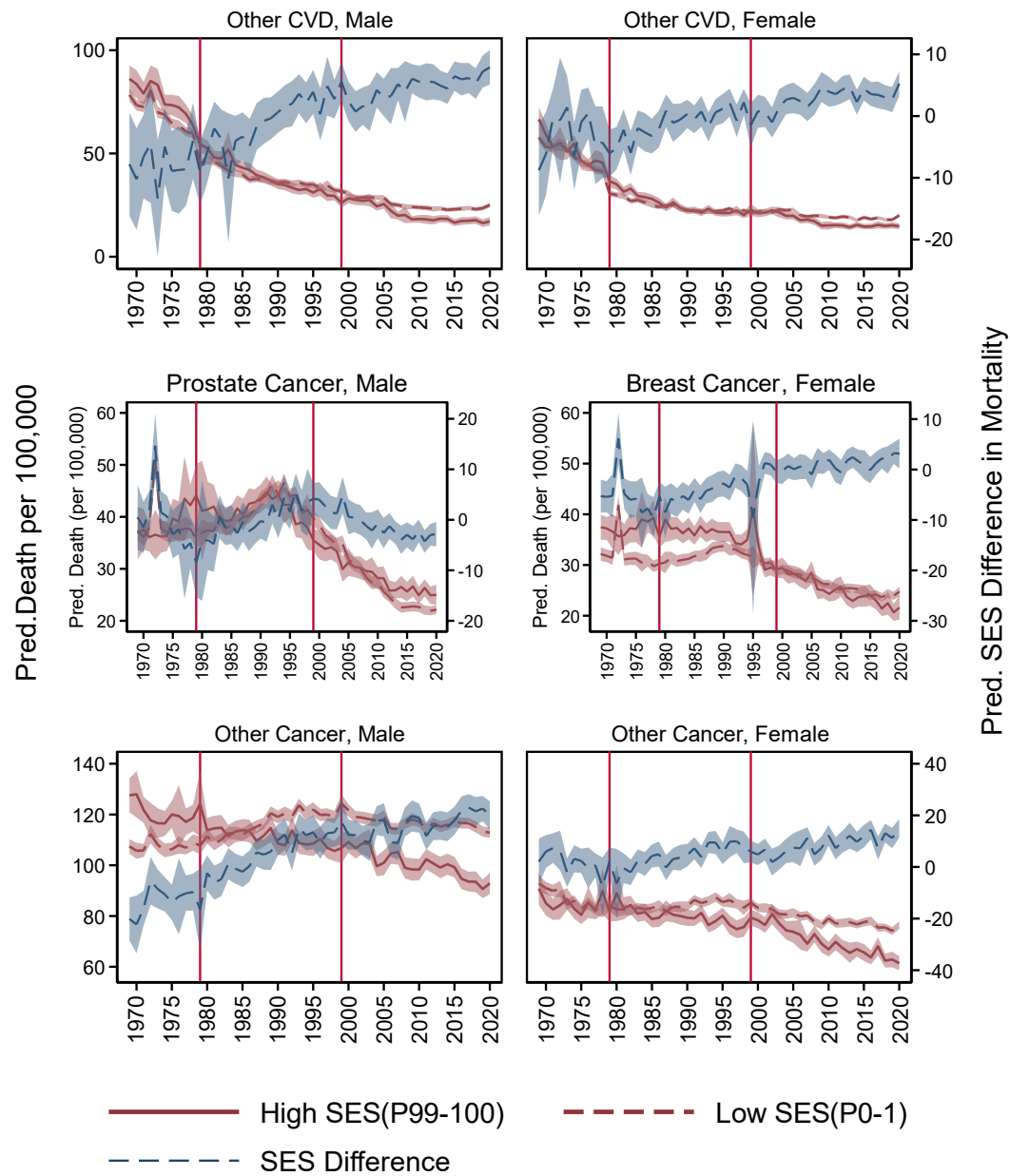
Notes: It is to be noted that the changes in ICD coding from ICD 8 (1969-1978), ICD-9 (1979-1988), to ICD-10 (1989-2020) could affect the comparability in mortality by cause-of-death. The mortality rates for some cause-of-death would be affected by different version of ICD codes. For example, the comparability ratio of heart disease (mortality rate under ICD 10 divided by mortality rate under ICD 9) is about 0.9858 (Anderson et al., 2001; Klebba & Scott, 1980).

