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Age, SES and Health: Old Topic, New Perspective

(A Longitudinal Perspective about the Relationship between SES and Health)

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Age, SES and Health: Old Topic, New Perspective

(A Longitudinal Perspective about the Relationship between SES and Health)

by

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Dedication

This dissertation is dedicated to my loving wife, Lixin Wang, and my son, James Y. Wu for their unconditional support during my graduate study.

I also dedicate this dissertation to my parents, Qingyuan Wu and Xiuyun Bai, my parents-in-law, Zhong Wang and Xihua Zhang, and my brothers and sister for their endless support and encouragement.

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Age, SES and Health: Old Topic, New Perspective

(A Longitudinal Perspective about the Relationship between SES and Health)

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Socioeconomic disparities in health are well documented in the United States in that the higher socioeconomic status (SES) a person is the lower level of incidence and the prevalence of health problem, disease and death will be. Researchers have argued that SES disparities in health are found in different races, across countries, and throughout life.

Furthermore, some researchers have argued that high SES persons enjoy their active life expectancy until quite late in life, while low SES persons start to suffer morbidity and functional limitation through middle and early old age. As a result, socioeconomic disparity in health becomes increasingly differential at middle age, while it diminishes until later old age due to physical fragility of human being and declining effect of SES.

This study extends previous research by applying the Multi Level Model for Change (MLMFC) and Multi Cohorts Accelerate Longitudinal Design (MCALD) to explore SES disparities in health over the life course. Functional limitation and chronic diseases are used as measurements of health status over time. This study introduces a new way to measure health status over time by combining number and duration of chronic diseases. The results suggest that using a combination of number and duration of chronic diseases as measurement of health over time is more optimal than only using the number of chronic diseases.

Both time variant and invariant independent variables are used to explore health trajectory. Findings from this study indicate that people from different SES groups have different health trajectories over the life course. Low SES people tend to age faster than their high SES counterparts. The process of aging in the United States is largely segregated according to individuals' SES.

Financial stress and health risk behaviors are two major mediators of SES disparities in functional limitation. However, SES (measured by income) is consistently a significant predictor of chronic diseases after controlling financial stress and health risk behaviors. Findings of this study suggest that the mechanisms through which SES impacts functional limitation may be different from its impact on chronic diseases.

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CHAPTER 1: INTRODUCTION

Socioeconomic disparities in health are well documented in the United States in that the higher SES a person is, the lower level of incidence of death, disease and prevalence of health problems will be (Adler and Ostrove 1999; Berkman and Breslow 1983; Gortmaker and Wise 1997; Haan et al. 1989; Houses et al. 1994, 2005; Kitagawa and Hause 1973; Marmot et al. 1997; Mulatu and Schooler 2002; Williams 1990). Researchers have argued that SES disparities in health are found in different races (Haan, Kaplan and Syme 1989), across countries (Beydoun and Popkin 2005; Mulatu and Schooler 2002), and throughout life (Haan, Kaplan and Syme 1989; House et al. 1994).

Furthermore, some researchers have argued that high SES persons enjoy their active life expectancy until quite late in life, while low SES persons start to suffer morbidity and functional limitation through middle and early old age (Haan et al. 1989; House et al. 1994; Siegrist and Marmot 2006). As a result, socioeconomic disparity in health becomes increasingly differential at middle age, while it diminishes until later old age due to physical fragility of human beings and declining effect of SES (House et al. 1994; Siegrist and Marmot 2006).

This study explores socioeconomic disparities in health over the life course by utilizing the longitudinal data set of Americans' Changing Lives (ACL): Wave I, II, and III, 1986, 1989, 1991. The life course perspective is adopted to explore health trajectories across SES groups, which "emphasizes that individuals continue to develop and change over time" (Umberson et al. 2005:1334). Functional limitation and chronic diseases, as

the major measurements of health, are used in this study. Mechanisms, through which SES is linked to health, are investigated. The following topics are covered:

1. What is the association between SES and health at the beginning of the ACL study in 1986? Do major health risk behaviors mediate the relationship between SES and health? Answers to these questions aim to provide a snapshot of SES and health, and potential explanations for the association of SES and health.
2. How is SES related to the rate of change in health over the 8-year study of ACL? Do major health risk behaviors mediate the rate of change across SES groups? This part of the study aims to indicate that people from different SES groups may have different rates of change in their health during the 8-year interval in ACL, and to identify the factors mediating SES disparities in the rate of change in health.
3. Beyond the scope of the 8-year interval in ACL, what is the association between SES and health over the life course? Do major health risk behaviors mediate SES disparities in health trajectories over the life course? This part of study aims to indicate that people from different SES may have different health trajectories over the life course, and SES disparities in health at different life stages may actually reflect such different health trajectories.

The remaining part of this section provides a brief discussion of theories/hypotheses which explain SES disparities in health, indicators of SES, methodology utilized in this study, and an overview of the study.

Theories or hypotheses about SES disparities in health

The socioeconomic disparities in health have generated considerable attention both in social science and biological science. Research has been conducted to better understand the mechanisms through which SES is associated with health. In social science, especially in sociology, it has been continuingly debated if the link between SES and health is a social problem or personal issue. Health risk behavior theory argues that poor health among lower SES groups is largely a personal issue. SES disparities in health largely reflect the choices people make in their daily lives. Lower SES people are more likely to adopt health risk behaviors, which erode their health (Macintyre 1997; Siegrist and Marmot 2006). The increased prevalence of major health-risk behaviors among people of lower socioeconomic positions largely accounts for SES disparities in health (Macintyre 1997; Siegrist and Marmot 2006). However, social causation theory argues that SES disparities in health are largely the product of social forces and not, or not solely, the product of different physical processes among different SES groups. Furthermore, it states that SES disparity in health is a social problem, which results in the unequal social stratification systems in the United States (Hayward et al. 2000; Link and Phelan 1995; Marmot and Wilkinson 1999). It argues that worse health among poor people is due to the fact that they are stratified into disadvantage status such that they lack necessary resources to control their daily lives and health (Hayward et al 2000).

Researchers have also drawn attention from other perspectives about SES and health. For example, selection hypothesis suggests that people's health actually impacts their socioeconomic status (Adler and Ostrove 1999; Bartley et al. 1999; Marmot et al. 1997;

Ross and Mirowsky 1995; Stern 1983). Unhealthy people are more likely to be excluded from employment (Ross and Mirowsky 1995); even employed, they are more likely to lose their jobs and harder to be re-employed due to their health (Bartley et al. 1999). They also have higher risk of unemployment and job insecurity (Marmot and Wilkinson 1999). The functional limitation or chronic disease of unhealthy people may undermine their performance at work, which in turn reduces the opportunities of staying or becoming employed full-time (Ross and Mirowsky 1995). Furthermore, even if they are employed, they are more likely to be in a lower position with lower pay and less likely to have a job with a high level of autonomy or control.

However, findings from previous studies are less in favor of selection hypothesis. Wilkinson (1986) argues that selection into SES does occur; however, the effect of selection is too marginal to exert any major impact on social disparities in health. Thus, selection into SES is of minor importance (Fox et al. 1985; Marmot et al. 1987; Marmot et al. 1997; Marmot and Wilkinson 1999; Wilkinson 1986).

Medical care access explanation suggests that SES disparities in health are largely due to the inequalities in the accessibility, utilization or quality of medical care (Mackenbach et al. 1989) as we know that medical treatment could postpone the onset of some chronic diseases and reduce some diseases (Preston and Taubman 1994). However, poor people have limited access to expensive medical care; they can't afford expensive medical treatment; thus they have lower chances of survive than higher SES people. Nevertheless, researchers have argued that medical care plays only a partial role in SES disparities in health. The disparities in the incidence of diseases and in mortality from

diseases, which are not amenable to medical intervention, are not likely due to the differences in medical care (Adler and Ostrove 1999; Mackenbach et al. 1989).

Although there are some other explanations for SES disparities in health, such as different exposure theory and different vulnerability hypothesis, health risk behavior theory and social causation theory have been receiving major attention in sociology. This study will follow health risk behavior theory and social causation theory to explore the effect of SES and health risk behaviors on health over the life course. By the same token, I am limiting the scope of literature review within health risk behavior theory and social causation theory.

Furthermore, this study will also examine social selection theory. If social selection theory is correct in its argument that people's SES is largely due to his/her health, then people whose health is worse at the baseline of a study will have lower income at later study points when their baseline income is controlled. Chapter 5 aims to examine social selection theory.

Previous studies have employed various indicators of health to explore SES disparities in health, such as mortality, morbidity, functional limitation, depression etc. Functional limitation and chronic diseases have been used frequently as measurements of health in previous research (e.g. Beydoun and Popkin. 2005; Hayward et al. 2000; House et al. 1994, 2005; Lantz et al. 2001; Link and Phelan 1995; Lorig et al. 2000). In order to compare the results from this study to previous research, I mainly focus on functional limitation and chronic diseases as measurements of health to explore the relationship between SES and health over the life course.

Indicators of SES

The traditional indicators of SES at the individual level have been income, education, and occupation, which reflect different aspects of social stratification (Siegrist and Marmot 2006; Adler and Ostrove 1999). They are often used interchangeably in research, since "similar associations with health have been found no matter which SES indicator is used (Adler and Ostrove 1999:10)." However, it has been suggested that each of SES indicators actually reflects somewhat different individual and societal forces associated with health and disease (Winkleby et al. 1992). For example, income reflects spending/buying power, clothing, housing, nutrition, and medical services; occupation measures prestige, responsibility, physical activity, and working environment; and education indicates skills requisite for acquiring social, psychological, and economic resources (Antonovsky 1967; Siegrist and Marmot 2006; Susser et al. 1985). Therefore, using only one indicator of SES may yield misleading results or provide less information than using multiple measures (Winkleby et al. 1992). Although occupational characteristics cover most relevant aspects of socio-economic inequalities, their application is limited to employed population (Krieger et al. 1997). Thus, in this study, I use income and education as measurements of individual SES, and include them together into statistical models to exclude the confounding effect between them.

Methodology utilized in this study

Findings about SES and health over the life course have come from both cross-sectional and longitudinal data analyses. In cross-sectional data analyses, an interaction term between SES and age has been included in models (e.g. House et al., 1994). The

significant interactive effects between SES and age are presumed to represent the effect of SES over the life course. "It would be risky, however, to infer that a cross-sectional age-outcome relationship mimics the trajectory of mean change of individuals" (Miyazaki and Raudenbush 2000:44). Because age and cohorts are confounded in a cross-sectional data, we can not know whether the interactive effect between SES and age reflects cohort differences or development effects (Miyazaki and Raudenbush 2000).

In response to the methodological weakness in cross-sectional data, efforts have recently been focused to use longitudinal data to explore the relationship between SES and health over the life course; however, typical statistics techniques of current longitudinal data analyses have been to regress health measured at the later wave on baseline covariates such as SES, age, risk factors, etc., by controlling the health measured at baseline (e.g. Grundy and Glaser 2000; House et al. 1994; Lantz et al. 2001; Ross and Wu 1995; Zimmer and House 2003). This model could be expressed as the following:

$$Y_{iL} = \beta_0 + \beta_1 * SES_{iE} + \beta_2 * Age_{iE} + \beta_3 * SES_{iE} * Age_{iE} + Y_{iE} + (other\ variables) + \varepsilon,$$

Where Y_{iL} is the health of individual i measured at a later wave, Y_{iE} is the health of individual i measured at the baseline, SES_{iE} and Age_{iE} are measured at baseline.

The above model is convenient in analyzing longitudinal data, especially when researchers do not have enough resources to repeatedly collect information from the same respondents over time; however, it is not optimal. Some significant weaknesses are associated with such method, which might restrict the generation of the findings: 1) the effect of the passage of time is not taken into account in the model; 2) SES, risk factors are treated as time-invariant variables which might fail to capture the reality that they

may actually vary over time, which in turn impact health; 3) Mathematically, we could control the baseline health when we consider the effect of other independent variables; however, in reality, health at baseline will vary corresponding to the change of SES and other independent variables. It is impossible to control health at baseline to explore the effect of SES and other relevant variables on health at a later wave. Thus, regressing health measured at a later wave on SES and other variables by controlling the baseline health may not be optimal; 4) such model doesn't reflect the possibility that people from different SES may have different rate of change in health over the life course; 5) models, using the interaction term between age and SES in order to take into account the different slopes for different SES persons, do not take into account residuals both in the slope and initial status across individuals.

In response to the methodological weaknesses associated with previous research, my study will apply the "Multi Level Model for Change (Singer and Willett, 2003)" (MLMFC)[1] to analyze socioeconomic disparities in health over the life course. The underlining assumption behind the MLMFC is that people from different SES may be differential both at the initial health when a study begins and the rate of change in health over the whole study periods (it is 8 years in Americans' Changing Lives: Wave I, II and III). In other words, higher SES persons may have better health at the beginning of the study and they may also age at a slower rate during the study interval compared to the lower SES ones. As a result, the health of lower SES persons declines faster than their higher SES counterparts. The MLMFC also takes into account the cohort effect of age

[1] The detail descriptions of MLMFC are in chapter 3: Data set and Management

over health in that age at baseline is a level-two predictor for the initial status and rate of change.

This study also explores SES disparities in health over the life course by applying the Multi Cohorts Accelerated Longitudinal Design (MCALD)[2], which uses age at a specific study wave to measure the passage of time. The MCALD suggests that human beings share a common health trajectory over the life course if everything is equal; however, individuals' health trajectories may vary around this common one due to their differentials in social, psychological, environmental and personal characteristics.

In sum, this study will explore SES disparities in health from a longitudinal perspective by applying the MLMFC and MCALD, and explore SES disparities in health over the life course. The other focus of this study is to explore the mediating roles of health risk behaviors (if any) in the link of SES and health over the life course.

Functional limitation and chronic diseases across the 8-year study period are used to measure health. SES, and health risk behaviors measured at baseline are applied to explore how people's health trajectories will be if they are in the same SES or keep the same health risk behaviors over time. Lantz et al. (1998) proposed that health risk behaviors are likely to be stable across different study waves in ACL. However, it is very likely that individuals' SES and health risk behaviors change over time. It is worth exploring how health trajectory changes corresponding to the changes in SES and health risk behaviors over time. Therefore, time variant income and health risk behaviors

[2] The detail descriptions of MCALD are in chapter 3: Data set and Management

measured at different study waves, which represent SES and lifestyle at different study points of time in ACL, are utilized in this study.

Overview of this study

This study consists of six chapters (including chapter 1: introduction). Chapter 2 is a literature review, which provides a conceptual framework for my study. In this chapter, I focus on social causation theory and health risk behavior theory as major theoretical explanations for the link of SES and health. Chapter 3 is data set and measurement. This chapter provides detail information about how variables are created for this study. Special attention has been focused to explain the rationale of using combination of number and duration of chronic diseases as a measurement of health over time. In previous studies, the number of chronic diseases at different study waves has been used to measure health over time. My study argues that the number of chronic diseases at different study waves is not an optimal measurement of health over time, because it may be constant while the health may actually change at different study periods, due to the characteristics of chronic disease. Thus, this study introduces a combination of number and duration of chronic diseases as a measurement of health over time. This measurement will be able to capture the change of health over time even if the number of chronic diseases remains constant. Chapter 4 is results. This chapter actually includes five chapters. Chapter 4-I uses functional limitation as a measurement of health, exploring health trajectory over 8-year interval in ACL. Chapter 4-II extends the findings in chapter 4-I to explore health trajectory over the life course by applying the MCALD. Chapter 4-III uses the number of chronic diseases as a measurement of health over time, exploring health trajectory during

8 years in ACL. Results from this chapter are mainly used to criticize the limitation of the number of chronic diseases as a measurement of health over time. Chapter 4-IV employs the combination of number and duration of chronic diseases as a measurement of health, exploring health trajectory over 8 years in ACL. Chapter 4-V extends the findings from chapter 4-IV to explore health trajectory over the life course by applying the MCALD. Chapter 5 explores social selection hypothesis. Chapter 6 summarizes my findings, contributions and limitations.

CHAPTER 2: LITERATURE REVIEW

1. SES and health

SES disparities in health have been observed back to the earliest records (Williams and Collins 1995) and persist to the present day despite the developed economy and expanded medical care in the United States (Gortmaker and Wise 1997). As a matter of fact, SES disparities in health have been widening since 1960 (Duleep 1989; Feldman et al. 1989; Hummer and Ebersein 1998; Williams and Collins 1995; Pappas et al. 1993). The positive relationship between SES and health has become the most consistent finding in the United States, as well as countries outside of the United States where research has been conducted (Marmot et al 1997; Marmot et al. 1987; Hahn et al. 1995; Mulatu and Schooler 2002; Williams and Collins 1995).

Lower SES groups are disadvantaged across a wide range of health indicators, including mortality, morbidity, disability, life expectancy, physical health, mental health and psychological well-being (Adler and Ostrove 1999; Marmot et al. 1987; Marmot et al. 1997; Guralnik et al.1993). Lower SES people have been observed to have a higher prevalence of all-cause mortality and morbidity; they are more likely to experience lower self-perceived health, mental health, such as anxiety, depression, and psychological well-being (Acheson 1997; Marmot et al. 1997; Turrell et al. 2007; Zimmer and House 2003; Van Rossum et al. 2000). Researchers consistently find strong relationships between SES and cardiovascular disease, stroke, lung cancer, arthritis, tuberculosis chronic respiratory disease, gastrointestinal disease, diabetes and other obesity related disorders (Acheson 1997; Adler and Ostrove 1999; Van Rossum et al. 2000). Lower SES people are also

more likely to experience an incidence of functional limitation, and to get worse upon its onset (Zimmer and House 2003; Grundy and Glaser 2000).

Researchers have indicated that SES disparities in health exist across the whole life course; however, the gap is largest during early childhood and middle age (Siegrist and Marmot 2006). At an early life stage, SES was inversely associated with prenatal mortality, infant mortality rates, prematurity, low birthweight, small size for gestational age, and late birth (Haan et al. 1989; Gortmaker and wise 1997; Wise and Pursley 1992). Gortmaker and Wise (1997) called SES disparities in infant mortality rates "the first injustice" in society. During middle age, lower SES people have a higher prevalence of heart disease and high blood pressure, a higher incident of diabetes, and a higher prevalence of orthopedic impairments associated with injury (Haan et al. 1989). The lowest SES groups continue to experience a higher level of mortality (Kitagawa and Hauser, 1973) even though the overall mortality rate falls; in fact Hummer and Eberstein (1998) argue that individuals at the lowest SES continuum have mortality rates several times higher than those at the highest SES level. During old age, the physical fragility of human beings becomes a major factor of health and the effect of SES declines, which leads to diminished SES disparities in health (House et al. 1994; Siegrist and Marmot 2006).

2. Health risk behaviors theory

A large amount of attention, especially in epidemiology, has recently been focused on health risk factors, which have been considered the major links between SES and health. Researchers have argued that socioeconomic disparities in health are largely due

to the disproportionable exposure of lower SES persons to psychosocial factors (House et al. 1994; Macintyre 1997; Williams 1990).