Figure S5.1 The predicted mortality by the highest SES percentile (P99-100) and lowest SES percentile (P0-1), and predicted inequalities for cause-specific infectious diseases.



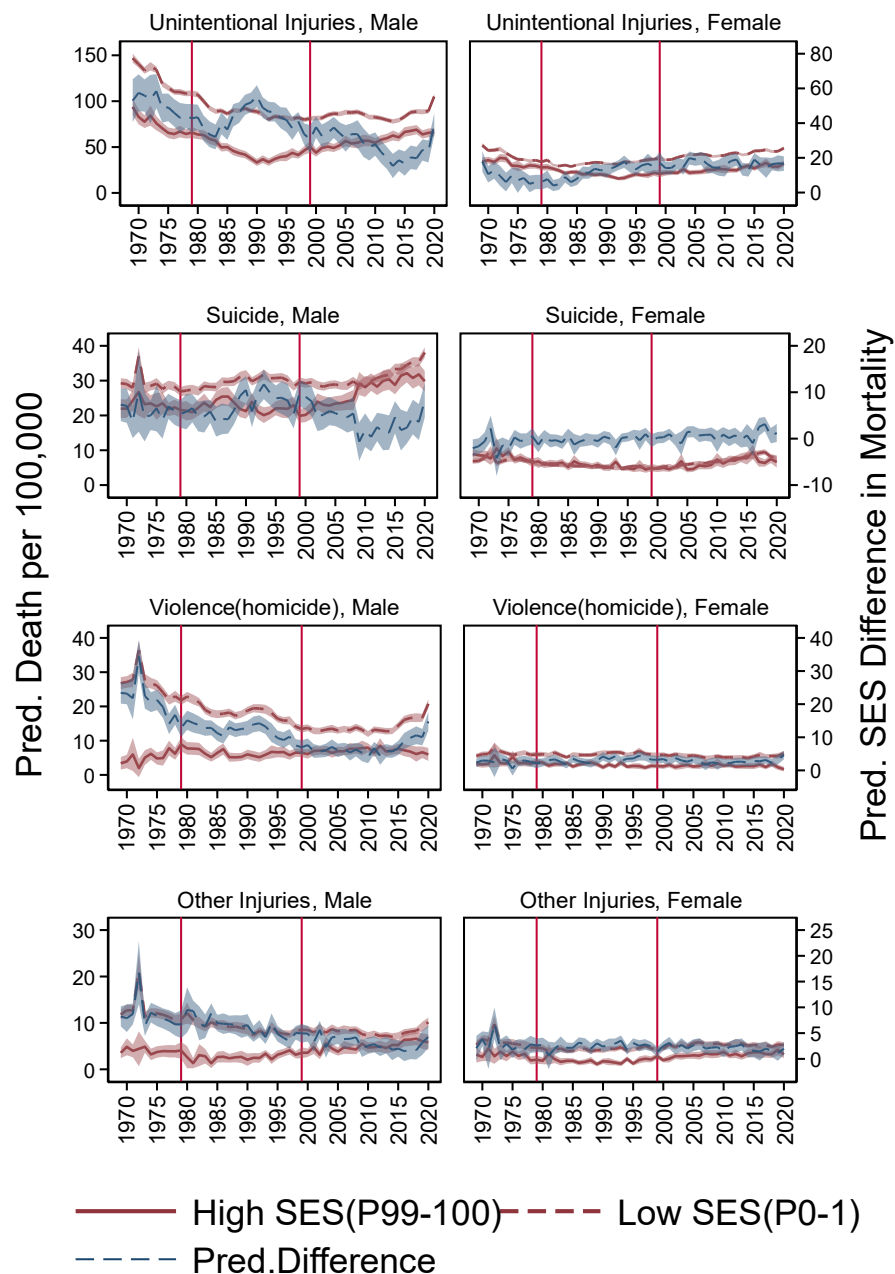
Note: Shaded areas represent 95% confidence intervals.

Figure S5.2 The predicted mortality by the highest SES percentile (P99-100) and lowest SES percentile (P0-1), and predicted inequalities for selected cardiovascular and cancer.



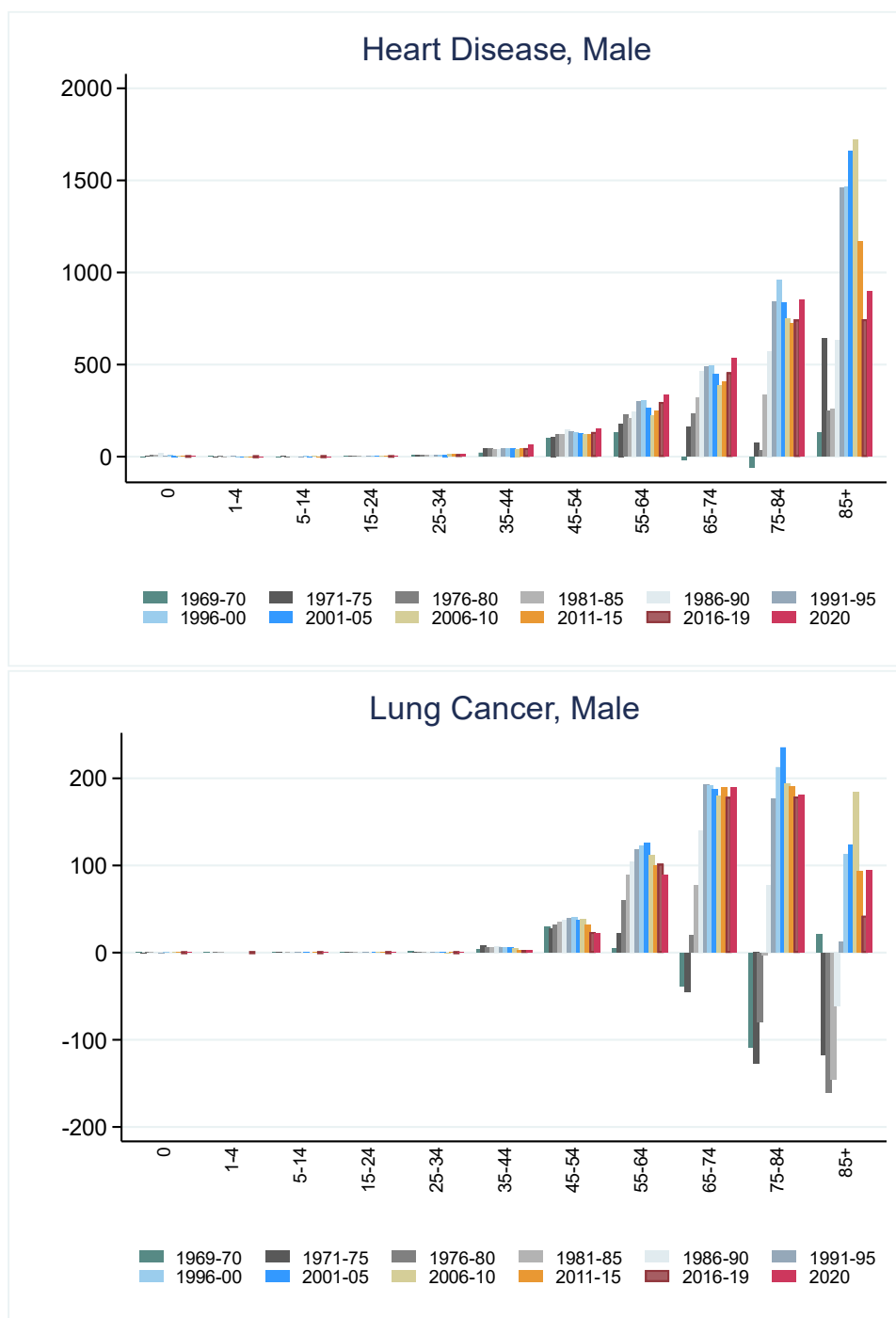
Note: Other CVD includes CVD except heart diseases and stroke.
Other cancer includes cancer except lung/colon/prostate/breast cancer

Figure S5.3 The predicted mortality inequalities between the highest SES percentile (P99-100) and lowest SES percentile (P0-1) from cause-specific injuries



Note: Shaded areas represent 95% confidence intervals.

Figure S5.4 The predicted mortality inequalities from heart diseases and lung cancer, by age groups, male (with 95% confidence interval).