Evidence from medical or biological areas has clearly shown that health risk behaviors, such as tobacco use, physical inactivity, obesity, and alcohol drinking, will cause Coronary Heart Disease (CHD), stroke, diabetes and other major chronic diseases (Doll et al. 1994; Hu et al. 2000; Kaukanen et al. 1997; Shaper et al. 1997; Siegrist and Marmot 2006). Lantz et al. (1998) find that smoking is associated with an increased risk of tumor deaths; heavy drinking is associated with an increased risk of tumor and cardiovascular deaths. Up to 90% of lung cancer is due to smoking (Harvey et al. 2000). Research from three large US cohort studies shows that exposure to at least one major risk factor (defined as cholesterol level, blood pressure, smoking, and diabetes) accounts for 87% to 100% of fatal Coronary Heart Disease (CHD), and 87% to 92% of non-fatal CHD among women and men respectively (Greenland et al. 2003). Thus, changing health risk behaviors will reduce the risk of diseases (Harvey et al. 2000). Empirical studies have shown that changing health risk behaviors can reduce the incidence of diabetes by 58% (Diabetes Prevention Program Research Group 2002; Finnish Diabetes Prevention Study Group 2001). Up to 80% of cases of CHD and up to 90% of cases of type 2 diabetes could potentially be avoided through changing health risk behaviors and about one-third of cancers could be avoided by eating healthily, maintaining normal weight and exercising throughout life (Stampfer et al. 2000; Hu et al. 2001; key et al. 2002). Marmot et al. (1997) argued that health risk behaviors, together with working environment,

account for about half of SES disparities in self-rated health and for all SES disparities in depression and psychological well-being.

It is clear that poor health behaviors may lead to poor health; however, the distributions of health risk behaviors are not equal in society. Empirical research has indicated that lower SES is associated with a significantly higher prevalence of health risk behaviors (Lantz et al. 1998; Macintyre 1997; Marmot et al. 1987; Siegrist and Marmot 2006). It has argued that lower SES persons are more likely to adopt health risk behaviors, such as smoking, heavily drinking, less physical activity, etc.; they are more likely to be overweight, to eat less healthy food with poor nutrition; they are more likely to be isolated due to their disadvantage status, hence lack of social ties and social supports; they are also more likely to experience both chronic and acute stress, to feel less competitive and less sense of control over their life (House et al. 1994; Jarvis and Wardle 1999; Lantz et al. 1998; Marmot et al. 1987; Siegrist and Marmot 2006; Williams 1990). Furthermore, researchers have argued that socioeconomic disparities in exposure to some health risk factors may vary with age such that socioeconomic differences in most risk factors are modest in early adulthood while they increase during middle age and then diminish later in life (House et al. 1994). Thus, socioeconomic disparity in health over the life course could largely be explained by the differential exposure to risk factors (House et al., 1994).

Some researchers have argued that the disproportional distribution of health risk behaviors among lower SES groups is largely due to personal choice, and independent of living or working conditions (e.g. Berkman and Breslow 1983; Jarvis and Wardle 1999;

Knowles 1977; USDHEW 1979). Researchers, following this argument, argued that individuals should be responsible for their health, because each person decides his/her daily activities. Health risk behaviors, such as smoking, drinking, exercise, sleeping hours, etc. tend to be viewed completely under individual control (Berkman and Breslow 1983; Jarvis and Wardle 1999).The individual has the power to maintain his/her own health by abiding by the rules of healthy behavior relating to lifestyle, such as sleep, exercise, diet and weight, alcohol and smoking (Knowles 1977). Therefore, it is suggested that reduction in socioeconomic disparities in health could come from the modification of prevalent risk factors in lower SES groups (House et al. 1994; Lynch et al. 1996), such as quitting smoking, drinking moderately, sleeping 7 or 8 hours, exercising physically (no sedentary lifestyle), having social ties, etc.

However, other researchers have argued that health risk behaviors are not, or not completely under personal control. "Health behaviors are induced and constrained by the social and material context (Williams 1990: 91). For example, people from lower SES may have limited power to control or influence their life events, and sense of powerlessness is positively associated with health risk behaviors (Coburn and Cope 1974; Williams 1990). Moreover, people from lower SES are more likely to face stress, and have fewer resources to deal with it. Adopting health risk behaviors, such as smoking, may be a way to alleviate the stress (Williams 1990). Social production theory argues that social status could be transferred from generations to generations. Children from different SES groups are socialized into different health behaviors and these behaviors are carried over to adulthood (Coburn and Cope 1974).

3. Social causation theory

Social causation theory, therefore, argues that people's health is socially determined (Marmot and Wilkinson 1999; Link and Phelan 1995; Hayward et al. 2000), and that social stratification is the fundamental social cause of diseases (Hayward et al. 2000; Lantz et al. 1998; Link and Phelan 1995). Position in the social structure is important for health (Brunner and Marmot 1999), because the core resources for health are scarce and unequally distributed across social positions (Siegrit and Marmot 2006). Both subjective reality and the objective conditions of life vary according to one's location in society, which determines differential access to goods and resources that are linked to health (Williams 1990). Social causation researchers argue that health behaviors and other psychosocial factors are the superficial causes that link social status to health outcomes; they have their root in social stratification systems (Williams 1990). "Health behaviors do not occur randomly throughout a population, nor do they occur in a vacuum. Powerful social influences bear on them (Berkman and Breslow 1983:221)". For example, low income neighborhoods have more liquor stores and afford fewer opportunities for exercise and less access to nutritious foods (Macintyre et al. 1993). The combination of individual characteristics and the environmental demands and constraints will affect the likelihood of enacting health-related behaviors such as tobacco use, alcohol use, exercise, and dietary practice (Adler and Ostrove 1999:11). Therefore, social causation theorists argue that "The general advantage or deprivation associated with a social class position would appear to have a sizable long-term influence, over and above the effect of the generally recognized risk factors" (Blane 1999:76). They further argue that "(health risk)

behaviors do not explain away class differences, but contribute to them, and push the explanatory task further back to ask why such behaviors are persistently more common in poorer groups" (Macintyre 1997:728).

Features of adult socioeconomic status do have a causal connection with health in adult life (Marmot et al. 1997:907). Social conditions powerfully influence both the onset and progress of diseases (Zimmer and House 2003). Hayward et al. (1994) argue that social stratification affects individual's ability to control his/her living environment, stress, health behaviors, powers, the nature of work and work environment, as well as access to medical care system.

People with higher income can afford more nutritious food, live in better communities with less pollution and more safety, face less economic uncertainty, and have greater access to better quality health care (Bowling 2004; Preston and Taubman 1994; Siegrist and Marmot 2006). Income influences not only the exposure to or experience of etiological risk factors for health, but also the resources available for the treatment or management of disease or for modifying life circumstances to reduce the factors (e.g. stress) producing or maintaining the disease (Zimmer and House 2003:1090).

Education represents both the socioeconomic position of individuals early in adulthood and a stock of human capital available to them from that time on, both of which influence long-term patterns of exposure to and experience of major psychosocial and biomedical risk factors (House and Williams 1995, 1996). Preston and Taubman (1994) argue that well-educated people are more likely to have higher wages and potential income; thus, they are less likely to face economic hardship. Higher SES people

are able to purchase health-related goods and services, such as medical care. They can also afford to live in a good neighborhood with less pollution and more public service facilities.

Mirowsky and Ross (2000) indicate that employment increases status, power, economic non-dependency, as well as non-economic rewards such as social supports and recognition. They argue that the stress to pay bill, to feed and clothe the family with inadequate income will generate psychological distress, malaise and susceptibility to disease. Those who are laid off or fired, those who cannot find a job, and those with lower sense of control and autonomy, are more likely to be distressed and thus, their physical well-beings are also harmed. "Chronic anxiety, insecurity, low self-esteem, social isolation, and lack of control over work appear to undermine mental and physical health" (Brunner and Marmot 1999:41).

Moreover, social and psychological factors are likely to cluster. As Blane (1995:904) states that social stratification system "structures the life experiences of their members so that advantages and disadvantages tend to cluster cross-sectionally and accumulate longitudinally (Blane 1995:904)." Blane (1999:65) further indicates that:

> Cross-sectionally, advantage or disadvantage in one sphere of life is likely to be accompanied by similar advantage or disadvantage in other spheres. A person whose working environment is free of hazards is likely to reside in good-quality housing, to live in an area of little air pollution, and to have an income that permits a varied diet. In contrast, someone who is exposed to physicochemical and psychosocial hazards during working is at greater risk of occupying damp and

inadequately heated accommodation, of being exposed to industrial and traffic exhaust atmospheric pollution in their area of residence, and of earning an income that restricts dietary choice. Social organization also structures advantage and disadvantage longitudinally. Advantage or disadvantage in one phase of the life course is likely to have been preceded by and to be succeeded by similar advantage or disadvantage in the other phases of life. A child raised in an affluent home is likely to succeed educationally, which will favor entry to the more privileged sectors of the labor market, where an occupational pension scheme will provided financial security in old age. At the other extreme, a child from a disadvantaged home is likely to achieve few educational qualifications and, leaving school at the minimum age, to enter the unskilled labor market where low pay and hazardous work combine with no occupational pension, which ensures reliance on welfare payments in old age.

4. Summary of hypotheses

In sum, evidence show that lower SES does lead to poor health and health risk behaviors do accelerate the disparities in health. Thus, Link and Phelan (1995) argue to contextualize individual level health risk behaviors to more comprehensively understand SES disparities in health.

Based on the literature review and methodology to be utilized in this study, this study will focus on the effect of SES and health risk behaviors on health trajectory over the life course, and to examine the theories of health risk behavior and social causation as

the explanation of SES disparity in health from a longitudinal perspective. Thus, I posit the following hypotheses:

Hypothesis 1: SES is positively associated with health at the beginning of ACL study, but is negatively associated with the rate of change in health over time. Major health risk behaviors mediate the relationship between SES and health both at the beginning of the study and the rate of change over time.

Hypothesis 2: People from different SES groups have different health trajectories over the life course in that higher SES groups age at a slower rate, compared to their lower SES counterparts. Major health risk behaviors significantly mediate SES's effect on health trajectories.

CHAPTER 3: DATA SET AND MEASUREMENT

DATA SET

ACL (Americans' Changing Lives: wave 1 through wave 3) is used for this study. ACL was conducted by the Survey Research Center of the University of Michigan on a stratified, multi-stage, area probability sample of non-institutionalized people aged 25 and older. Black and people aged 60 or over were over sampled (House 2003).

The sample size at the beginning of the survey (wave I in 1986) is 3,617. In 1989 (wave II), 2,867 respondents from first wave participated in the survey. In 1994 (wave III), 2,562 respondents participated in the survey, which included 164 proxy respondents who were not from the original wave I (House 2003).

MEASUREMENT

Chapters in this part focus on the description of variables. Chapter 3-I describes relevant variables used in this study. In Chapter 3-II, I introduce a new method to measure health over time by combining the number and duration of chronic diseases. In previous studies, the number of chronic diseases has been used as a measurement of health over time. However, my study argues against this methodology. Due to the characteristics of chronic diseases, individuals who have chronic diseases at the beginning of the study are likely to have same chronic diseases in the following waves. Thus, their number of chronic diseases is likely to be constant during the study periods unless new chronic diseases occur. This will suggest a constant health over time. Using the number of chronic diseases may not capture the actual change of health behind the constant the number of chronic diseases.

As we know that the incidence of a chronic disease is the result of a cumulative declining process starting with cell level (Lorig, Sobel and Gonzalez 2000). Let us suppose, if we do nothing about chronic disease, we will certainly see that the individual's health will continue to decline until death. Thus, the intensity of a chronic disease will certainly be different depending on its duration. In this study, I argue that the duration of a chronic disease may be a good indicator of intensity, which should be an additional dimension in the measurement of health.

Preliminary mortality analysis shows that individuals who consistently report a chronic disease in wave 1 and wave 2 have significant higher mortality odds in wave 3 than those who only report experiencing chronic disease at either wave 1 or wave 2, and those without any chronic disease have the lowest mortality odds in wave 3. This result indicates that the duration of a chronic disease does matter to individuals' health. Therefore, the combination of number and duration of chronic diseases captures the latent change of health over time even if the number of chronic diseases is constant. I will go into this issue more in depth in chapter 3-II.

CHAPTER 3-I: DESCRIPTION OF VARIABLES

Dependent variables

Functional limitation and chronic disease are two self-report indicators of physical health, which are treated as dependent variables in this study. They were repeatedly measured three times during the 8-year study period, which represents a changing curve in health over time.

Functional limitation at each wave was measured by an index consisting of four self-reported indicators of physical health. The questions were asked: 1) Are you currently in bed or a chair for most or all of the day because of your health (the response categories are 'yes' or 'no')? and How much difficulty do you have: 2) bathing by yourself? 3) climbing a few flights of stairs? 4) walking several blocks? The response categories include: a) no difficulty, b) a little difficulty, c) some difficulty, d) a lot of difficulty, e) cannot do.

A Gutman-type scale was formed to tap the level of functional impairment for the respondent (a revised version based on House 2003): (4) respondents who are currently in bed or chair for most or all of the day because of health are categorized as having the most severe level of functional impairment, (3) respondents who have a lot of difficulty or cannot bathe, or climb stairs or walk blocks, but who were not in the previously defined severity level are categorized as moderately severe, (2) respondents who have some difficulty bathing or climbing stairs or walking blocks, but who are not in the two previously defined severity levels are categorized as least severe level, and (1) respondents who answered no to all of the functional impairment questions are

categorized as no functional impairment. There were no missing values on *functional limitation* at wave 1, however, there were 750 instances of attrition in wave 2 (of whom 214 returned at wave 3), and 1055 instances of attrition in wave 3 due to respondents' dropping out or mortality (536 cases dropped out since wave 2 and 519 cases dropped out only at wave 3).

Chronic diseases were measured by asking the following questions: "We'd like to know if you have experienced any of the following health problems during the last 12 months, 1) a lung disease? 2) hypertension, sometimes called high blood pressure, or have you taken medication for it? 3) a heart attack or other heart trouble? 4) diabetes or high blood sugar, or have you taken medication for it? 5) cancer or a malignant tumor of any kind? 6) a stroke?

The above questions are about life threatening diseases. ACL also asked questions about non-life threatening diseases, including arthritis, foot problem, urinary inconvenience, broken bone. I employ life threatening chronic diseases as a measurement of health in this study, because life threatening and non-life threatening diseases may have different pathological mechanisms.

Both the number of chronic diseases and combination of number and duration of diseases are used in this study. Chapter 3-II: Measurement of Health in Term of Chronic Diseases provides detail description of the rationale and creation of combination measurement.

Measuring the passage of time

Singer and Willett (2003: 10) argue that "Time is the fundamental predictor in every study of change." The measurement of time is crucial for a longitudinal study. In this study, I apply two measurements for the passage of time: one is the passage of years since the first interview at wave 1 (1986). This measurement will provide the landscape about people's health trajectories over the 8-year study periods in ACL. Since ACL respondents had different interview schedules, in which they were interviewed on different days in different months in different waves (1986, 1989, and 1994), the passage of time is not simply 3 years, and 8 years. Instead, different respondents had different time intervals. By arbitrarily assigning 3 and 8 for all respondents (as the latent growth curve model does), the health trajectories might be biased, because some respondents might not have been exposed to chronic diseases for exactly 3 years at wave 2 or 8 years at wave 3. There are variances in the exposure. In this study, I use the actual years between two interview dates as the measurement of the passage of time. For example, if a respondent was first interviewed on November 10, 1986, and he/she was interviewed in May 1989 again, the passage of time is 2.5 years, instead of 3 years.

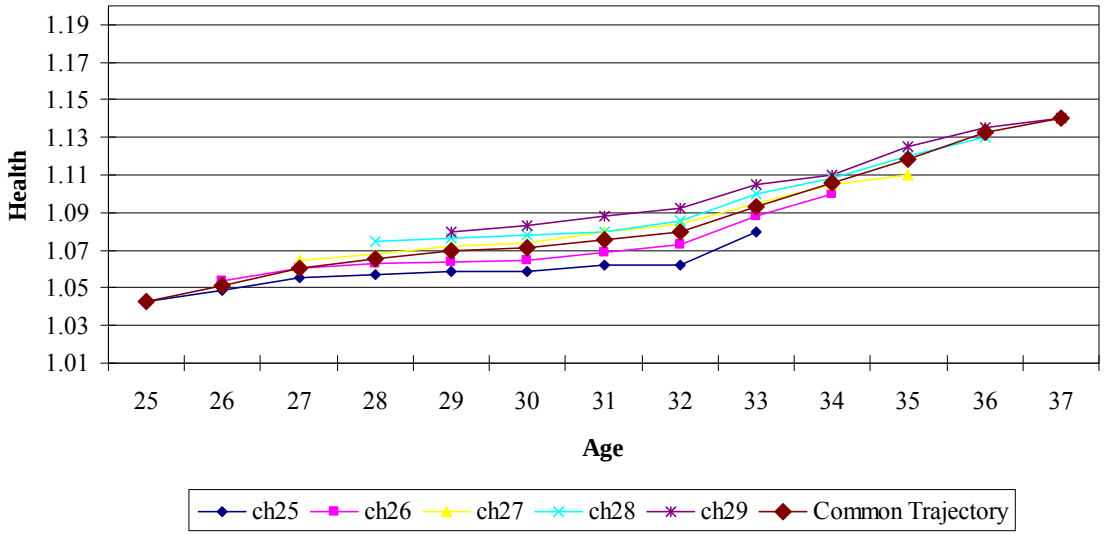
It is, however, more valuable to analyze people's health in the scope of the whole life course. Thus, I also use actual age at a specific wave as a measurement of the passage of time. For example, the health of a 25-year-old respondent at wave 1 will represent the health at age 25; in wave 2 when he/she is 28 years old, the health will represent the health at age 28. Thus, health measured during 3 waves in ACL represents the health trajectory of aging over time. In this way I actually treat ACL as the Multi Cohort

Accelerated Longitudinal Design (MCALD) (Miyazaki and Raudenbush 2000) in that individuals of different ages at first wave are considered as different age cohorts. Thus, there are 71 cohorts in ACL (age varies from 25 to 95 at first wave). Each cohort has an 8-year span during the 3 study waves in ACL. Cohort 1 (age 25 cohort at first wave) ages 25-33; cohort 2 (age 26 cohort at first wave) ages 26-34…cohort 71 (age 95 cohort at first wave) ages 95-103. Therefore, each age cohort has a health trajectory over the 8-year study period, which consists of 71 health trajectories. If health trajectories of adjacent age cohorts overlap within their common age interval (for example, age 25 cohort has common age from 26 through 33 as age 26 cohort during 8 year study interval in ACL), it indicates that these age cohorts share the same health trajectory in the specific age interval. When the age-outcome trajectories do not vary by cohorts, they may be said to converge (Miyazaki and Raudenbush 2000:45). A general life course health trajectory could be created by using the mean levels and rates of change of adjacent cohorts (Miyazaki and Raudenbush 2000). Figure 1 on page 28 is only for illustrative purposes, which is not based on ACL. It aims to illustrate how a common health trajectory could be created based on multi cohorts' trajectories over a relative short period of time. This figure shows that age cohorts of 25 through 29 have their own health trajectories over the 8-year-period. For the age 25 cohort, the health trajectory is from 25 through 33, for the age 26 cohort, the health trajectory is from 26 through 34,..for the age 29 cohort, the health trajectory is from 29 through 37. The adjacent health trajectories overlap during their common age intervals. For example, health trajectories of age cohort 25 and 26 overlap during age 26 through 33. Health trajectories of age cohort 26 and 27 overlap

during age 27 through 34…etc. Thus, a common health trajectory, extending from age 25 through 37, could be used to describe the health trajectory from age 25 through 37, which is the one with diamond as the marker. A common health trajectory over the life course (from age 25 through 103) for ACL could be created based on the same principle. The common health trajectory provides an overall landscape about how people's health change as they age over the life course.