Chapter Six: Conclusion and Discussion

The persistent and widening disparities in mortality have emerged as an urgent public health issue in the United States. To effectively tackle this challenge and reduce these inequalities, it is essential to gain a deeper understanding of the complex connections between socioeconomic status (SES) and health, specifically how these disparities have evolved over time. However, current research has encountered two perplexing puzzles that remain unsolved: 1) the growing inequalities by SES despite declining overall mortality levels and expansion of social welfare policies, and 2) the declining disparities at younger ages but growing inequality at older ages. These challenges underscore the limitations of existing theories in explaining the complex dynamics of mortality inequalities.

This dissertation aims to bridge this critical gap by presenting an alternative theoretical framework. While previous studies have mainly focused on social and economic inequalities as the primary drivers of increasing mortality disparities, this research proposes that mortality inequalities can evolve independently of socioeconomic factors. The key to understanding this phenomenon lies in the shifting disease patterns towards chronic diseases and advancements in health-beneficial innovations. By analyzing county-level US mortality rates from 1968 to 2020, categorized by age, sex, and cause of death, this paper reveals a notable transition of mortality inequalities from infectious diseases to chronic diseases. Here are the key findings of this study:

Firstly, mortality inequality has been consistently increasing since 1969, primarily due to a faster decline in mortality rates for the higher socioeconomic status (SES) group compared to

the lower SES group. The inequality escalated notably during specific periods: between 1980 and 2000 for males and between 1985 and 2005 for females (with a 5-year delay compared to males). Additionally, it continued to rise for both males and females from 2010 to 2020.

Secondly, the trends in mortality inequality vary significantly across age groups. Mortality inequalities among children, adolescents, and young adults (ages 0-24) decreased from the 1970s to the 1980s and have remained relatively low and stable since then. Conversely, the increase in mortality inequality since the mid-1980s mainly affected middle-aged adults (ages 45-64) and older adults (ages 65-84). Moreover, even among middle-aged and older adults, the increase is much faster among older age groups than among the middle aged. This diverging increase in mortality inequality across age groups resulted in a concentration of mortality inequalities in later life. These findings provide support for the theoretical framework presented in Chapter three, which includes Hypothesis 1 concerning the overall trends of all-cause mortality inequality and Hypotheses 3 and 4 regarding the diverging trends across age groups.

Thirdly, when examining mortality inequality by cause of death, particularly in relation to infectious diseases versus chronic diseases, distinct trends emerge. Initially, the mortality inequality in infectious diseases declined in the early 1970s, with a minor increase in the 1990s due to the emergence of HIV and drug-resistant strains of some previously known infectious diseases. On the other hand, the mortality inequality in chronic diseases remained at a low level during the 1970s but saw a significant increase during the 1980s and 1990s. This rise can be mainly attributed to growing inequalities in diseases such as cardiovascular diseases, lung cancer(especially for males). During the 1990s and early 2000s, the increase in heart diseases

mortality inequality slowed down (especially for males), leading to a slowdown in increase of overall mortality inequality. The inequalities in cancer (excluding male lung cancer), COPD, and diabetes continued to increase during 1980-2020. Since 2010, unintentional injuries, suicide, violence-related deaths, and certain chronic diseases, such as chronic liver diseases have also played a role in shaping the widening mortality disparities.

Lastly, the findings also reveal notable variations in mortality inequalities from infectious diseases, chronic diseases, and injuries across different age groups and genders. Mortality inequality from chronic diseases is more pronounced in middle-aged and older adults, with a larger increase observed in older age groups compared to younger ones. The age distribution of mortality inequality for each major chronic disease shifts progressively towards older ages. For gender differences: The increase in mortality inequality of non-communicable disease (especially in heart diseases, lung cancer, and COPD) started later for males than females. Injuries contribute to the largest proportion of mortality inequality among children and young adults. But inequalities in injuries have increased in importance over time for women while they've decreased for men. This suggests greater risks over time for low SES women (especially among women aged 25-54) and raises concerns about women's health, particularly among those with fewer SES resources. The reason for these gender differences is not well understood; one possibility is a worsening of women's risk behaviors in recent decades. For example, women have shown lower cessation rates in smoking in recent decades (Osler et al., 1999). Furthermore, as women increasingly take on roles beyond the traditional sphere of home and become integrated into broader employment and social domains, the preservation of traditional health

roles weakens—for example, women have greater independence and less attachment to marriage, which can be protective for women's health. Simultaneously, women might adopt risky behaviors traditionally associated with masculinity or be exposed to health risks typically prevalent in male-dominated environments (Annandale, 2009; King et al., 2018; Pampel, 2001).

The trends in mortality inequality related to major diseases such as heart diseases and cancer align with previous studies (Phelan & Link, 2005; G. Singh, 2014; G. K. Singh, 2003, 2020; G. K. Singh et al., 2002; G. K. Singh & Siahpush, 2002). However, this study is the first to organize and compare mortality inequality across different causes of death based on the epidemiological transition. These findings provide support for the theoretical framework and hypotheses about diverging trends across causes of death, particularly the decline in mortality inequality from infectious diseases and increase in inequality from chronic diseases, as well as the age group patterns in mortality inequality by cause of death. The emergence of huge mortality inequalities from chronic diseases, which mainly concentrated on middle-aged or older population, sheds light on the two "public health puzzles": 1) the growing inequalities by SES despite declining overall mortality levels and expansion of social welfare policies, and 2) the declining disparities at younger ages but growing inequality at older ages.

This study contributes to the existing literature with a new theoretical perspective to understand the developments of mortality inequalities over time. The core innovation of this study, in a nutshell, is that the extent and direction of mortality inequality are collectively affected by three separate but interlocked factors: disease patterns, prevention/treatment innovations, and social and economic inequalities. By examining these factors together, rather

than solely focusing on pre-existing social and economic inequality, we gain valuable insights into understanding mortality inequality trends in recent decades and can also explore potential scenarios for the future. To be specific:

The first insight it offers us is that the characteristics of diseases dominating during specific historical periods form a crucial context for understanding developments in mortality inequality. Those diseases with high disease burden—do not automatically result in health inequality (e.g., heart disease had high death rates but showed minor disparities in the 1960s). Yet they often motivate the development of innovative medical solutions in treatment and prevention which typically are first adopted by those with more resources, thereby exacerbating health inequalities. The findings of this study suggest that this process has profoundly changed our past, and it will likely shape our future as well.

Across the centuries, remarkable progress in medicine, public health, living standards and nutrition have significantly diminished deaths caused by infectious diseases. As chronic diseases became the dominating killers, medical innovations in prevention, detection and treatment have helped to delay the onset, and reduce the health impact of many chronic diseases, raising average life expectancy and improving population health. However, at the same time we see these improvements, we also see how health inequalities are reinforced by this process. As we look ahead, chronic diseases appear to be the predominant future burden. Further, mortality inequality is steadily shifting towards older age groups. As we look ahead, the influence of older adults in overall mortality inequality will become of increasing significance. Meanwhile, the life course experiences (the accumulation of risk and exposure during the whole life span) will also become

more and more important. At the same time, the reemergence of infectious diseases has brought about significant uncertainty for the future—this might be exacerbated even more by climate change(Watts et al., 2018).

The second insight it offers is the interplay of medical innovation and social-political circumstances. We don't have to stop progress in medical innovations to address inequalities—this is not favored by most people. Medical knowledge and technology affect population health as well as health inequalities through “a thick distribution of social, political and economic circumstances”(Link 2008). As health-beneficial innovations make humans have more capacity in prevention/treatment of diseases, it is those human hands and social policies that determine whether and how the capacity “is deployed, at what rate the uptake occurs in different places and at different times, and who benefits most and least” (Link 2008).

This highlights the important role of “social shaping” (Link, 2008) in determining future population health and health inequalities. The social-political factors will become more—not less—important for population health (Link, 2008). If we witness a prolonged period where the health of the high socioeconomic status (SES) population consistently improves while the health of the low SES population remains stagnant or even deteriorates (the stagnation or slowdown of transition from stage 2 to stage 3 of Clouston's ideal type, see chapter 3), it serves as a clear indication of social failure in population health (the stagnation of heart disease mortality inequality might be an example).

Several limitations for this study should be mentioned. The first limitation of this study is data constraint. The study couldn't fully capture the significant decline of infectious diseases in

the early years due to the lack of data prior to the late 1960s. Future research with US historical data from the first half of the 20th century or other counties where infectious diseases persisted in the second half of the 20th century could help address this limitation. Additionally, using county-level data carries the risk of the ecological fallacy (Robinson, 2009). Although recent empirical studies suggest that trends in county-level socioeconomic status (SES) inequalities in mortality align with trends at the individual level (Lokar et al., 2019), it is essential to conduct further research that examines individual-level inequalities in mortality, morbidity, and other health conditions.