The advantage of the Multi Cohort Accelerated Longitudinal Design (MCALD) is that you can model the health trajectory of the life course using data collected over a short time interval (Miyazaki and Raudenbush 2000; Singer and Willett 2003). The MCALD can also separate the historical effect associated with age cohort from that of age (Miyazaki and Raudenbush 2000; Nesselroade and Baltes 1974).

Figure 1: Common Health Trajectory Based on Adjacent Cohort Health Trajectories



Demographic variables

Age is measured by the respondents' actual age in years on the interview day at each wave. The age of respondent i at each study wave is calculated by the actual days between interview day and birthday divided by 365 days.

In the MCALD, continuous age is the level-one predictor for health, which measures the passage of time. Age is used as a predictor in the level-two model in the MLMFC, which explores the effect of age cohort on health over the 8-year study period. (The MLMFC model is explained in chapter 3-IV)

Socioeconomic variables

Education is measured by the highest school year that respondents finished by wave 1, which varies from 0 to 17. It is categorized into less than high school, high school, college and over. This three-category measure of education is the most parsimonious and useful (Zimmer and House 2003). Less than high school is the reference group. Since most respondents completed their formal education by early adulthood, education is treated as a time invariant variable in data analyses. Twelve respondents who did not report their education level are treated as missing in data analyses. All missing values of variables used in this study are imputed via multiple imputation method, which is explained in chapter 3-III.

Family Income is measured by the income from all resources earned by a family at each wave. Family income at first wave is coded into three categories as House et al (1994) did: $0-9,999, $10,000-29,999, and 30,000 or more, which is treated as a time invariant variable in order to analyze the different health trajectories of different income

cohorts. However, it is very likely that people's income changes during the 8-year study period of ACL. Thus, I also treat family income as a time variant variable to reflect the actual family income at different study periods. The time variant income provides the opportunity to explore how health varies correspondingly to the change of family income.

Health risk behaviors

Health risk behaviors are indicated by smoking, sleeping, drinking, body mass index, and physical activity. They are used as both time invariant and variant variables in analyses. Health risk behaviors measured at the baseline are treated as time invariant. They are used to explore the health trajectory given the assumption of stability of health risk behaviors across different study waves in ACL (Lantz et al. 1998). However, as it is the case in income, health risk behaviors are likely to change over time. Do these changes lead to corresponding changes in health? Time variant health risk behaviors help to explore if the change of health risk behaviors leads to the change of health trajectory.

Due to the availability of health risk behaviors in ACL, I utilize the following health risk behaviors in data analyses.

Smoking is categorized into current smoking versus no smoking.

Sleeping is measured by the hours of sleep (including naps) in a 24-hour period. It is categorized into three dummies: sleep less than seven hours, sleep between seven and nine hours, and sleep more than nine hours. Sleep between seven and nine hours is the reference category.

Drinking is measured by the number of drinks consumed in the past month. I follow previous studies (e.g. House et al. 1994; Lantz et al. 2001) to dichotomize drinking into

three dummies: nondrinker (0 drinks in the past month), moderate drinker (1 to 89 drinks), and heavy drinker (90 or more drinks). Moderate drinker is the reference group.

Body mass index includes three categories: under weight (lowest 5% of cases), normal weight (including low normal (next to lowest 25%), mid-normal (middle 30%), and high normal (next to highest 25%)), and over weight (highest 15%). The categories were developed separately for males and females in ACL (House 2003).

Physical activity is measured by an index based on the following questions: 1) How often do you work in the garden or yard 2) How often do you engage in active sports or exercise 3) How often do you take walks? The response categories are often, sometimes, rarely or never. The standardized index varies from -2.47 to 1.49 (House 2003). A higher score stands for a higher level of physical activity.

Indicator of Stress

Financial chronic stress is used as an indicator of stress level faced by individuals. It is indicated by a standardized index (House 2003), consisting of three items (alpha =0.80): 1) How satisfied are you with (your/your family's) present financial situation -- completely, very, somewhat, not very or not at all satisfied? 2) How difficult is it for (you/your family) to meet the monthly payments on your (family's) bills? Is it extremely difficult, very difficult, difficult, somewhat difficult, slightly difficult, not difficult at all? 3) In general, how do your (family's) finances usually work out at the end of the month -- do you find that you usually end up with some money left over, just enough money to make ends meet, or not enough money to make ends meet? Financial chronic stress in wave 1 is included in models as a time invariant variable to explore how the health trajectory will

be if individuals constantly face the same financial stress. It is obvious that individuals' financial situation may change over time, which leads to different financial stress. Thus, financial chronic stress index measured during 3 waves is used as a time variant variable to explore the relationship between health trajectory and financial chronic stress trajectory over time. The maximum financial stress index is 2.79, and the minimum is -1.8. A higher value stands for a higher level of stress.

Control variables

The main purpose of this study is to explore the effect of SES and health risk behaviors on health trajectory over the life course; therefore, race, sex, and employment status are treated as control variables. They are used as time invariant variables in this study.

Sex is coded as (1) females, (0) males. There were 2259 male and 1358 female at wave 1 in ACL.

Race includes White (2323 respondents), Back (1174 respondents), Hispanic (43 respondents), American Indian (47 respondents), and Asian (30 respondents) in ACL. Since the cases for Hispanic, American Indian, and Asian are small, they are grouped together with White. Therefore, race is recoded as Black versus non-Black.

Employment status is measured by four dummy variables: employed, unemployed, retired, and other. Employed is the reference category.

Chapter 3-II: Measurement of Health in Term of Chronic Diseases

It is very common that chronic diseases are used as a measurement of health. Previous studies used the number of chronic diseases at different study points as the measurement of health over time (e.g. House et al, 1994, 2005). However, I argue that using the number of chronic diseases at different study points as a measurement of health could not track the actual change of health over time due to the essential characteristics of chronic diseases. Unlike "acute" diseases, chronic diseases start with diseased cells. The occurrence of a chronic disease is a result of the accumulation of cellular malfunction, which in turn leads to organ or tissue dysfunction (Lorig, Sobel and Gonzalez 2000). Thus, chronic diseases begin slowly and proceed slowly (Lorig, Sobel and Gonzalez 2000:2). People won't realize that they have a chronic disease until they have symptoms (such as shortness of breath, fatigue, pain, felling blue, etc.) (Lorig, Sobel and Gonzalez 2000).

Using the number of chronic diseases at different study points may not actually reflect the process of chronic diseases. In this study, I combine both the incidence and duration of chronic diseases as the measurement of health over time. This measurement will capture the process of chronic diseases by including the duration of diseases; in turn change of health over time would be indicated even thought the total number of chronic diseases may remain constant. In the following paragraphs, I mainly discuss the rationale and advantages associated with the combination of number and duration of chronic diseases as a measurement of health over time and the disadvantages of count measurement in a longitudinal study.

Using the number of chronic diseases at each study period as the measurement of health

Previous researchers used the total number of chronic diseases experienced by respondents as the measurement of health at each wave (e.g. House et al 1994, 2005). This measurement gives a snapshot of the incidence of chronic diseases at each study point, which indicates the existence of chronic diseases. It measures the change of health by compared the total number of chronic diseases at different study periods (e.g. House et al. 1994, 2005). If a respondent's total number of chronic diseases increases, then his/her health is considered to decline. Otherwise, if his/her total number of chronic diseases is constant, then his/her health is considered to be stable. However, researchers have argued that chronic diseases are often long-lasting and persistent in their effect over time (AIHW 2007; Lorig, Sobel and Gonzalez 2000; Walker and Peterson 2007). Therefore, the number of chronic diseases is likely to be stable over time unless a new chronic disease appears. In a longitudinal study, the measurement of health over time should be able to capture the change of health even though the number of total chronic diseases may remain stable over time. Obviously, using the number of chronic diseases as a measurement of health over time is not able to fulfill this task. Moreover, in the count measurement, there are two unstated assumptions: 1) the same number of chronic diseases across different people indicates same health, and 2) the same number of chronic diseases of a respondent at different study periods indicates the same health. However, these assumptions are questionable. The following paragraphs explain the two assumptions.

People with the same number of chronic diseases are considered having the same health in the count measurement

In the count measurement, people having the same amount of chronic diseases are considered having the same health, no matter how long a person has experienced the chronic disease. For example, a person who experienced a chronic disease for over 5 years will be considered as having the same health as those who experienced the same chronic disease for only 1 month at a specific study period, because they have same the number of chronic diseases. However, it is common sense to believe that the same amount of chronic diseases may have different meanings for these two persons. Count measurement largely ignores the intensity of chronic diseases over time; that is, the length of chronic diseases experienced by respondents is not taken into account in measuring health. In a cross-sectional study, researchers lack necessary information to measure the intensity of a chronic disease; thus, using the number of chronic diseases as a measurement of health may be an optimal choice. However, in a longitudinal study, researchers have the advantage to repeatedly gather information from the same persons. Thus, the intensity of a disease as a dimension of health should be included in measuring health in a longitudinal study.

People with the same number of chronic diseases across study points are considered as having stable health in the count measurement

In the count measurement, people whose the number of chronic diseases remains constant across different study periods are considered as having stable health. However, it

is reasonable to assume that the same counts of chronic diseases at different study points may actually have different meanings.

As we know that the decline of health is accumulated with time. Chronic diseases don't occur all of a sudden; they proceed gradually (Lorig, Sobel and Gonzalez 2000; Rose 1982). The occurrence of a chronic disease may be the result of an accumulation process. Chronic diseases can lead to a loss of function, stress, depression, or even death (Lorig, Sobel and Gonzalez 2000; Walker and Peterson 2007). Managing chronic diseases requires lots of resources, including financial, emotional and social resources.

The longer a chronic disease exists, the more resources are needed. In turn, an individual may experience stress, frustration, or hopelessness (Lorig, Sobel and Gonzalez 2000). Different diseases management may lead to different health outcome (Lorig, Sobel and Gonzalez 2000), because the severity of chronic diseases and the impact are possible to be reduced (Walker and Peterson 2007; Lorig, Sobel and Gonzalez 2000). For lower SES people, they may not have equal resources to manage chronic diseases, while higher SES is in a much better position in term of resources. Therefore, same chronic diseases may lead to different health outcomes for people from different SES. The longer a chronic disease persists, the more of an impact it will have on a person's health.

Therefore, the duration of a chronic condition may act like a latent variable that actually indicates a person's health. It makes sense to assume that a respondent has worse health if he/she has the same chronic diseases across three waves than the one who only has chronic diseases at either wave. Only using the number of chronic diseases as a

measurement of health over time may lose large amounts of information about changes in health.

Disadvantages associated with count measurement

It is convenient to use the number of chronic diseases as a measurement of health for respondents in a cross-sectional data set; however, in a longitudinal study, the goals are not only to measure health of respondents at a specific point of time, but also to track the changes of health over time such that researchers will be able to explore the different health trajectories across SES groups and to identify factors responsible for such differences. Thus, it is crucial for a health measurement in a longitudinal study to actually track the change of health over time.

Obviously, it is not optimal to use the number of chronic diseases as a measurement of health in a longitudinal study due to the complexities and essence of chronic diseases.

Using the number of chronic diseases as a measurement of health over time may lead to biased results

It is obvious that the same the number of chronic diseases of respondents at different waves may have different meanings if the duration of diseases is different. For example, two persons reported experiencing diabetes in the ACL study. The first person consistently experienced diabetes over the 3 waves, which means he/she suffered diabetes for at least 8 years; the second person experienced diabetes only at wave 3. The corresponding disease occurrence patterns for these two persons were 111 and 001 respectively (where 1 stands for occurrence and 0 stands for nonoccurrence). The first person's health was considered stable over the 8-year study period because his/her

chronic disease count was constant, while the second person's health declined over time. We might further conclude that people would eventually reach equal health as time goes by even though they had different health at the beginning of the study. However, the truth is that large amounts of information about the intensity of chronic diseases experienced by the first person were excluded from analysis. The first person had already been suffering from diabetes for at least 8 years; he/she was consistently facing stress, getting worse or even dying; while the second person just started to experience diabetes at the end of the study. Thus, in the count measurement, the health of respondents with more chronic diseases at wave 1 was downwardly biased at wave 2 and wave 3.

Advantages associated with using the combination of number and duration of chronic diseases as measurement of health

Using the number of chronic diseases is not optimal in measuring health over the life course as indicated above. In this study, I use a combination of number and duration of chronic diseases as a measurement of health over time, which includes both occurrence and intensity of chronic diseases. The following paragraphs show how count and duration of chronic diseases are combined as a measurement of health.

There are eight patterns of the occurrence of a chronic disease during 3 waves: 111, 110, 101, 100, 011, 010, 001, and 000 (where 1 stands for occurrence, 0 stands for no occurrence). The distribution of chronic diseases during 3 waves is in Table 3.1 on page 46. Due to the left censoring of chronic diseases in ACL, there is no information about the duration of chronic diseases before wave 1. So I use the number of chronic diseases experienced by respondents during the last 12 months as the indicators of health of

respondents at wave 1. In wave 2 and wave 3, the duration of chronic diseases since wave 1 is taken into account in measuring respondents' health, which are calculated as followed: In pattern 111, the chronic disease is assigned 1 point at wave 1, 3 points at wave 2 (which indicates that this disease lasts for 3 years since wave 1), and 8 points at wave 3 (which indicates that this disease lasts for 8 years since wave 1). In pattern 110, the chronic disease is assigned 1 point at wave 1, 3 points at wave 2, and 0 point at wave 3. In pattern 101, the chronic disease is assigned 1 point at wave 1, 0 point at wave 2 and 1 point at wave 3. This same rationale is applied to the left occurrence patterns of chronic diseases. Thus, the corresponding scores of the chronic disease to the eight occurrence pattern would be: 138, 130, 101, 100, 015, 010, 001 and 000.

The overall measurement of health of respondents at each wave is the sum of the combination of number of chronic diseases and duration of them experienced by respondents. A higher value indicates worse health. In data analyses, I use a natural logarithm of it to reduce positive skewness of this variable.

The advantage of the above measurement is that it not only takes into account the occurrence and duration of a chronic disease, but also reflects the intensity of chronic diseases during 3 waves.

For the purpose of comparison, I parallel employing both count and combination measurements to the same models and compare the results.

Inconsistency in chronic diseases pattern over time

Previous research has indicated that the intensity of most chronic diseases could go up and down over time (Lorig, Sobel and Gonzalez 2000). Chronic diseases can be

prevented, controlled, or even cured (Lorig, Sobel and Gonzalez 2000; Walker and Peterson 2007; WHO 2007). Data from ACL is consistent with previous findings. Based on ACL data, chronic diseases did come and go for some respondents over the three study waves. Some respondents reported experiencing chronic diseases at an early wave; however, he/she didn't report the same chronic disease at a later wave. As illustrated in Table 3.1 on page 47, T1 refers to those cases where respondents report individual chronic disease at wave 1 only, but neither at wave 2 nor at wave 3; T2 refers to cases where respondents report individual chronic disease at wave 2, but neither at wave 1 nor at wave 3. T3 refers to those cases where respondents report individual chronic disease at wave 3, but neither at wave1 and wave 2; T1&T2 refers to those cases where respondents report individual chronic disease at both wave 1 and wave 2, but not at wave 3; T1&T3 refers to those cases where respondents report individual chronic disease at wave 1 and wave 3, but not wave 2; T2&T3 means that respondents report individual chronic disease at wave 2 and wave 3, but not wave 1. T1, T2&T3 means that respondents consistently report experience chronic disease during all three waves.

Two possible explanations for this inconsistency of chronic diseases over the 8-yearstudy interval are: 1) those respondents may misreport their chronic diseases either at early or later wave. The patterns for possible misreporting may include 100, 101, and 010 (where 1 stands for occurrence, 0 stands for nonoccurrence). That is, those respondents who reported chronic diseases at early waves but not at later wave may actually not experience chronic diseases at the early wave (in this case, the chronic disease pattern should be 000, 000, and 000), or they may experience chronic diseases at later waves, but

they failed to report them (in this case, the chronic disease pattern should be 111, 111, and 111); or 2) the respondents might control the chronic diseases due to good disease management, medical surgery (e.g. cancer), diet, or healthy behaviors, etc. (Lorig, Sobel and Gonzalez 2000; Walker and Peterson 2007; WHO 2007), such that the impact of chronic disease was under "control" or became "inactive" and respondents were able to live a healthy life. Thus, the inconsistency in chronic diseases over three waves might actually reflect the respondents' health ups and downs over time.

However, it is hard to prove either explanation based on survey data from the ACL. Since the number of respondents who reported chronic diseases come and go is not negligible, it is unlikely that all those respondents misreported their chronic conditions. Moreover, previous research has argued that chronic diseases can be controlled, or cured. Thus, it is very likely that some chronic diseases do "go away" or are under control over time for some people. It is reasonable to assume that a person is healthier when chronic diseases are under "control" than when chronic diseases are still active. The longer a chronic disease is active, the worse a person's health may be. Therefore, people's health can go up or down over time even if they had chronic diseases.

The inequality in a disease's controlling among people points to the need to look into the factors responsible for such inequality as we know that chronic diseases may require lots of resources to deal with. The longer a chronic condition lasts, the more resources or efforts are required. In turn, the impact of the chronic disease on daily life is larger. According to Link and Phelan (1995), social condition is the fundamental cause of diseases. SES may play an important role in the inequality in a disease's control among

people. High SES people may have more resources to control the environment where they live, such as living in less polluted areas, working in less hazardous environments, having a healthy lifestyle, etc., such that they have a lower rate of disease incidence. They may also have more resources to control or even cure a disease, such as medical surgery, using the health care system, etc.. Therefore, they may have a higher chance to stay healthy even if they have chronic diseases. In contrast, low SES people lack necessary resources to control their living environment such that they are more likely to live in disadvantaged communities, eat less healthy food, as well as lead a less healthy lifestyle, etc. They may also lack resources to cure or control a disease such that the negative effect of a disease will be consistently active.

As a result, there is not only an inequality in the incidence and prevalence in diseases among different SES people, but also an inequality in the disease control among them. This may result in double jeopardy for low SES people: on the one hand, low SES people have a higher prevalence of chronic diseases than high SES ones; on the other hand, they will suffer chronic diseases longer and will be less likely to cure (or control) them.

If it is true that SES plays an important role in disease control, we might further ask: Do high SES people have an advantage in all chronic disease control over low SES people or just in some particular chronic diseases. However, this is beyond the scope of my dissertation.

[TABLE 3.1 ABOUT HERE]

Duration of chronic diseases and mortality

Does the duration of a chronic disease affect people's longevity more than the chronic disease per se? In other words, is the combination of number and duration of chronic diseases a better indicator of health over time than the number of chronic diseases only? Previous research does not explore this topic. This paragraph aims to provide an empirical support for using the combination of number and duration of chronic diseases as a measurement of health in a longitudinal study by exploring the relationship between the duration of a chronic disease and mortality.