Secondly, this study reveals that temporal trends in cause-specific mortality inequality align with the anticipated expectations regarding the influence of medical innovations on inequality trends. However, these analyses remain descriptive or suggestive in nature, lacking statistical tests to establish causal effects of medical innovations on inequality trends.

Further, the patterns of mortality inequalities in certain diseases are not well understood. Take cancer, for example; during the early 1970s, mortality rates in some cancers were higher in high socioeconomic status (SES) counties, leading to negative SES inequality. This discrepancy could be attributed to changes in who gets diagnosed and how accurately. Because of their better access to high quality health care, high SES individuals might have had more accurate death diagnoses, resulting in higher mortality rates in certain disease groups like cancer. On the other hand, low SES individuals may have faced higher "competing risks" of death, which might have claimed lives at an earlier stage (Phelan and Link 2005). Unfortunately, the current study couldn't account for these explanations in its empirical analyses, emphasizing the need for future

individual-level research. This dissertation also did not consider other dimensions of social inequality like race, gender, sexual orientation, etc.

Furthermore, the changes in mortality inequality for some diseases do not completely align with the fundamental cause theory. For instance, mortality rates from prostate and breast cancer have declined since the 1990s, but the decline occurs in both high and low SES groups, resulting in stable and low-level inequality. On the other hand, female unintentional injuries have increased since 1990-2010, but the rise is faster in the high SES group, leading to a decline in mortality inequality. Deaths from suicide, violence, unintentional injuries, and chronic liver diseases (associated with alcohol use) increases in recent years, but the increase seem to be more closely related to changes in social order and associated beliefs/behaviors (e.g., deaths of despair), rather than changes in health-beneficial knowledge. The mortality inequalities among the oldest old group (85+) also present unique trends and need more future research. These insights highlight the complexities involved in understanding mortality inequalities.

This study also emphasizes several critical areas that warrant further investigation in the future. The first is trends of SES inequality in health during different stages of epidemiological transition (e.g., when infectious diseases are still the main burden of disease or when both infectious and degenerative diseases are dominant). For example, future research could examine health inequalities during different historical period in high income countries, or the cross-country comparison between developing and developed countries, or the gender-race heterogeneity in health inequality trends and its association with the changes in disease patterns.

The second one is the extension of life course perspective. The framework in this paper could be used to enrich the research of historical changes (or trends over time) under life course perspectives. For example, the cumulative advantage/disadvantage of health disparities over the life course may differ when the disease patterns in a population change over time. It is possible that the cumulative process is much stronger for man-made, degenerative diseases than infectious diseases. If this is the case, the cumulative advantage/disadvantage process would be more salient in a population with chronic conditions as the main disease burden than with infectious diseases as main burden.

Further, life course theory predicts that as early-life nutrition and health improve universally, later-life health will also improve, and health inequalities will decline. But empirical research gets mixed results (e.g., the health of baby boomers in their later life are not always improved, and health inequalities have been widening across cohorts). The changes in disease risks across cohorts may provide an answer. As early-life nutrition and health improve universally, more people survive to their later life and face an elevated risk of degenerative diseases. The shift in disease patterns itself could alter the content, magnitude, and shape of life course patterns.

The next one is the interplay of disease patterns, medical knowledge and technology, and social-political circumstances in shaping health inequalities. Link & Phelan proposed that SES benefits health through “flexible resources” which include “a wide range of broadly serviceable resources including money, knowledge, prestige, power, and beneficial social connections”(Link & Phelan, 1995). However, previous studies largely operationalize the “flexible resources” as

individual level characteristics. This individual level perspective has been more and more criticized in recent years. Differences in state level policy within the US, or the comparison of policy context between US and Canada/Europe shows that welfare-state policy helps explain the troubling trends and inequalities in US life expectancy (Avendano & Kawachi, 2014; Beckfield & Bambra, 2016; Montez et al., 2019, 2020; Van Dyke et al., 2018). The most recent development in FCT focuses power relations as the root of health inequality (Dickie E et al., 2015; Freese & Lutfey, 2011; Givens et al., 2018; McCartney et al., 2021; Whitehead et al., 2016). Future studies could do more to include disease patterns and implementation of medical technologies into the analyses of individual SES as well as other dimensions of power relation—the race, gender, religion under various kinds of “thick distribution of social, political, and economic circumstances” (Link 2008).

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