As shown in Table 3.1, some respondents report experiencing chronic disease(s) at only one wave, some report experiencing chronic disease(s) at two waves, and some consistently report experiencing chronic disease(s) through the 8-year study period in ACL. If the duration of a chronic disease does not add additional information to the occurrence of the disease in predicting mortality, then those who report chronic diseases at one wave should have the same probabilities of death as those who consistently report chronic diseases in 2 waves or 3 waves given other things constant.

To test this hypothesis, I apply logistic regression of the log-odds of the probability of mortality after wave 2 (1989) on individual chronic disease at wave 1 and wave 2. The model could be expressed as:

$$Logit(p) = \log(\frac{p}{1-p}) = \beta_0 + \beta_1 * X_1 + \beta_2 * X_2 + \beta_3 * X_{12}$$

Where p is the probability of death after wave 2, X_1 is a dummy variable, indicating the occurrence of individual chronic disease at wave 1 only, X_2 is another dummy

variable, indicating the occurrence of individual chronic disease at wave 2 only, and X_{12} is a dummy variable, indicating the occurrence of individual chronic disease at both wave 1 and wave 2. The reference group is those without chronic diseases at either wave.

Table 3.2 on page 47 shows the results from logistic regression of the log-odds of the probability of mortality on 6 individual chronic diseases: in general, respondents who report any chronic disease at either wave have a higher mortality rate after wave 2 than those without any chronic disease. Moreover, those who consistently report same chronic diseases at 2 waves have a higher mortality rate than those only reporting chronic diseases at 1 wave. If an individual chronic disease exists at both wave 1 and wave 2, the duration of this individual chronic disease is longer than that of individual chronic disease existing at only one wave. Preliminary results from the logistic regression model provide evidences that the duration of chronic diseases does increase the probability of death. Including the duration of chronic diseases as a measurement of health may provide additional information in measuring a person's actual health over time.

Since the above models separately use individual chronic disease as a predictor of mortality rate, there may be a confounding effect from other chronic diseases. In order to exclude the confounding effect, I include 6 chronic diseases together in predicting the mortality rate. Model 1 (in table 3.3 on page 48) shows that individuals who report experiencing chronic diseases generally have a higher mortality rate than those without any chronic diseases. Moreover, individuals who consistently report experiencing chronic diseases at both waves have a higher mortality rate than those reporting chronic diseases at either wave 1 or wave 2.

Model 2 adds age to model 1. It shows that individuals who report experiencing chronic diseases only at wave 1 have the same mortality rate as those without chronic diseases at all; those who report experiencing chronic diseases (except hypertension and heart attack) only at wave 2 and those individuals who consistently report experiencing chronic diseases (except hypertension and stroke) at both waves continue to have a higher mortality rate than those without chronic diseases. Those consistently reporting chronic diseases (except stroke) at 2 waves have a higher mortality rate than those only reporting chronic diseases at wave 2.

Age is positively associated with mortality rate when keeping chronic diseases constant. This, to some extent, may represent the fragility of human body due to the aging process and the effect of other life threatening diseases not included in the ACL study.

Results from table 3.2 and table 3.3 indicate that the duration of a chronic disease does have significant effect on mortality. It provides additional information to the occurrence of chronic disease(s) in predicting the probability of mortality. Thus, it is optimal to include both number and duration of chronic diseases as a measurement of health in a longitudinal study.

[TABLE 3.2 ABOUT HERE]

[TABLE 3.3 ABOUT HERE]

Table 3.1: The Distribution of Chronic Diseases over Three Waves

	Hypertension	Lung disease	Heart attack	Diabetes	Cancer	Stroke
None	1323	2078	1964	2036	2176	2239
T1	66	35	62	23	28	5
T2	60	53	72	28	29	15
T3	202	100	120	78	79	62
T1&T2	73	15	26	11	5	1
T1&T3	67	8	26	13	5	1
T2&T3	75	14	28	33	12	8
T1, T2&T3	469	30	46	118	7	2
1 Wave	328	188	254	129	136	82
2 Waves	215	37	80	57	22	10
3 Waves	469	30	46	118	7	2

Note:
T1 refers to cases where respondents report chronic diseases at wave 1, but neither at wave 2 nor at wave 3.
T2 refers to cases where respondents report chronic diseases at wave 2, but neither at wave 1 nor at wave 3.
T3 refers to cases where respondents report chronic diseases at wave 3, but neither at wave 1 nor at wave 2.
T1&2 refers to cases where respondents report chronic diseases both at wave 2 and wave 1, but not at wave 3.
T1&3 refers to cases where respondents report chronic diseases both at wave 1 and wave 3, but not at wave 2.
T2&3 refers to cases where respondents report chronic diseases both at wave 2 and wave 3, but not at wave 1.
T1, 2&3 refers to cases where respondents report chronic diseases all 3 waves.
1 wave refers to cases where respondents report chronic diseases at only 1 wave.
2 wave refers to cases where respondents report chronic diseases at 2 waves.
3 wave refers to cases where respondents report chronic diseases at 3 waves.

Table 3.2: Logistic Regression of the Log-odds of the Probability of Mortality on Individual Chronic Disease at Wave 1 and Wave 2

		Coefficient	Coefficient	Coefficient	Coefficient	Coefficient	Coefficient
Hypertension	Intercept	-2.15***					
	T1 only[a]	1.14***					
	T2 only[a]	0.77***					
	T1&T2[b]	0.96***					
Lung disease	Intercept		-1.80***				
	T1 only[a]		0.21				
	T2 only[a]		1.02***				
	T1&T2[b]		1.23***				
Heart attack	Intercept			-1.92***			
	T1 only[a]			1.08***			
	T2 only[a]			1.06***			
	T1&T2[b]			1.46***			
Diabetes	Intercept				-1.94***		
	T1 only[a]				0.98**		
	T2 only[a]				1.21***		
	T1&T2[b]				1.56***		
Cancer	Intercept					-1.78***	
	T1 only[a]					1.12***	
	T2 only[a]					1.11***	
	T1&T2[b]					1.24**	
Stroke	Intercept						-1.78***
	T1 only[a]						1.44*
	T2 only[a]						1.78***
	T1&T2[b]						2.47**
	Chi-SQR	2,365.28	2,416.73	2,368.43	2,342.19	2,423.03	2,405.29
	df	4	4	4	4	4	4

Note:

a: stands for occurrence of chronic condition at either wave 1 or wave 2 only. The reference is non-occurrence of chronic disease at both waves.

b: stands for occurrence of chronic condition at both wave 1 and wave 2. The reference is non-occurrence of chronic disease at both waves.

* $p< 0.05$; ** $p< 0.01$; *** $p< 0.001$

Table 3.3: Logistic Regression of the Log-odds of the Probability of Mortality on Individual Chronic Disease at Wave 1 and Wave 2, Age at wave 1

		Model 1	Model 2
		Coefficient	Coefficient
	Intercept	-2.38***	-7.25***
Hypertension	T1 only[a]	0.74***	0.14
	T2 only[a]	0.43†	0.24
	T1&T2[b]	0.48***	-0.06
Lung disease	T1 only[a]	-0.05	-0.17
	T2 only[a]	0.62*	0.59*
	T1&T2[b]	0.94**	1.13***
Heart attack	T1 only[a]	0.59**	0.29
	T2 only[a]	0.60**	0.32
	T1&T2[b]	0.75**	0.48*
Diabetes	T1 only[a]	0.56	0.39
	T2 only[a]	0.76**	0.67*
	T1&T2[b]	1.26***	1.11***
Cancer	T1 only[a]	0.87*	0.46
	T2 only[a]	1.04***	0.69*
	T1&T2[b]	1.06*	0.96†
Stroke	T1 only[a]	0.79	0.46
	T2 only[a]	1.40***	0.78*
	T1&T2[b]	1.58†	1.45
Age at T1			0.08***
	Chi-SQR	2,204.24	1,830.74
	df	19	20

Note:

a: stands for occurrence of chronic condition at either wave 1 or wave 2 only. The reference is non-occurrence of chronic disease at both waves.

b: stands for occurrence of chronic condition at both wave 1 and wave 2. The reference is non-occurrence of chronic disease at both waves.

† $p<0.10$; * $p< 0.05$; ** $p< 0.01$; *** $p< 0.001$

Chapter 3-III: Missing Data in a Longitudinal Study

A commonly encountered problem in the analysis of survey data is that some of the data are missing (Glasser 1964; Rubin 1976; Little 1992; Allison 2002). In a longitudinal study such as ACL, there are two types of missing data: one is due to attrition, i.e. some respondents may drop out due to various reasons. In this case, they may not provide information at all study time periods (Hedeker and Gibbons, 1997, Singer and Willet, 2003). Second reason for missing data is when some responses are not observed on some variables. That is, although respondents are interviewed at each study period, they don't answer or provide information on some variables. For example, respondents might refuse to report their income in ACL. Or respondents might forget to answer or overlook some of the questions. Or it is possible that respondents simply do not have the information required for some questions. Missing data may also result from invalid coding. In general, we distinguish those unobserved responses from others as "missing" in data analyses because they provide no information for that particular characteristic (e.g. income in the example above). Since statistical inference is the fundamental objective of quantitative research (Mackelprang 1970), the validity of statistical inference with missing data is of great importance to researchers.

Little and Rubin (1987, 2002) and Allison (2002) indicate that making sampling distribution inferences about a statistic is appropriate if the missing data are "missing completely at random" or "missing at random." The data on variable x are said to be missing completely at random (MCAR) if the probability of missing data on variable x is unrelated to the value of x itself or to the values of any other variables in the data set

(Allison 2002:3). The data on variable x are said to be missing at random (MAR) if the probability of missing data on variable x is unrelated to the value of x itself. For example, if variable x is income and being missing on income does not relate to the value of income nor does it relate with other variables, it is called "MCAR". Then standard statistical inference for the population is valid. If respondents with high income are less likely to respond, then missing on variable x is not "missing at random"; standard statistic inference for the population is not valid. If older respondents are less likely to report their income, this means missing on variable x is associated with variable age rather than with income per se, then the missing data are "missing at random" but not "missing completely at random"; In this case, standard statistical inference for the population is valid. In a longitudinal design, ignorable attrition falls under "missing at random" (MAR) (Hedeker and Gibbons, 1997).

Dealing with missing data due to attrition

In a longitudinal study, there are two kinds of data collection schedules: time-structured and time-unstructured (Singer and Willet 2003). If every respondent is interviewed on an identical schedule, we say that the data is time-structured. If data collection schedules vary across individuals, the data is time-unstructured (Singer and Willet 2003). In ACL, the data was time-unstructured in that individuals have different interviewed schedules. Moreover, ACL could be considered as an unbalanced design due to attrition during the 8-year study period. 750 respondents dropped out at wave 2, among whom 214 returned at wave 3. These 214 respondents could be considered as being interviewed twice (instead of three times) in the 8-year study period. 536 respondents

dropped out since wave 2. Additional 519 respondents dropped out only at wave 3. Those dropping out since wave 2 and those dropping out at wave 3 only would be considered as missing at wave 3.

A major advantage of the multilevel model for change is its flexibility for dealing with attrition (Hedeker and Gibbons 1997; Singer and Willet 2003). Respondents don't have to participate in all study time points to contribute the data set. Respondents dropping out in the following study period are still included in data analyses. "Although they provide less, or no, information about within-person variation and hence do not contribute to variance component estimation—they can still contribute to the estimation of fixed effects where appropriate. Ultimately, each person's fitted trajectory is based on a combination of his: 1) observed trajectory, and 2) a model-based trajectory determined by the values of the predictors" (Singer and Willet, 2003:148). In a longitudinal design, the multilevel model for change with maximum likelihood estimation provides valid inferences if attrition is ignorable (Laird 1988). Ignorable attrition means that the probability of drop out depends on observed covariates and previous values of the dependent variable from the drop-out respondents (Hedeker and Gibbons, 1997). To be more specific, if the respondent attrition is related to previous level of dependent variable (e.g. respondents who drop out due to bad health) and other observable subject characteristics, then this drop out is ignorable and maximum likelihood based multilevel model for change provides valid statistical inferences (Hedeker and Gibbons 1997).

The raw data in ACL is organized as person-level data in that each person has only one record and repeated measurements of each outcome at different study points appear

as additional variables (Singer and Willet 2003). Person-level data has to be reorganized into person-period format for the multilevel model for change, such that each person has multiple records, one for each study period in which he or she was observed (Singer and Willet 2003). The following table (on page 54) gives an example of how longitudinal data set is organized and how ignorable attrition (MAR) is handled in a person-period data set.

Person-level data set
Table A

	Wave 1 (1986)		Wave2 (1989)		Wave3 (1994)	
ID	Age1	Gender1	Age2	Gender2	Age3	Gender3
001	24	1	27	1	32	1
002	30	2	33	2	.	.
003	45	1	.	.	53	1
..	..	..	..	..	..	..
..	..	..	..	..	..	..
3617	60	2	.	.	.	.

⇩

Person-period data set
Table B

ID	Age	Gender	Wave
001	24	1	1
001	27	1	2
001	32	1	3
002	30	2	1
002	33	2	2
002	.	.	3
003	45	1	1
003	.	.	2
003	53	1	3
..	..	..	..
..	..	..	..
3617	60	2	1
3617	.	.	2
3617	.	.	3

⇩

Person-period data set for MLMFC
Table C

	ID	Age	Gender	Wave
	001	24	1	1
	001	27	1	2
	001	32	1	3
Case 002 has 2 records	002	30	2	1
	002	33	2	2
Case 003 has 2 records	003	45	1	1
	003	53	1	3
	..	..	..	..
	..	..	..	..
Case 3617 has 1 record ←	3617	60	2	1

As shown in table A, each person has only one record in a person-level data set. Repeated measurement of age and gender is indicated as age1, age2, age3, gender1, gender2 and gender3. In a person-period data set, each person (shown in table B) has three records and repeated measurement of same outcome in different study points appear in one variable. If a respondent drops at any wave, all his/her variables (except ID) become missing. Table C represents a person-period data set excluding those dropped cases; this is the data set used in the Multi Level Model for Change (MLMFC). As we see in table B, case 002 drops at wave three and case 003 drops at wave 2 but comes back at wave 3. In table C, these two cases both have two records. Since ACL is a time-unstructured design, case 002 and 003 could be considered as scheduled to be interviewed twice (instead of three times). Case 002 would contribute to the estimation of within-individual change and between-individual changes for 3 years (1986 through 1989). Case 003 would contribute to the estimation of both within and between individual changes for 8 years. Case 3617 only has wave 1 information, thus, it could not provide information about within-individual change; however, it contributes to the estimation of the fixed effect at wave 1.

In sum, attrition in ACL could be considered as "Missing at Random"; references based on reduced sample size over the 8-year study period are valid.

Dealing with missing data due to no response to some variables

In a longitudinal study as well as a cross-sectional study, missing information on some variables is almost unavoidable. Researchers have proposed several methods to deal with missing data due to no information on observed variables, which can be classified

into three types: 1) complete-case analysis; 2) available-case analysis; and 3) imputation of missing data.

Complete-case analysis is also known as *listwise* or *casewise* deletion (Allison 2002). Cases with missing values are simply excluded from the analysis and standard statistical method are be applied to the remaining cases. This strategy has been applied frequently in social science. It is simple and no special computational methods are required (Little 1992; Little and Rubin 2002; Allsion 2002). However, listwise deletion often excludes a large fraction of the original sample. For example, suppose a data set has 10,000 cases and we want to estimate a multivariate regression model with 20 variables. Also suppose that each variable is missing data on 5% of the cases, independently of other variables. This will only leave 0.95^{20} (which is the proportion .3584) of the cases with complete data. We would only have 3584 cases left from the original sample, losing much useful information of the dropped cases. A relevant question is whether a subsample with such a dramatically reduced number of observations will still represent the population. According to Allison (2002): if the missing data are MCAR, then the reduced sample will be a random subsample of the original sample. Thus, for any parameter of interest, if the estimates would be unbiased for the full data set (without missing data), they will also be unbiased for the listwise deleted data set…. On the other hand, if the data are not MCAR, but only MAR, listwise deletion can yield biased estimates. For example, if the probability of missing data on schooling depends on occupational status, regression of occupational status on schooling will produce a biased estimate of the regression coefficient.

Available-case analysis is also called *pairwise deletion* (Allison 2002). The largest sets of available cases are used for estimating individual parameters (Little and Rubin 1987, 2002). This method seems to be an improvement over listwise deletion. However, there are obvious disadvantages. The sample base changes from variable to variable according to the missing data pattern. Moreover, the correlation coefficients between two variables can lie outside the range from -1 to +1 (Little and Rubin 2002).

Imputation of missing data is a method, which has been applied broadly. The basic idea for imputation is to substitute some reasonable guess (imputation) for each missing value and then do the analysis based on the complete data from imputation (Allison 2002). There are various methods for imputation:

1) *Marginal mean* imputation, in which the mean from the observed cases of a variable is imputed for the missing value so that the data set becomes complete. This is the simplest type for imputation, but marginal mean imputation underestimates the variance and covariance between variables (Little and Rubin 2002).

2) *Least squares* imputation, in which a missing value on a variable is imputed by linear regression using the observed cases. This method is also called conditional mean imputation (Allison 2002). For example, if x_1 has missing data while x_2 to x_5 have complete cases, we could regress x_1 on x_2 to x_5 from the complete cases, then impute the missing value of x_1 with the predicted values x_1_hat from the regression. However, this method underestimates the standard errors and in turn overestimates test statistics and statistical significance (Allison 2002).

The above two methods of imputation substitute an expected value for the missing value, leading to an underestimate of the variance of those variables with missing values. Single value imputation does not reflect sampling variability about the actual value as well as additional uncertainty (Rubin 2004). Therefore, these methods are not optimal.

3) *Maximum likelihood imputation*.

Anderson (1957) first introduced maximum likelihood methods to deal with missing data. A maximum likelihood (ML) estimate of θ is a value of $\theta \in \Omega_\theta$ that maximizes the likelihood $L(\theta|Y)$, or equivalently, the loglikelihood $\ell(\theta|Y)$ (Little and Rubin 2002:99). The basic principle of maximum likelihood estimation is to choose estimates that maximize the probability of observing the sample (Allison 2002).

There are a number of ways to obtain maximum likelihood estimators, and one of the most common is called the Expectation-Maximization algorithm, abbreviated as the EM algorithm. Allison (2002) and Little and Rubin (2002) give a detailed description of the EM algorithm, but it can be briefly summarized as follows. The EM algorithm include two steps. In the expectation step (E), the conditional expectated values of the "missing data" given the data and unknown parameters are estimated, and these expected values are substituted for the "missing data". In the maximization step (M), the unknown parameters are estimated, using the imputed data and maximum likelihood. Once we get estimates for the mean and variance matrix, we begin another round of E and M steps until the estimates converge.

4) *Multiple imputation*.

Multiple imputation is the technique that replaces each missing value with two or more acceptable values representing a distribution of possibilities (Rubin 2004). The idea behind multiple imputation is that for each missing datum we impute several values, say *m,* instead of just one. The *m* imputations for each missing datum create *m* complete data sets. Each complete data set is analyzed using standard procedures for complete data (Rubin 2004). Therefore, we get *m* sets of regression coefficients. We use *the means of the regression coefficients* as the final regression coefficient.

By imputing more than one value for each missing observation, uncertainty is introduced into analysis. Multiple imputation has more advantages compared to other methods. However, multiple imputation does not produce a determinate result. Every time we do multiple imputation for the same data set, we get slightly different estimates and associated statistics (Allison 2002).

By compared and contrasting disadvantages and advantages associated with the different methods, I apply multiple imputation in my dissertation to impute missing values.

Chapter 3-IV: METHODOLOGY

This study will apply the Multi Level Model for Change (MLMFC) which uses the passage of years since first interview in the ACL study as the measurement of time and the Multi Cohorts Accelerate Design (MCALD) which uses age at interview as the measurement of time to explore SES disparities in health over the 8-year study and over the life course.

Multi Level Model for Change (MLMFC)

The MLMFC model could be expressed as the following equation (**Equation 1**):

$$Y_{ij} = \pi_{0i} + \pi_{1i} * Time_{ij} + \varepsilon_{ij} \longrightarrow \text{Level-one Model}$$

$$\left.\begin{aligned} \pi_{0i} &= \gamma_{00} + \gamma_{01} * age_{i1} + \gamma_{02} * age_{i1}^{\ 2} + \gamma_{03} * SES_{i1} + \xi_{0i} \\ \pi_{1i} &= \gamma_{10} + \gamma_{11} * age_{i1} + \gamma_{12} * age_{i1}^{\ 2} + \gamma_{13} * SES_{i1} + \xi_{1i} \end{aligned}\right\} \text{Level-two Model}$$

here Y_{ij} is the health for individual i measured at time j, $Time_{ij}$ is the passage of year since first wave, which is a level one predictor. π_{0i} is the health of individual i at the beginning of the ACL study; π_{1i} is the slope (or the rate of change) associated with time; SES_{i1} is the socioeconomic status of individual i at the beginning of the study and age_{i1} is the age of individual i at the beginning of the study. SES_{i1} and age_{i1} are level two predictors. ε_{ij}, ξ_{0i}, and ξ_{1i} are variations associated with level one and level two models.

This model suggests that age cohorts and SES groups have different health at the beginning of the study; moreover, the rate of health change over time is different among

them. There are advantages associated with the above model: 1) it takes into account the effect of time, which has been largely excluded from cross-sectional data analyses, 2) the effects of independent variables (which are SES and age) on the initial health and the rate of change in health over time are taken into account, 3) it posits that people from different SES may have different health when the ACL study began and they may also have different slopes in health change over the 8-year study period, and 4) It could be easily expanded to include other time variant or invariant variables, such as race, employment status, health risk behaviors etc., in models to analyze the trajectory of health.

Multi Cohorts Accelerated Longitudinal Design (MCALD)

The above model mainly explores health trajectories of age cohorts over the 8-year study period. However, it is debatable that different health trajectories among different age cohorts are actually due to genetic differences among them. "Presumably, if the analytic model explicitly incorporated all effects of demography and history, cohort differences in age-related change would be null" (Miyazaki and Raudenbush 2000:45).

Therefore, it is likely that human beings share a common mean-age health trajectory over the life course; however, an individual health trajectory may vary around the common mean-age trajectory due to different personal, social, and environmental factors. Thus, it is more valuable to explore the common health trajectories over the life course (Miyazaki and Raudenbush, 2000) and explore mechanisms through which personal and social factors affect health trajectories.

The Multi Cohorts Accelerated Longitudinal Design (MCALD) (Tonry, Ohlin, and Farrington 1991) can reasonably approximate age-related change over a longer period of time based on data collected over a shorter time interval (Miyazaki and Raudenbush 2000). The development of an individual health is tracked by repeatedly measuring his/her health status at different study periods in a longitudinal study. Each age cohort has a common mean-age health trajectory over the study period. If the health trajectories of two adjacent cohorts overlap over the common age intervals, it is said that the two trajectories converge (Miyazaki and Raudenbush 2000). In other words, the two cohorts are said to share common health trajectory over the age range.

In ACL, age at first interview varies from 25 to 95 years old. People of same age are considered as the same cohort. Thus, there are 71 age cohorts in ACL. Each cohort has a health trajectory over the 8-year study period. The age 25 cohort (cohort 1) ages from 25 to 33 during the 8-year study. The age 26 cohort (cohort 2) ages from 26 to 34. The age 27 cohort (cohort 3) ages from 27 to 35…The age 95 cohort (cohort 71) ages from 95 to 103. Adjacent age cohorts have a number of years of overlap in the 8-year study period. For example, the age 25 cohort share the same age interval with the age 26 cohort from age 26 to 33. If the health trajectories of the two age cohorts converge in the age 26 to 33 interval, we will say that they share a health trajectory over the age range of 25 to 33. This step could be repeated until a single common health trajectory over the life course (from age 25 to 103) is generated.

The relationship between age cohorts and health at the beginning of the study suggests a quadratic shape between age cohort and health. If different age cohorts have

different health trajectories over the life course, the health model would be formulated as the following equation:

$$Y_{ij} = \pi_{0i} + \pi_{1i} * (Age_{ij} - 25) + \pi_{2i} * (Age_{ij} - 25)^2 + \varepsilon_{ij} \longrightarrow \text{Level-one Model}$$

$$\left.\begin{aligned} \pi_{0i} &= \gamma_{00} + \sum_{k=2}^{71} \gamma_{0k} * C_{ik} + \xi_{0i} \\ \pi_{1i} &= \gamma_{10} + \sum_{k=2}^{71} \gamma_{1k} * C_{ik} + \xi_{1i} \\ \pi_{2i} &= \gamma_{20} + \sum_{k=2}^{71} \gamma_{2k} * C_{ik} + \xi_{2i} \end{aligned}\right\} \text{ Level-two Model}$$

here i refers to respondents, it varies from 1 to 3617; j indicates age spanning from 25 to 103; k varies from 1 to 71, which stands for 71 age cohorts. π_{0i} is the health of individual i at age 25; π_{1i} is the instant rate of change associated with age; π_{2i} is the acceleration rate of change with age. C_{ik} is the cohort to which individual i belongs. γ_{00} is the expected health for persons in cohort 1 (the age 25 cohort) at age 25. γ_{0k} is the mean difference in health between cohort (k+1) and cohort 1 at age 25. γ_{10} is the expected instant rate of change in health of cohort 1; γ_{1k} is the average difference in the instant rate of change between cohort (k+1) and cohort 1. γ_{20} is the expected acceleration in the rate of change of cohort 1; γ_{2k} is the average difference in the acceleration rate of change between cohort (k+1) and cohort 1. ε_{ij}, ξ_{0i}, ξ_{1i}, and ξ_{2i} are variations associated with level one and level two models.

The above model suggests that different cohorts have different health trajectories over the life course. However, a preliminary analysis (not shown here) applying the above model identifies that γ_{1k} and γ_{2k} are not statistically significant, which suggests

no cohort effect in health trajectory over the life course. This suggests that different health trajectories among different cohorts may be largely due to random variations around a common mean-age trajectory. In other words, there is a latent common health trajectory, around which individuals' health trajectories may vary due to their different personal, social, and environmental characteristics.

Correspondingly, a reduced model that excludes cohort effects is specified as the following equation (**Equation 2**):

$$Y_{ij} = \pi_{0i} + \pi_{1i} * (Age_{ij} - 25) + \pi_{2i} * (Age_{ij} - 25)^2 + \varepsilon_{ij} \longrightarrow \text{Level-one Model}$$

$$\left.\begin{aligned} \pi_{0i} &= \gamma_{00} + \xi_{0i} \\ \pi_{1i} &= \gamma_{10} \\ \pi_{2i} &= \gamma_{20} \end{aligned}\right\} \text{Level-two Model}$$

here γ_{00} is the average health of population at age 25; γ_{10} is the average instant rate of change as age increases by one year; γ_{20} is the average rate of acceleration in the instant rate of change. The $\pi_{1i} * (Age_{ij} - 25)$ and $\pi_{2i} * (Age_{ij} - 25)^2$ terms allow the intercept to be defined as the model-implied value of health at age 25. π_{1i} and π_{2i} are fixed at population average level. The random effects associated with $(Age_{ij} - 25)$ and $(Age_{ij} - 25)^2$ are constrained to zero.

This model suggests that people, from different age cohorts, share a common health trajectory over the life course; however, individuals' actual health trajectories may vary around the common one. The above model also suggests a curvilinear relationship between age and health. The main difference between Equation 1 and Equation 2 is the level-one model predictor. In Equation 1, the passage of year since first wave is the level

one predictor. It mainly focuses on health trajectory over the 8-year study period; however, Equation 2 uses age at each study wave as the level-one predictor, which links the health to a specific age such that the relationship between health and age actually represents the effect of aging process.

My study utilizes the model (indicated by Equation 2) to explore the role of SES in health trajectories over the life course. It explores the mechanisms through which SES channels individuals into different health trajectories and the possible mediators linking SES to health over the life course.

In summary, the MLMFC (using the passage of years since first wave) and Multi Cohorts Accelerated Longitudinal Design (MCALD) will be applied in parallel. By doing so, I am able to explore the landscape of health trajectory during the 8-year study period and the corresponding responsible factors. More importantly, I am able to explore health trajectory over the life course based on information collected during 8-year study period of ACL.

CHAPTER 4: RESULTS

This part of study provides core findings about SES disparities in health over the life course. The MLMFC and MCALD are applied. Functional limitation, the number of chronic diseases, and the combination of number and duration of chronic diseases as measurements of health are utilized in parallel models.

Chapter 4-I provides descriptive analysis of variables used in this study, which offers a general idea about the distribution of variables. Chapter 4-II-1 utilizes functional limitation as a measurement of health, and explores SES disparities in health during the 8-year study interval of ACL. Chapter 4-II-2 expands the findings from chapter 4-II-1 to explore SES disparities in health over the life course by applying the MCALD model. Chapter 4-II-3 is a summary of findings in chapter 4-II-1 and 4-II-2.

Chapter 4-II-4 uses the number of chronic diseases as a measurement of health, and explores SES disparities in health over the 8-year study interval in ACL. This chapter provides empirical findings against using the number of chronic diseases as a measurement of health over time. Chapter 4-II-5 applies the combination of number and duration of chronic diseases as a measurement of health over time. The same models in chapter 4-II-4 are applied using the combination of number and duration of chronic diseases as the dependent variable. Results of this chapter suggest that the combination measurement is better in measuring health over time than the count measurement in a longitudinal study. Chapter 4-II-6, by applying the MCALD, expands the findings from chapter 4-II-5 to explore health trajectory over the life course (instead of only 8-year study period).

Chapter 4-I: Descriptive Analysis

Functional Limitation

As illustrated in Table 4.1 on page 70, at the beginning of the ACL study (wave 1, 1986), 70.97% respondents said they had no functional limitation, 15.43% reported some functional limitation, 8.57% reported moderately severe functional limitation, and only 5.03% reported most severe functional limitation. At wave 2 (in 1989), 71.26% of respondents reported no functional limitation, 15.66% reported some functional limitation, 7.39% reported moderately severe functional limitation, and 5.69% reported most severe functional limitation. At wave 3 (in 1994), 69% of respondents reported no functional limitation, 13.32% reported some functional limitation, 8.28% reported moderately severe functional limitation, and 9.41% reported most severe functional limitation.

Chronic Diseases

The distributions of chronic diseases at the three waves are shown in table 4.1: At wave 1, 32.21% respondents reported experiencing *hypertension* during the last 12 months**,** 4.46% reported experiencing a *lung disease* during the last 12 months, 8.85% reported experiencing *heart attack* during the last 12 months, 9.38% reported experiencing *diabetes* during the last 12 months**,** 2.49% reported experiencing *cancer* during the last 12 months**,** and 0.86% reported experiencing a *stroke* during the last 12 months.

At wave 2, 31.04% respondents reported experiencing *hypertension* during the last 12 months**,** 5.59% reported experiencing *lung disease* during the last 12 months, 8.8%

reported experiencing *heart attack* during the last 12 months, 9.78% reported experiencing *diabetes* during the last 12 months, 2.65% reported experiencing *cancer* during the last 12 months, and 1.89% reported experiencing a *stroke* during the last 12 months.

At wave 3, 35.21% respondents reported experiencing *hypertension* during the last 12 months**,** 6.42% reported experiencing a *lung disease* during the last 12 months, 9.38% reported experiencing *heart attack* during the last 12 months, 10.29% reported experiencing *diabetes* during the last 12 months, 4.34% reported experiencing *cancer* during the last 12 months, and 3.36% reported experiencing a *stroke* during the last 12 months.

Demographic variables

Age: the mean age at first wave was 54.24 with a standard deviation of 17.67, the mean age at second wave was 56.21 with a standard deviation of 17.13, and the mean age at third wave was 59.58 with a standard deviation of 16.68.

Sex: 62.46% of respondents were female at first wave; it was 63.83% and 63.9% at second and third wave respectively.

Race: 64.22% respondents were White at first wave; it was 66.48% and 68.38% at second and third wave respectively. 32.46% respondents were Black at first wave; it was 30.48 and 28.84% at second and third wave respectively. 1.3% respondents were American Indian; it was 1.4% and 1.29% at second and third wave respectively. 0.83% respondents were Asian at first wave; it was 0.73% and 0.78% at second and third wave

respectively. 1.19% respondents were Hispanic at first wave; it was 0.91% and 0.7% at second and third wave respectively.

Employment status: at the beginning of the ACL study, 47.55% respondents were employed, 6.03% were unemployed, 26.1% were retired, and 26.1% were other.

Socioeconomic variables

Education: 20.07% respondents at first wave had an elementary school degree; 46.36% respondents had a high school degree and 33.56% respondents had a college and above degree.

Family Income: At wave 1, 32.51% respondents were in a low income group; 40.78% respondents were in a mid income group; and 26.71% were in a high income group. At second wave, 28.88% respondents were in a low income group; 39.1% respondents were in a mid income group; and 32.02 were in a high income group. At third wave, 27.09% respondents were in a low income group; 34.35% respondents were in a mid income group and 38.56% respondents were in a high income group.

Health risk behaviors

Smoking: 29.31% respondents said they currently smoked at first wave. The percentage was 24.49 and 20.02 at second and third wave respectively.

Drinking: At first wave, 50.79% respondents said they didn't drink, 43.68% respondents were moderate drinker and 5.53% were heavy drinker. At second wave, the percentages are 44 .21, 43.19 and 2.6 correspondingly. At third wave, the percentages are 51.11, 45.86 and 3.04 correspondingly.

Sleeping: At first wave, 49.41% respondents said that they slept less than 7 hours per day, 33.87% said they slept between 7 and 9 hours a day, and 16.73% said they slept more than 9 hours. At second wave, the percentages are 53.41, 29.87 and 16.72 correspondingly. Since no questions about sleeping hours were asked at third wave, information about sleeping hours at third wave was not available.

Body mass index: At first wave, 5.14% respondents were under weighted, 76.09% were normal weighted and 18.77% were over weighted. At second wave, the corresponding percentages are 4.2, 75.26 and 20.54. At third wave, the corresponding percentages were 4.33, 72.21 and 23.46.

Physical activity: The mean physical activity index at first wave was -0.19 with a standard deviation of 1.06. At second wave, mean physical activity index was -0.33 with a standard deviation of 1.02. At third wave, mean physical activity index was -0.03 with a standard deviation of 1.01.

Indicator of Stress

Financial chronic stress: The mean financial stress index at first wave was 0.091 with a standard deviation of 1.071; it was -0.041 at second wave with a standard deviation of 1.001; it was -0.086 at third wave with a standard deviation of 0.996.

[TABLE 4.1 ABOUT HERE]

Table 4.1: Descriptive Analysis of Variables

	Wave 1	Wave 2	Wave 3
	mean (std)	mean (std)	mean (std)
Funcional Limitation			
No limitation	70.97	71.26	69
Some limitation	15.43	15.66	13.32
Moderate limitation	8.57	7.39	8.28
Severe limitation	5.03	5.69	9.41
Chronic Diseases			
Hypertension	32.21	31.04	35.21
Lung disease	4.46	5.59	6.42
Heart attack	8.85	8.8	9.38
Diabetes	9.38	9.78	10.29
Cancer	2.49	2.65	4.34
Stroke	0.86	1.89	3.36
Financial stress	0.091	-0.041	-0.086
	(1.04)	(0.97)	(0.98)
Age	54.24	56.21	59.58
	(17.67)	(17.13)	(16.68)
Sex (1=female)	62.46	63.83	63.9 (.50)
	(0.5)	(.50)	
Race			
White	64.22	66.48	68.38
Black	32.46	30.48	28.84
American Indian	1.3	1.4	1.29
Asian	0.83	0.73	0.78
Hispanic	1.19	0.91	0.7
Education at wave 1			
Elementary school	20.07		
High school	46.36		
College	33.56		
Family income			
Low income	32.51	28.88	27.09
Mid income	40.78	39.1	34.35
High income	26.71	32.02	38.56
Health behavior (wave 1)			
Smoking	29.31	24.49	20.02
Drinking			
Nondrinker	50.79	44.21	51.11
Moderate drinker	43.68	53.19	45.86
Heavy drinker	5.53	2.6	3.04
Sleep hour			
Less than 8 hours	49.41	53.41	
Equals to 8 hours	33.87	29.87	
Greater than 8 hours	16.73	16.72	
Body Mass Index			
Under weight	5.14	4.2	4.33
Normal weight	76.09	75.26	72.21
Over weight	18.77	20.54	23.46
Physical activity	-0.19	-0.33	-0.03
	(1.06)	(1.02)	(1.01)
Emolyment status			
Employed	47.55		
Unemployed	6.03		
Retired	26.1		
Other	20.32		

Chapter 4-II: Results from MLMFC and MCALD

The rest parts of this study focus on results from statistical models. Two major components are covered: the first component focuses on functional limitation as a measurement of health. This part applies the Multi Level Model for Change (using the passage of years since first wave in the ACL study as a measurement of time) to explore "Age, SES and Health Trajectory" over the 8-year study period and utilizes the Multi Cohorts Accelerated Longitudinal Design to explore "Age, SES and Health Trajectory" over the life course based on the 8-year data set of ACL.

The second component focuses on chronic diseases as a measurement of health. The Multi level model for change (using the passage of years since first wave as a measurement of time) and Multi Cohorts Accelerated Longitudinal Design are applied. Two types of measurements of health (The number of chronic diseases, combination of number and duration of chronic diseases) are used as dependent variable to the same models.

Functional Limitation as a Measurement of Health

Function limitation is a major component of health, which has attracted significant attention from researchers. This part of study will explore how health (measured by functional limitation) is associated with Age and SES from the longitudinal perspective.

I break down this part into two chapters: Chapter 4-II-1 explores the relationship of health trajectories to age cohorts and SES during the 8-year study period. This chapter aims to identify SES disparities (if any) in health trajectories over time and to examine if health risk behaviors mediate the relationship between SES and health trajectories. In this chapter, the passage of years since first interview is used as a measurement of time. The effect of age at first wave is considered as cohort effect. Equation 1 from Chapter 3-IV is used as the base model in this chapter.

Chapter 4-II-2 expands the findings from chapter 4-II-1 in that the Multi Cohorts Accelerated Longitudinal Design (MCALD) is used to explore health trajectory over the life course (not just over the 8-year study period). The MCALD assumes that people share a common health trajectory over the life course; however, individuals' health trajectories may vary around this common one due to the differences in their social, psychological, environmental, and individual characteristics. Equation 2 in chapter 3-IV is applied in this chapter.

1. Age, SES and Functional Limitation in the 8-year study period of ACL

Coefficients describing the growth curve in physical health are shown in table 4.2 on page 96. The Multi Level Model for Change (using the passage of years since first wave) allows us to explore the hypotheses that individuals are differential in their physical health at the beginning of the ACL study (wave I in 1986), and that they may also experience different rates of change in their physical health over time. Table 4.2 presents the effect of age, SES, health risk behaviors (smoking, drinking, overweight, and physical activity), financial stress, and control variables on the initial physical health at the beginning of the study and the rate of change over the 8-year study period.

Results are based on mixed-effects models, which have been employed by researchers to analyze longitudinal data sets (Singer and Willet 2003). The models in this table are mainly to explore people's physical health trajectories over the 8-year study period, and to identify factors responsible for SES disparities in health over time.

Model (1) is an unconditional growth model, in which time is the only predictor of physical health. The intercept in the initial status is statistically significant, which means that on average, people's health is somewhat between "no functional impairment' and "least severe level" at the beginning of the study. The intercept in the rate of change is significant, which means that individuals' physical health significantly decline as time goes by. The significant variances in the initial status (0.4632***) and the rate of change (0.0063***) indicate that people's health at the beginning of the study are different, and the rate of change over time among them varies as well. This is mostly reflecting the reality in that individuals (especially from different age cohorts) may have different

health at any given point of time, and they may also have different rate of decline in health over time. However, the health at the beginning of the study does not correlate with the rate of change as indicated by the insignificant covariance between the initial status and rate of change. In other words, health at the beginning of the study does not significantly predict the rate of change in health over time. In summary, results from Model (1) suggest that people's health trajectories over the 8-year interval in ACL are different.

The unconditional growth model provides significant variation in the initial status and rate of change, for which level-2 predictors are responsible. The following part of this chapter is devoted to explore the predictors (or factors) that explain the different health trajectories among people.

Age cohort and health trajectories

Model (2) through Model (10) in table 4.2 provides stepwise mixed-effects regression results. Age (centered at age 25), age square, SES, health risk behaviors, control variables, and various combinations, are treated as predictors of the initial status and rate of change. These models consistently indicate curvilinear relationships between age cohorts and health at both the initial status and rate of change in the 8-year study interval of ACL study.

Age cohort and health at the beginning of the study

The curvilinear relationship between age and health at the beginning of the study (model 2 through model 10 in table 4.2) indicates that older age cohorts' health is better off than younger ones until the age cohort when health reaches the healthiest point; after

that age cohort point, older cohorts are worse off than younger ones (This conclusion is based on negative first derivative and positive second derivative). However, the age cohort, where health is the best varies in different models when other variables are controlled. For example, the healthiest point in model (2) is at age 51.52; however, the healthiest point in model (3) is at age 45.25, the one in model (4) is at age 54.75, and the one in model (10) is at age 56.48. This suggests that age is not the only predictor for health; other factors may also impact health such that the age at the healthiest point may vary across different models.

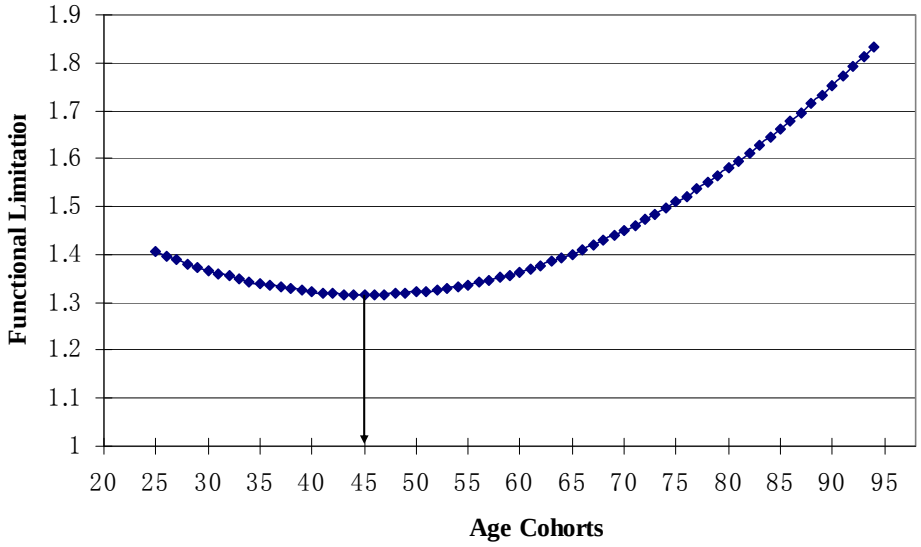
Age cohort is consistently significant in predicting health at the beginning of the study across models; however, SES, health risk behaviors, and control variables mediate part of its effect. The results suggest that health may not be simply due to the biological process of aging; social, environmental, and individual characteristics (such as SES, health risk behaviors, etc.) are part of the determinants of the aging process.

The relationship between age cohorts and health at the beginning of the study is illustrated in Figure 4.1 on page 78. This figure, which is based on model (3), is used for illustrative purposes. It provides an efficient way to demonstrate the curvilinear relationship between age cohort and health at the beginning of the study.

Figure 4.1 shows that before age 45 cohort, the older age cohorts are better off than the younger ones in term of functional limitation. However, the gap diminishes beyond the point (as shown positive coefficient in age square) such that the older age cohorts are worse off than the younger ones after age 45.25.

Does the relationship between age cohorts and health at the beginning of the study mimic the trajectory of health over the life course? That is, does the age cohort effect on health actually reflect the effect of age in a life course? If the answer is yes, then health trajectory over the life course could be described as follows: People's health improves as they age until they reach the healthiest point; after that point, people's health starts to decline. However, as shown in the above models, the healthiest point in life is largely affected by the combination of age, social, and biological factors such that aging and health is stratified (House et al. 1994, 2005). The relationship between age cohorts and health at the beginning of the study provides a base for further exploration of age and health over time. The Multi Cohorts Accelerated Longitudinal Design (MCALD) (Tonry, Ohlin, and Farrington 1991) offers a valid inference about the relationship between the health and age over the life course based on a study of short duration with multi cohorts (Miyazaki and Raudenbush 2000). The description of the MCALD is provided in chapter 3-IV. The results based on the MCALD are presented on Chapter 4-II-2.

Figure 4.1: Age Cohorts and Initial Functional Limitation



Age cohort and health at the rate of change

Coefficients in the rate of change provide information about health trajectory in the 8-year study interval and indicate factors associated with the rate of change. In the unconditional growth model (model 1 in table 4.2 on page 97), the intercept is positive, which suggests that, in general, people's health declines as time goes by; however, the declining rates are not unique, and may vary among individuals (which is indicated by the significant variance in the rate of change).

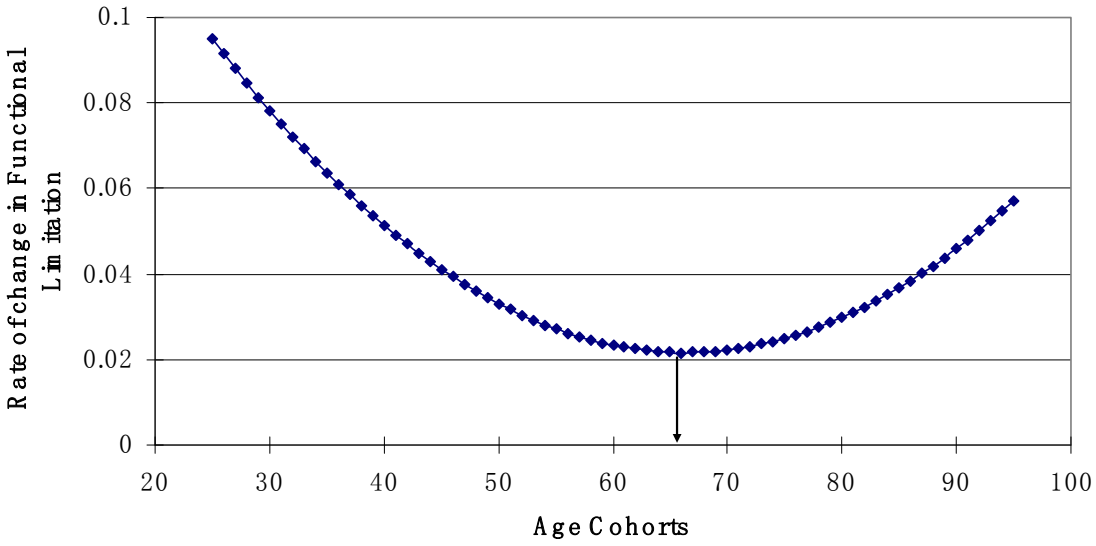
Models (2) through (10) in table 4.2 include age cohort, age cohort square, SES, health risk behaviors, control variables, and various combinations of these variables as predictors of the rate of change in health. Coefficients of age cohort and age cohort square on the slope continue to be significant, which indicates a U-shape relationship between age cohort and the rate of change in physical health. Similar to the results at the initial status, the flat point where the rate of change becomes zero varies in different models. The effect of age cohort on the rate of change is partially mediated by SES. Income mediates the effect of age cohort more than that of education. Results from model (2) through model (10) indicate that age is not the only factor that determines the rate of health change, SES, health risk behaviors, and control variables mediate part of its effect. Variances in the rate of change are consistently significant across models, which means that there are other factors (besides those including in the models) remaining to be identified as predictors of health.

Figure 4.2 on page 81 is based on model (3). The figure is only for illustrative purposes. In this model, the point when the slope equals zero is at age 66.27. The main

point of the figure is that the rate of health change (or decline) is not constant; there is a curve such that the rate of change declines as age increases until it reaches the point where the rate of change becomes zero; after that point the rate of change increases as age increases. Therefore, the later a person reaches the age point where the rate of change is flat, the longer he/she enjoys a healthy life.

As indicated by Figure 4.2, the relationship between the rate of change in health and age cohorts is curvilinear. Before the age 66.27 cohort, older age cohorts have low decline rate compared to younger cohorts; however, after that point, there is a turn over such that older age cohorts have higher decline rate in health.

Figure 4.2: Age Cohorts and the Rate of Change in Functional Limitation



SES and Health Trajectory

It is well documented that SES is positively associated with health (Bowling 2004; House et al. 1994, 2005; Preston and Taubman 1994; Siegrist and Marmot 2006). However, less attention has been focused on how SES impacts health trajectory in a life course. Are lower SES people consistently worse off than higher SES ones across the whole life? In other words, is the relationship between SES and health linear over time? Do different SES groups age at the same rate? Does different life expectancy among various SES groups indicate different aging processes among them? Empirical research does not provide much information about the rate of aging across different SES groups. This study makes significant contributions to previous research in that it identifies different rates of change in health over time across different SES groups. People from different SES groups age differently, they are largely "segregated" into different life trajectories by their SES status. Lower SES groups not only have worse health at a given point of time (here at the beginning of the ACL study), they also have a higher rate of change in health over time. In short, lower SES people suffer poor health, their body function decline faster than higher SES people as well. The aging process in the United States is segregated according to how much people earn, and how many years of education they have had.

Results from table 4.2 empirically support the above statement. Model (3) and model (4) include income (recoded as low income, mid income, and high income) and education at wave 1 separately as a predictor of the initial status and rate of change. These models are used to test the effects of income and education on both the initial status and rate of

change. Model (3) indicates that income is negatively related to functional limitation at the beginning of the study in that people with lower income have a higher level of functional limitation. Moreover, rates of health change over time are also different across different income groups: lower income groups decline faster than higher income ones do (the coefficient is negative and significant at $p<0.01$ both for middle and high income groups). Model (4) includes education as a predictor of health at the beginning of the study and rate of change. The results are similar to the effect of income in that the higher education level a person has, the lower functional limitation he/she will likely have at the beginning of the study (in 1986). However, only people with college degree have a significant lower rate of change than people with elementary school. There is no significant difference between high school and elementary school in the rate of change in health.

The above two models assume that the effect of income is constant across different education levels and verse visa. However, it is reasonable to suppose that income's effect on health may vary across different education levels. Model (7) includes an interaction term between income and education as a predictor of health. This model is used to answer questions such as: is the effect of income on health constant across different education levels? Or does the effect of income depend on education levels?

In order to better interpret the coefficient of the interaction between education and income, I use continuous income (instead of categorized income) in this model. Model (7) identifies an interaction effect between income and education both at the initial status and

rate of change. However, the direction of the effect at the initial status and rate of change are opposite.

At the initial status, as income increases by $10,000, people with an elementary school degree will have 15.6% improvement in functional health; however, the same amount of increase in income will only improve the functional health of people with a high school degree by 8% and that of people with a college degree by 2.7%. It is obvious that the effect of income on health diminishes as educational level increases. Thus, it suggests that income is more important for lower educated people for their health. The same amount of increase in income will benefit lower educated individuals more than it does to higher educated ones. At the same income level, people with higher education levels are better off than those with lower education levels. However, the return from increased education diminishes as income increases. For example, at $10,000 income level, people with a high school degree will be 20.2% better than those with an elementary degree, people with a college degree will be 29.3% better than those with an elementary degree. At income level of $30,000, people with a high school degree will be 5% better than those with an elementary degree, people with a college degree will be 3.5% better than those with an elementary degree. At income level of $50,000 and above, people with an elementary degree are actually better off than those with a high school and college degree. However, $50,000 was a very high income in 1986. People with an elementary degree and earning $50,000 or more per year are not representative. They could be considered as outliers. The following cross-tab of education level and income in 1986 indicates that only 4.6% respondents with an elementary degree earn $50,000 or

more in 1986. However, it is 47.2% among people with a college and over degree that earn $50,000 or more. It is obvious that education is positively related to income. Therefore, we could conclude that generally people's health is better off as income increases; however, the effect diminishes as education increases.

Education Level and Income in 1986

Education	Low Income	Middle Income	High Income
Elementary	68.5%	26.9%	4.6%
High School	32.2%	46.3%	21.5%
College & over	11.2%	41.6%	47.2%

Model (8) adds financial stress into model (7). The coefficient is positively significant, which suggests that high financial stress is associated with worse health. The effect of income and education actually declines when financial stress is controlled. This suggests that part of the SES disparities in health is due to the financial stress faced by low income and low-educated groups.

The effect of income and education in the rate of change reveals that people with higher education levels will have slower rates of decline in health as income increases. And income enhances the effect of education in that the higher education a person has had, the lower decline rate in health will be as income increases. Thus, the interaction between education and income in the rate of change is opposite to that in the initial status. Income effect doesn't diminish as education increases; it instead amplifies the effect of education on the rate of change.

An interesting finding in the rate of change is that people with an elementary degree will have a higher rate of decline as their income increase. In other words, people with an

elementary degree will age faster as their income increase. This finding is contrary to common sense. In general, income benefits health; therefore, it is reasonable to believe that people's rate of decline will be slowed down if their income increase. However, this may just be one side of the story. We should also explore the other side of the story, which is the way a person (especially a low-educated person) uses to increase his/her income. Since the job market is highly segregated according to education levels, low-educated people are more likely to be channeled to a low paid, poor-working-environment job market. It is hard for them to find a high paid job. Increasing income by $10,000 may be much harder for them. In order to increase their income, they may have to work longer, to have multiple jobs. In other words, low-educated people may sacrifice their health to increase income, which actually harm their health in a long run. Therefore, health of low-educated people decline faster as their income increase.

For high-educated people, however, they are in a better shape in the job market. They have well-paid jobs, good benefits, and better working environments. They could enjoy the benefits brought by increased income without sacrificing their health.

Financial stress is positively associated with the rate of change in health. People facing a higher level of financial stress will be aging faster. However, financial stress does not mediate the effect of SES in the rate of change, which suggests that the effects of SES and financial stress are cumulative in the rate of change.

In summary, education and income interact to impact people's health trajectories. For people with lower education, income is an important source impacting people's health. However, for higher educated people, income effect is less important.

The above findings about the interaction between education and income support the resource substitute theory (Mirowsky and Ross 2005) in that one resource can substitute for another such that the less a group has of one resource, the more important another will be. In part this could be due to diminishing returns of any one resource, such that gains in a resource at low levels have more of an impact than gains at high levels.

Age Cohort, SES and Health Trajectory

House et al. (1994) argue that SES disparity in health is largest at middle age, while it diminishes until later old age due to physical fragility of human beings and the declining effects of SES. If the statement is true, there must be an interactive effect between age and SES on health trajectory in that SES disparity becomes larger and larger until it reaches the peak. After the peak point it starts to diminish.

Model (5) includes age, education, and the interaction term of them. The interaction term is neither significant at the initial status nor at the rate of change. The insignificant interaction term of age and education at the initial status indicates that the effect of education on health is constant across different age cohorts at the beginning of the study. In the rate of change, the effect of education does not interact with age either. Results from model 5 suggest that across all age cohorts, higher educated people have slower rates of change in health than lower educated people. Correspondingly, they age at a slower speed.

Model (6) indicates that there is an interactive effect between age cohort and income at the beginning of the study. The relationship between age cohort and health for low income people is linear (the coefficient associated with age is significant; however, the

one associated with age square is not). That is, among low income people, older age cohorts' health is consistently worse than younger cohorts. However, for mid income and high income groups, the relationships between age cohort and health are curvilinear in that older cohorts are healthier than younger ones before the age point with the healthiest status, after that age point, older cohorts are worse than younger ones in health.

Findings from model (6) lead to examine cohort effect versus age effect. If cohort effect on health at the initial status actually reflects the effect of age, results from model (6) will provide strong supports for the statement that the largest SES disparity in health in middle age is largely due to the postponed decline age for higher SES people. In other words, the health decline of higher SES people is compressed to the very late of life, while the health of low SES people starts to decline at early age and at faster rates. Results from models using age as a measurement of time support the statement that cohort effect at the initial status actually reflects the effect of age (Next Chapter provides detail explanations).

The effect of interaction between age and income, however, is not significant at the rate of change, which indicates that income's effect on health is constant across age cohorts in the rate of change.

The results from the initial status and rate of change models suggest that, in general, low SES people not only suffer more than higher SES ones, they also age faster. It is like a double jeopardy for low SES people in the process of aging.

Health risk behaviors and health trajectory

Previous researchers link SES disparities in health to the disproportional distribution of health risk behaviors among low SES people. They (e.g. House et al. 1994) argue that SES disparity in health over the life course is largely due to the different exposure to risk factors over the life course.

Model (9) and (10) in table 4.2 (continued) on page 97 explore the effect of health risk behaviors on health trajectory after controlling SES, race, and employment status. Results show that moderate drinking benefits health at the beginning of the study compared to no drinking at all; however, dinking status doesn't significantly impact the rate of change in health over time. Contrary to previous research, heavy drinking people don't have worse health than those drinking moderately. One possible reason for this finding may be due to the way that researchers categorize people into moderate or heavy drinking. In ACL, the total cans of beer, glasses of wine, and drinks of liquor were summed up as the total number of drinks in last month. Therefore, the same number of total drinks may not mean the same thing to different people due to different alcohol tolerance levels. For example, the same number of beer may have less effect on health than that of liquor. Thus, using the number of drinks as a measurement of heavy drinker may not be optimal.

Both under weight and over weight significantly harm health at the beginning for the study compared to normal weight. In a long run, overweight people's health decline faster than people with normal weight as time goes by. However, underweight people

don't significantly decline faster than normal weight ones. It is obvious that over weight significantly deteriorates health over time.

Physical activity benefits people's health. People having high levels of physical activity are better off at the beginning of the study than those having low levels of physical activity. However, physical activity does not significantly impact the rate of health change over time. One possible reason may be due to the fact that physical activity is treated as time invariant in this model. As we know that people may change their lifestyle over time, those with low levels of physical activity may exercise more in the following study points than they do at the beginning of the study, which in turn benefits their health. For those having high levels of physical activity, they may exercise less in the following study points than they do at the beginning of the study, which in turn harms their health. Treating physical activity as a time invariant variable could not reflect changes over time, which may actually cancel out the effect of physical activity.

Smoking doesn't significantly affect people's health at the beginning of the study; however, a person who smokes has a higher rate of change in health over time. This result indicates that the effect of smoking is accumulative, the longer a person smokes, the faster his/her health will decline.

People sleeping less than 7 hours or more than 9 hours have worse health at the beginning of the study than those sleeping between 7 and 8 hours. However, there is no significant difference in the rate of change on health among them.

In summary, major risk behaviors (no drinking, under or over weight, less physical activity, and sleeping less than 7 hours or more than 9 hours) negatively affect health.

Health risk behaviors together explain 46% of income effect on health at the beginning of the study among people with an elementary degree (based on the comparison between model (9) and model (10)). This result supports previous research in that SES disparity in health is largely due to different exposure of different SES groups to health risk behaviors.

Health risk behaviors (except smoking and over weight) do not have significant effect on the rate of change. This result leads to the need to explore the effect of time variant health risk behaviors on health trajectory, which assumes that people may change their health risk behaviors over time and such change may lead to corresponding change in their health.

[TABLE 4.2 ABOUT HERE]

[TABLE 4.2 (Continued) ABOUT HERE]

Time Variant SES (measured by income) and Health

Models in table 4.2 treat income as a time invariant variable, which assumes that people's SES status is static across time. However, it is reasonable to suppose that people's income may change over time, therefore, their SES status are likely to change as well. Treating income as a time variant variable helps to explore how health trajectory varies corresponding to income trajectory over time.

The model could be presented by the following equation:

$$Y_{ij} = \pi_{0i} + \pi_{1i} * Time_{ij} + \pi_{2i} * Income_{ij} + \varepsilon_{ij}$$

$$\pi_{0i} = \gamma_{00} + \upsilon_{0i}$$
$$\pi_{1i} = \gamma_{10} + \upsilon_{1i}$$
$$\pi_{2i} = \gamma_{20}$$

here i refers to respondents, varying from 1 to 3617, j varies from 1 to 3, indicating study waves in ACL. This model invokes a constraint on income such that the effect of income is constant across population. However, this model allows the effect of time to vary randomly across individuals in the population. γ_{10} is the population average rate of change in health by controlling income; γ_{20} is the population average change in health over time as income increases by $10,000.

Table 4.3 on page 98 shows the results of mixed-effects regression of health on time, time variant income, the interaction term of time and income, financial stress, and health risk behaviors.

Model 1 is an unconditional growth model. The unconditional growth model indicates the average health at the beginning of the study (in 1986) and the average rate of change over the 8-year study period. Variance components indicate that people's health are different in 1986, and the rates of change in health are also different.

Model (2) includes income, and interaction term of income and time as level-one predictors. This model is used to test if the effect of increased income on health is moderated by time; or if the effect of time on health is moderated by income. The interaction term is positively significant at $p<0.05$ and the main effect of income is negatively significant at $p<0.001$. This indicates that income benefits health over time; however, the benefit diminishes as time goes by. Therefore, people's health trajectories could change correspondingly to their income change. If an individual's income increases, his/her health trajectories will be on a better health path correspondingly. If an individual's income decreases, his/her health trajectories will be on a worse path.

The above models assume that the effect of increased income is constant across people. However, it is likely that an increase in income benefits low SES groups more than it does to high SES groups. Thus, it leads to the need to include time invariant SES as level-two predictors of π_{2i}; however, it is beyond the scope of this study.

Model (3) adds financial stress. Results indicate that people's health trajectory will be on a worse path if their financial stress level increases. The interaction term between income and time become insignificant after controlling financial stress. This suggests that income will linearly benefit health over time if financial stress is constant.

Time variant health risk behaviors and health

In order to test the effect of time variant health risk behaviors, I include time variant variables into models. It can be described as the following:

$$Y_{ij} = \pi_{0i} + \pi_{1i} * Time_{ij} + \sum_{i=1}^{n}\sum_{j=1}^{3}\sum_{k=1}^{6}\pi_{2ki}X_{ijk} + \varepsilon_{ij}$$

$$\pi_{0i} = \gamma_{00} + \upsilon_{0i}$$

$$\pi_{1i} = \gamma_{10} + \upsilon_{1i}$$

$$\pi_{2ki} = \gamma_{2k0}$$

here i refers to respondents, which varies from 1 to 3617; j refers to study waves, which varies from 1 to 3; k refers to the number of health risk behaviors. This study mainly explores the effects of drinking, body mass index, physical activity, and smoking on health over time. The above model assumes that the effect of time will vary for different people. However, it constrains the effect of health risk behaviors to be constant across population.

The above model is used to explore the effects of time variant health risk behaviors on health over time. Simply put, this model answers such questions as: how will people's health trajectories change correspondingly if they change their health risk behaviors? Model (3) and (4) in table 4.3 indicates that people's health trajectories will change corresponding to the change of health risk behaviors: Moderate drinking benefits health over time than non-drinking, and such benefit amplifies with time. There is no significant change in health trajectory associated with a change from moderate drinking to heavy drinking.

Both underweight and overweight are associated with worse health over time, compared to normal weight. Moreover, the health decline of underweight people accelerates over time than normal weight people. However, the effect of overweight does not interact with time.

Physical activity, however, is beneficial to health over time, and such benefit amplifies as time goes by. The more physical activity a person is involved in, the healthier he/she will be over time. Smoking is harmful to health over time, and such harm accelerates with time.

The parameter estimate for time (γ_{10}) in model (4) suggests that the yearly rate of decline in health has been reduced by 41% compared to that in model (1) (decrease from .032 in model 3 to .019 in model (5)) after including time variant health risk behaviors. This suggests that health risk behaviors explain 41% of decline rate associated with time. Moreover, health risk behaviors also mediate the main effect of income by 21.1% (based on the comparison of model (5) and (3)).

The results in the above models show that health risk behaviors do impact health trajectory over time. Therefore, the insignificant effects of time invariant health risk behaviors on the rate of change in table 4.2 is mainly due to the inappropriate way of treating time variant variables as time invariant. Thus, it is optimal to treat time variant variables as the way they are in a longitudinal study.

[TABLE 4-3 ABOUT HERE]

Control Variables and Health

Race, employment status, and gender are considered as control variables in this study. Model (9) in table 4.2 (continued) on page 98 shows that the Black are not worse off at the beginning of the study than non-black; however, Black have a higher rate of change over time. This suggests that Black will have worse health as time goes by compared to non-Black. When controlling health risk behaviors in model (9), Black' health is actually better than non-Black at the beginning of the study, although Black continue to have a high rate of change in health over time. This result suggests that health risk behaviors are largely responsible for Black' worse health over time.

Employed are healthier than unemployed at the beginning of the study; however, there are no significant differences in the rate of change between them. Disable people have worse health both at the beginning of the study and the rate of change on health. Worse health among retired than unemployed at the beginning of the study may actually reflect the fragility of human beings at the later stage of life.

[TABLE 4.2 (continued) ABOUT HERE]

Table 4.2: Mixed-effects Regression of Physical Health on Age, Education, Income, Health Risk Behaviors and Interaction Terms

		Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
Fixed Effects									
Initial status (π_0)	Intercept	1.408***	1.286***	1.406***	1.639***	0.897*	0.423*	1.645***	1.489***
	(Age-25)/10		-0.167***	-0.088†	-0.179***	0.056	0.263**	-0.104*	-0.126*
	(Age-25) ^{2}/100		0.032***	0.022***	0.030***	0.012	-0.007	0.022***	0.027***
	Mid income[a]			-0.309***			1.079***		
	High income[a]			-0.388***			1.113***		
	High school[b]				-0.271***	0.438		-0.278***	-0.231***
	College[b]				-0.347***	0.576		-0.422***	-0.343***
	Income($10,000)							-0.156***	-0.104***
	Highsch*income							0.076*	0.059†
	College*income							0.129***	0.099**
	(Age-25)/10*midinc						-0.511***		
	(Age-25) ^{2}/100*midinc						0.043***		
	(Age-25)/10*highinc						-0.539***		
	(Age-25) ^{2}/100*highinc						0.044**		
	(Age-25)/10*highsch					-0.229			
	(Age-25) ^{2}/100*highsch					0.018			
	(Age-25)/10*college					-0.296			
	(Age-25) ^{2}/100*college					0.022			
	Financial stress								0.128***
Rate of change (π_{1i})	Intercept	0.029***	0.093***	0.095***	0.109***	0.158†	0.103*	0.099***	0.102***
	(Age-25)/10		-0.040***	-0.036***	-0.0402**	-0.052†	-0.037*	-0.038***	-0.039***
	(Age-25) ^{2}/100		0.005***	0.004***	0.005***	0.005*	0.004**	0.005***	0.005***
	Mid income			-0.012**			-0.016		
	High income			-0.015**			-0.053		
	High school[b]				-0.011	-0.052		-0.006	-0.006
	College[b]				-0.017*	-0.07		-0.007	-0.006
	Income($10,000)							0.004***	0.004***
	Highsch*income							-0.004***	-0.004***
	College*income							-0.006***	-0.006***
	(Age-25)/10*midinc						-0.002		
	(Age-25) ^{2}/100*midinc						0.004		
	(Age-25)/10*highinc						0.014		
	(Age-25) ^{2}/100*highinc						-0.001		
	(Age-25)/10*highsch					0.009			
	(Age-25) ^{2}/100*highsch					-0.0004			
	(Age-25)/10*college					0.012			
	(Age-25) ^{2}/100*college					-0.001			
	Financial stress								0.0003***
Variance Components:									
Level_1: within-person	σ_ε^2	0.2814***	0.282***	0.2819***	0.2821***	0.282***	0.282***	0.2821***	0.2818***
Level_2: In initial status	σ_{0}^2	0.4632***	0.3618***	0.3385***	0.3485***	0.3485***	0.3335***	0.3363***	0.3225***
In rate of change	σ_{1}^2	0.0063***	0.0057***	0.0057***	0.0057***	0.0057***	0.0057***	0.0057***	0.0057***
Covariance	σ_{01}^2	-0.0008	-0.0069***	-0.0079***	-0.00771***	-0.007***	-0.0078***	-0.0078***	-0.0079***
Deviance (-2 log likelihood)		21531.2	20613.6	20399.6	20490	20487.5	20362.5	20391.7	20278.6
Degree of freedom		6	10	14	14	22	22	20	22
AIC		21543.2	20633.6	20427.6	20518	20531.5	20406.5	20431.7	20322.6
BIC		21580.4	20695.5	20514.3	20604.7	20667.8	20542.7	20555.6	20458.8

Note:
a: low income is reference category
b: elementary school is reference category
† p<0.10; * p<0.05; ** p<0.01; *** p<0.001

Table 4.2 (Continued)

		Model 9	Model 10
Fixed Effects			
Initial status (π_{0i})	Intercept	1.630***	1.278***
	(Age-25)/10	-0.106*	-0.089†
	(Age-25) ^{2}/100	0.019***	0.017***
	High school[b]	-0.251***	-0.148**
	College[b]	-0.325***	-0.131*
	Income ($10,000)	-0.117***	-0.063*
	Hschool*income	0.063*	0.061*
	College*income	0.099**	0.076**
	No drink		0.132***
	Heavy drink		-0.057
	Under weight		0.157**
	Over weight		0.143***
	Physical activity		-0.207***
	Smoking		0.017
	Sleep<7 hours		0.067*
	Sleep>9 hours		0.168***
	Financial stress		0.105***
	Black	0.038	-0.073**
	Employed	-0.123*	-0.111*
	Retired	0.127*	0.162**
	Other	.267***	0.224***
	Female	0.016	-0.052*
Rate of change (π_{1i})	Intercept	0.090***	0.101***
	(Age-25)/10	-0.037***	-0.044***
	(Age-25) ^{2}/100	0.004***	0.005***
	High school[b]	-0.004	-0.004
	College[b]	-0.004	-0.006
	Income ($10,000)	0.005***	0.004***
	Hschool*income	-0.005***	-0.005***
	College*income	-0.006***	-0.006***
	No drink		-0.003
	Heavy drink		0.016
	Under weight		-0.014
	Over weight		0.011†
	Physical activity		0.004
	Smoking		0.013*
	Sleep<7 hours		-0.003
	Sleep>9 hours		-0.007
	Financial stress		0.001
	Black	0.015**	0.015**
	Employed	-0.003	-0.002
	Retired	-0.001	-0.001
	Other	-0.013	-0.011
	Female	0.005	0.007
Variance Components:			
Level_1: within-person	σ_{ε}^2	0.282***	0.282***
Level_2: In initial status	σ_0^2	0.2738***	0.2209***
In rate of change	σ_1^2	0.0056***	0.0055***
Covariance	σ_{01}^2	-0.0058**	-0.0055**
Deviance (-2 log likelihood)		19906.5	19394.3
Degree of freedom		31	52
AIC		19970.5	19489.3
BIC		20168.7	19820.3

Table 4.3: Mixed-effects Regression of Physical Health on Time Variant Income, Financial Stress, and Health Risk Behaviors

		Model 1	Model 2	Model 3	Model 3	Model 4
Fixed Effects						
Initial status (π_0)		1.408***	1.510***	1.491***	1.293***	1.331***
Time (Rate of change π_1)		0.030***	0.030***	0.032***	0.028***	0.019***
Income			-0.045***	-0.038***	-0.025***	-0.030***
Income by Time			0.002*	0.001	0.001	0.002*
Financial stress				0.056***	0..046***	0.046***
No drink					0.225***	0.191***
No drink by Time						0.014**
Heavy drink					-0.031	-0.036
Heavy drink by Time						0.006
Under weight					0.225***	0.229***
Under weight by Time						0.034**
Over weight					0.092***	0.098***
Over weight by Time						-0.001
Physical activity					-0.230***	-0.205***
Physical activity by Time						-0.010***
No smoking					-0.133***	-0.101***
No smoking by Time						-0.016**
Variance Components:						
Level_1: within-person	σ_ε^2	0.2814***	0.2865***	0.2851***	0.2942***	0.2927***
Level_2: In initial status	σ_0^2	0.4632***	0.4306***	0.428***	0.311***	0.317***
In rate of change	σ_1^2	0.0063***	0.0063***	0.0063***	0.0055***	0.0054***
Covariance	σ_{01}^2	-0.0008	-0.0022	-0.002	-0.0071**	-0.008***
Deviance (-2 log likelihood)		21531.2	21393.8	21359.6	20406.1	20362.5
Degree of freedom		6	8	9	14	21
AIC		21543.2	21409.8	21377.6	20436.1	20404.5
BIC		21580.4	21459.3	21433.3	20529	20534.5

† $p<0.10$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

2. Age, SES and Functional Limitation over the life course

This chapter applies the Multi Cohorts Accelerated Longitudinal Design (MCALD) to explore the development of health over the life course based on ACL data set. Age at interview waves is the measurement of passage of time. The MCALD suggests that there is a latent common health trajectory over the life course, around which individuals' health trajectory may vary. As indicated in the chapter of methodology, the model could be expressed as following:

$$Y_{ij} = \pi_{0i} + \pi_{1i} * (Age_{ij} - 25) + \pi_{2i} * (Age_{ij} - 25)^2 + \varepsilon_{ij} \longrightarrow \text{Level-one Model}$$

$$\left.\begin{aligned} \pi_{0i} &= \gamma_{00} + \sum_{k=1}^{n} \gamma_{0k} * X_{ik} + \xi_{0i} \\ \pi_{1i} &= \gamma_{10} + \sum_{k=1}^{n} \gamma_{1k} * X_{ik} + \xi_{1i} \\ \pi_{2i} &= \gamma_{20} + \sum_{k=1}^{n} \gamma_{2k} * X_{ik} + \xi_{2i} \end{aligned}\right\} \text{Level-two Model}$$

Level-one model identifies the health trajectory over the life courses, in which age is the only predictor of health. Results from the Multi Level Model for Change in previous chapter suggest a curvilinear relationship between age and health over time. The above model helps to indicate if cohort effect on health from a snapshot data set actually reflects the effect of age.

Level-two model explores factors (k refers to the number of social, psychological, environmental, and personal characteristics) that are responsible for health trajectory. It suggests that the instant rate of change in health at a given age and acceleration rate are associated with k factors. In other words, it suggests that people may have different

health trajectory due to their differences in these factors. Predictors in level-two model are considered as time invariant.

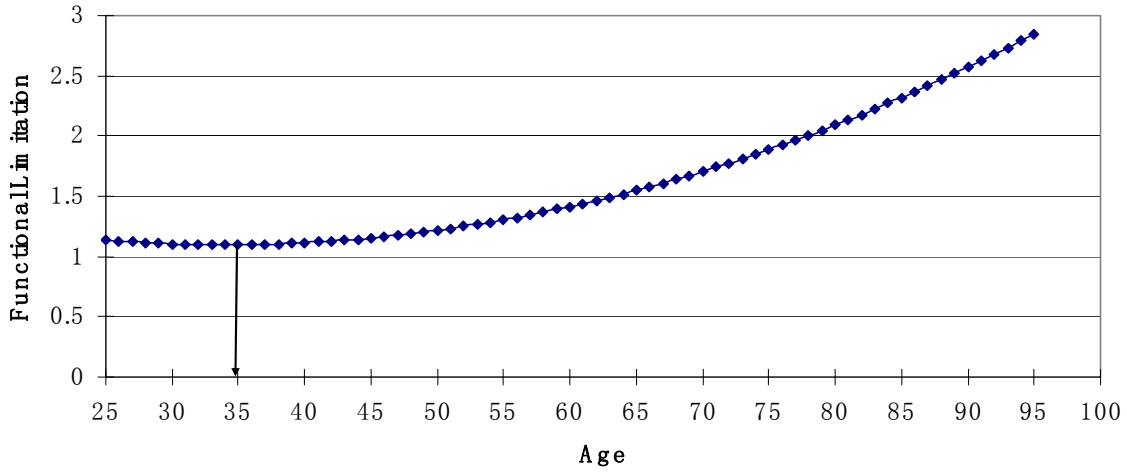
Age and Health Trajectory

Model (1) in table 4.4 on page 117 is unconditional growth model, in which age is the only predictor of physical health. Unconditional growth model suggests a curvilinear relationship between age and health. The intercept at age 25 is statistically significant, which means that people's health is somewhat between "no functional impairment' and "least severe level" at age 25. The instant rate of change at a given age is negatively significant at $p<0.001$) and the acceleration rate is positively significant at $p<.001$. This suggests that in general people's health are actually getting better after age 25 until they reach age 34.9 years old, which is the age with best health (the age, when first derivative equals to zero, is the one when health is the best). Therefore, the development of health over the life course could be described as following: people's health is improving as aging before age 34.9, although the rate of improvement is diminishing. However, after age 34.9, people's health starts to decline as aging, and the decline rate is accelerating with age.

Model (1) in table 4.4 is graphically illustrated in Figure 4.3 on page 102. This figure is a general development of health over the life course, in which predictors (except age) are not taken into account. This model provides a base for further explorations.

As shown in Figure 4.3, the health for an average person will actually get better after age 25 and this trend will continue to age 34.9. After age 34.9, his/her health starts to decline in an accelerated rate.

Figure 4.3: Age and Health Status Over Life Course



SES and Health Trajectory over the life course

Model (2) in table 4.4 includes income as predictor of health trajectory. This model suggests that SES (measured by income) impacts health trajectory over time, such that the health trajectories of people from different SES groups vary around the common health trajectory. This model identifies the overall effect of income (both direct and indirect effects from income) on health trajectory.

As shown in model (2), there is no significant difference in health across SES groups at age 25. This suggests that the effect of SES on health may not be explicit at young age. As a matter of fact, I hypothesize that biological factors of human beings offset the effect of SES at early stage of life such that SES differences (if any) is not explicit. However, the instant rates of change at a given age are significantly different across SES groups in that low SES people have a positive rate at significant level of $p<.001$, while mid and high SES groups have a negative rate at significant level of $p<.001$. The instant rate of change is adjusted by the rate of acceleration over time such that the differences in the

rate of change across different SES groups diminish over time. The combination of the instant rate of change at a given age and the rate of acceleration identifies three different health trajectories across SES groups: For low income groups, their health consistently decline since age 25, while for mid and high income groups, their health improve after age 25 until they reach the healthiest point (it is age 38.63 for mid income groups; it is age 37.62 for high income groups) in their life. After the healthiest age point, their health starts to decline. Thus, compared to low income groups, mid and high income groups have lagged age when their health start to decline. The health trajectories across SES groups are different such that health of low income groups decline faster than those of mid and high income groups over time.

Figure 4.4 on page 99 illustrates the different health trajectories across income groups. The two arrows point to the ages when mid and high income groups reach their healthiest point in their life (age 38.63 for mid income groups; age 37.62 for high income groups). The healthiest point for low income groups is beyond the scope of the age range in ACL, which suggests that the healthiest point for low income groups might be some age younger than age 25.

Low income groups consistently have worse health than mid and high income groups over the life course. The health gaps, between low income groups and mid, high income groups, start at early life stage, become larger in middle age, and decline to some extent in late life (however, never disappear). Mid and high income groups have similar health trajectories over the life course; however, high income groups enjoy healthy life longer than mid income groups.

Figure 4.5 on page 105 illustrates health gaps between low income and mid, high income groups over the life course. The largest health gap between low and mid income groups is about age 62; however, the largest health gap between low and high income groups is about age 70. This result suggests that the higher income a person is, the longer he/she will enjoy healthy life. The functional limitation is largely compressed to the very late of life for high SES groups.

Results from model (2) suggest that the effect of SES (measured by income) over health is not constant across the life course. At young age, the effect of SES is not explicit; however, it cumulates to be significant at middle age, and diminishes (but never disappears) at late age. The different effects of SES at different life stage contribute to SES disparities in health over the life course.

The findings from this study support the statement of House et al. (1994, 2005) in that SES differences in health start small at early life stage, increase with age in middle age and decline to some degree late in life. However, this study suggests that the different timing of aging processes between high and low SES groups (instead of greater exposure of low SES to health risk behaviors in the middle age as House et al. (1994, 2005) suggest) lead to the observed SES disparities in health in the middle age.

Figure 4.4: Age and Health Trajectory by Income Groups

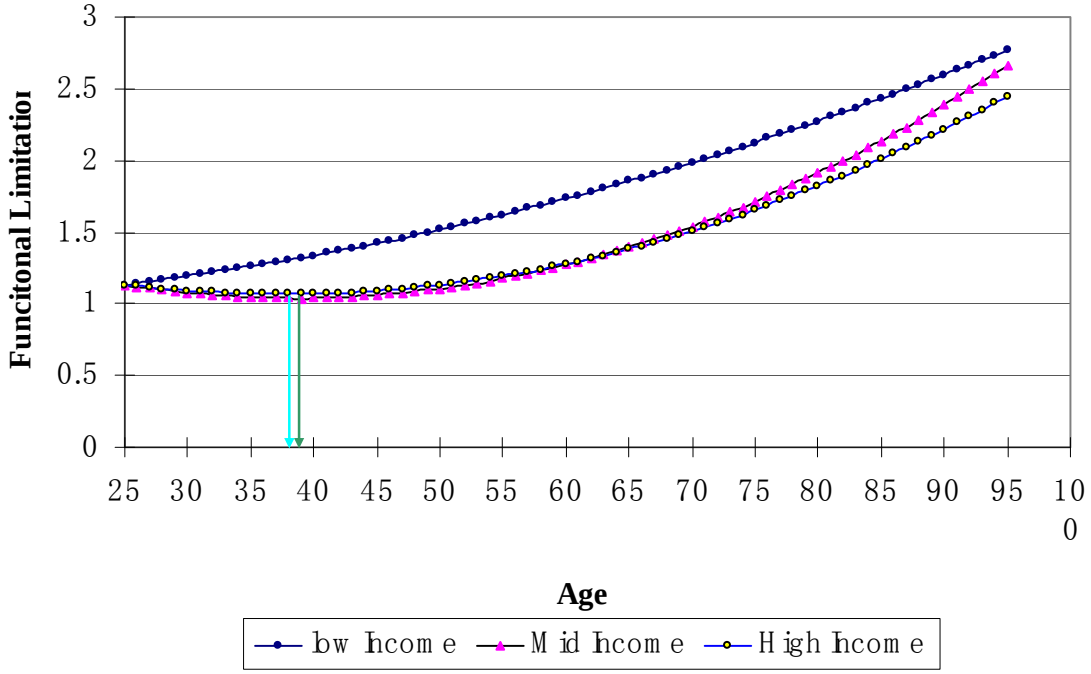
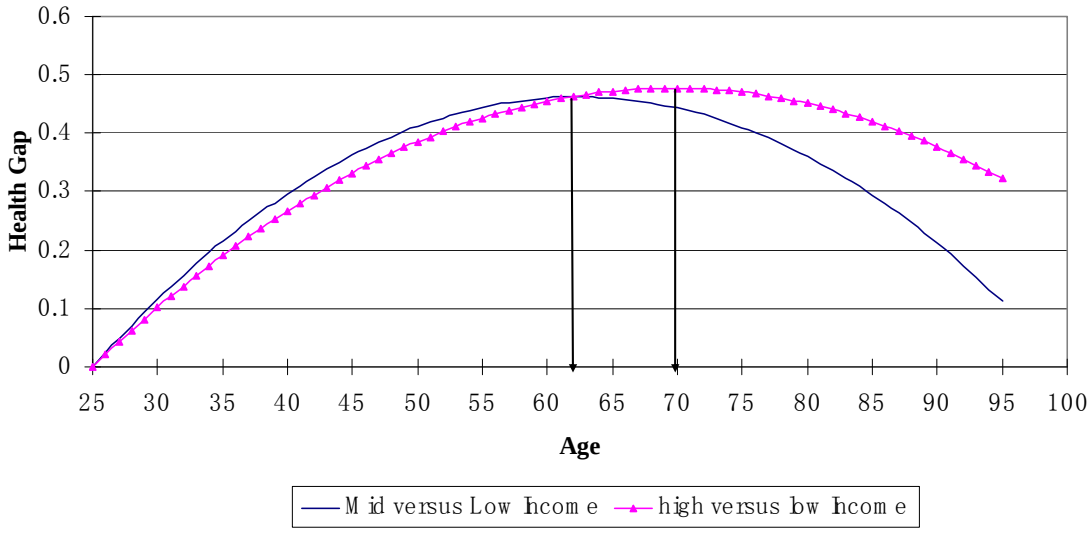


Figure 4.5:Health Gap Between Mid, High Income Groups and Low Income



Model (3) through (6) in table 4.4 explores the effect of education, interaction between education and income, and financial stress on health trajectory. Model (3) shows that education does not have significant effect on health trajectory; however, income consistently affects health trajectory even after controlling education (as shown in model (4)). This suggests that the effect of income on health is not mediated by education. And income, as a measurement of SES, is more informative in predicting health trajectory than education.

Model (5) explores the interactive effect between education and income. Income in this model is continuous (instead of categorized as in previous models) in order for better interpretation of coefficients. There is no interactive effect between education and income at age 25. However, there are interactive effects of income and education on instant rate and acceleration rate in health. People with college degree have lower instant rate than those with elementary degree as income increases; however, this gap diminishes and eventually cross over in middle life. Income has constant effect on instant rate of change between those with high school and elementary degree. In acceleration rate, people with college degree have lower acceleration rate with every $10,000 increase in income, and this gap gets large as income increases. People with high school degree marginally have lower acceleration rate than those with elementary degree with every $10,000 increase. Although increased income also benefits people with elementary degree in the instant rate of change, it harms the acceleration rate over time.

The interaction effect between income and education on the rate of acceleration suggests that increased income benefits higher education groups more than it does to low-

educated groups. However, including interaction term between education and income in model (5) uses more degree of freedom; moreover, the deviance is larger than that in model (4). Models comparison, based on deviance and degree of freedom, indicates that model (4) is a better model than model (5). Thus, model (4) is used as base model for further exploring.

Model (6) adds financial stress at first wave into model (4). Financial stress is negatively associated with higher level of functional limitation at age 25, which is opposite to expectation. It is positively associated with the instant rate of change at a given age; it is negatively related with the rate of acceleration. This result suggests that financial stress declines individuals' health over time. However, the effect of financial stress over health diminishes over time. This may be due to the fact that the longer a person experiences financial stress, the more tolerant he/she will be. In turn, the negative effect of financial stress diminishes.

Model (6) shows that the effect of SES is largely mediated by financial stress. People of high income are not better off than low income if financial stress is same. People of mid income are still better off than those of low income after holding financial stress constant; however, the magnitude largely decreases (instant rate of change decreases by 97.2%; and the rate of acceleration decreases by 27.8%). Therefore, the differential health trajectories among different SES groups are largely due to the fact that lower SES groups are more likely to experience financial stress, which in turn negatively affect health over time.

[TABLE 4.4 ABOUT HERE]

Health risk behaviors and health trajectory over the life course

Model (2) in table 4.5 on page 118 includes health risk behaviors as predictors of health over the life course after including SES, financial stress, and control variables. Health risk behaviors are treated as time invariant variables, which assume that people are likely to keep their lifestyle over the life course.

Health risk behaviors are not associated with health at age 25; neither are they associated with the instant rate of change at a given age (except physical activity is marginally associated with the instant rate of change at a given age) and the rate of acceleration.

Insignificant coefficients of health risk behaviors in model (2) do not necessarily lead to the conclusion that health risk behaviors are not negatively associated with people's health. It is very possible that people do change their health risk behaviors over time such that their health may get better or worse correspondingly. Treating health risk behaviors as time invariant may actually cancel out the effect of changed health risk behaviors on health over time. In a mixed-effects model the change of health over time has been taken into account. Correspondingly, the change of health risk behaviors should be expressed in a mixed-effects model as well. Time invariant health risk behaviors do not reflect such changes. This leads to the need to explore how changes in health risk behaviors may correlate with the change of health over the life course.

[TABLE 4.5 ABOUT HERE]

Time variant SES (measured by income) and health over the life course

The above models are based on the assumption that people's SES is static over the life course, which leads to the exploring of time variant income as predictor of health trajectory.

The model, with time variant income as predictor, could be described as the following:

$$Y_{ij} = \pi_{0i} + \pi_{1i} * (Age_{ij} - 25)/10 + \pi_{2i} * (Age_{ij} - 25)^2/100 + \pi_{3i} * Income_{ij} + \varepsilon_{ij}$$

$$\pi_{0i} = \gamma_{00} + \upsilon_{0i}$$
$$\pi_{1i} = \gamma_{10}$$
$$\pi_{2i} = \gamma_{20}$$
$$\pi_{3i} = \gamma_{30}$$

here i refers to respondents, varying from 1 to 3617, j refers to age, varying from 25 to 103. This model invokes a constraint on income such that the effect of income is constant across population. γ_{10} is the population average instant rate of change in health at a given age, controlling income; γ_{20} is the population average rate of acceleration; γ_{30} is the average change in health as income increases by $10,000. The above model suggests that health trajectory is not static over the life course; change in income will lead to the change in health trajectory.

Table 4.6 on page 119 shows the results of mixed-effects regression of health on age, time variant income, the interaction term between them, and health risk behaviors.

Model (1) is an unconditional growth model, which indicates the general health trajectory over the life course. Model (2) includes income as time variant predictor. The coefficient of income is negatively significant at $p<.001$, which suggests that health

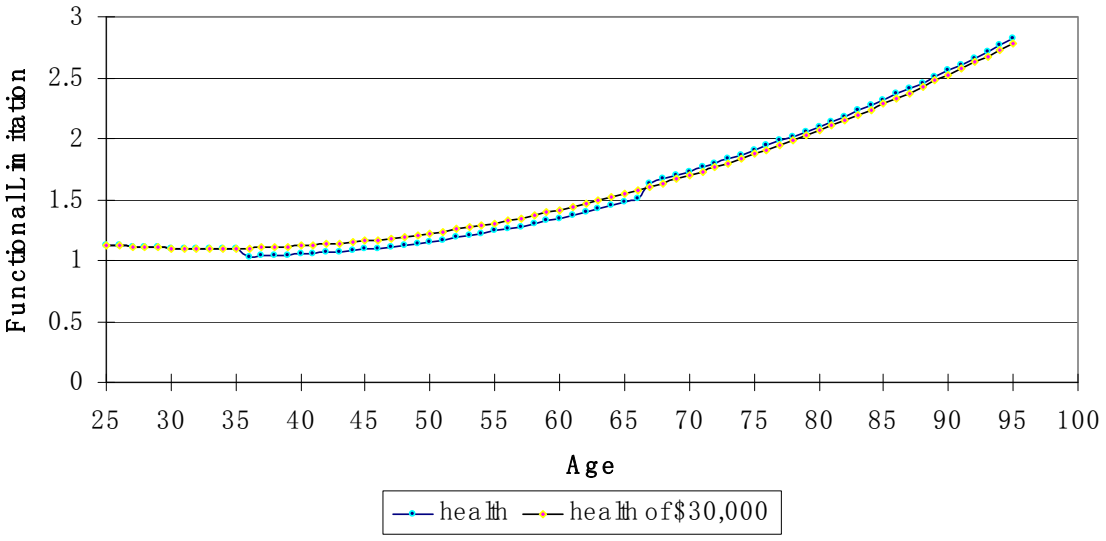
trajectory does respond to change of income. (Model not shown here, includes an interaction term between age and income to test if the effect of income on health depends on age. The coefficients of interaction term are not significant, which suggests that increased income constantly benefits health over the life course.)

Model (3) includes financial stress. Financial stress is positively related with health. This suggests that people's health will be on worse path if their financial stress increases. Financial stress mediated 53% effect of income on health (based on comparison of model (3) and (2)).

Figure 4.6 on page 111 illustrates the health trajectory of a person who earns $30,000 at age 25 through 35, $70,000 at age 36 through 66, and $10,000 at age 67 through 95. This figure is for illustrative purpose.

The line in the middle is the predicted health trajectory for an average person whose income is consistently $30,000over the life course. The line under the middle one predicts the switch of health trajectory if the person's income increases from $30,000 to $70,000. The line above the middle one predicts the health trajectory of the person whose income decrease from $70,000 to $10,000 in late life. The figure shows that people's health trajectory do switch ups and downs corresponding to the changes of income over time.

Figure 4.6: Time Variant Income and Health Trajectory



Time variant health risk behaviors and health

In order to test the effect of time variant health risk behaviors, I include time variant variables into model. The base model could be described as following:

$$Y_{ij} = \pi_{0i} + \pi_{1i} * (Age_{ij} - 25)/10 + \pi_{2i} * (Age_{ij} - 25)^2/100 + \pi_{3i} * Income_{ij} + \sum_{i=1}^{n}\sum_{j=25}^{103}\sum_{k=1}^{6}\pi_{4ki}X_{ijk} + \varepsilon_{ij}$$

$$\pi_{0i} = \gamma_{00} + \upsilon_{0i}$$

$$\pi_{1i} = \gamma_{10}$$

$$\pi_{2i} = \gamma_{20}$$

$$\pi_{3i} = \gamma_{30}$$

$$\pi_{3ki} = \gamma_{3k0}$$

here i refers to respondents, which varies from 1 to 3617; j refers to age; k refers to the number of health risk behaviors, which is 6 in this study.

This model explores the relationship between health trajectory and change of health risk behaviors after controlling income. Model (4) and (5) in table 4.6 (time variant health risk behaviors and health) indicates that changes in health risk behaviors are correlated with changes in health. People without drinking have worse health over time than people with moderate drinking; however, heavy drinking people do not seem to have worse health than moderate drinking people. Under weight and over weight both harms health over time, compared to normal weight. People, used to be under weight, will have a better health trajectory if they are normally weighted, although this benefit diminishes. Overweight is negatively associated with health and such association is not moderated by aging. Physical activity is positively associated with health in that higher level of activity is related with healthier life trajectory over time. The effect of smoking on health is moderated by age. The longer an individual smokes, the more significant differences in

health will be between smoker and non-smoker. This suggests that the effect of smoking is accumulative over time.

The result in model (4) and (5) indicates that health risk behaviors mediate 47% effect of income on health (this statement is based on the comparison between model (3) and model (4) in table 4.6 after controlling financial stress). Income is not significant when financial stress and major health risk behaviors are included. This suggests that the effect of SES on health is largely due to the fact that poor people are more likely to experience financial stress and to adopt health risk behaviors.

Model (5) includes the interaction term between age and health risk behaviors as predictors of health. This model is to examine if the effect of health risk behaviors on health vary at different age. All health risk behaviors do not have significant effect on health at age 25, which suggests the firmness of human body at young age such that the effects of health risk behaviors are not explicit at early age. However, some health risk behaviors do significantly affect the instant rate of change at a given age and the acceleration rate of change.

Under weight and over weight are positively related with the instant rate of change at a given age; however, the acceleration rate associated with under weight is negative and no difference between over weight and normal weight at the acceleration rate. This suggests that underweight people get worse faster than normal weight people; however, the rate is diminishing as aging. Overweight people consistently get worse faster than people of normal weight over the life course.

Physical activity does not have significant effect on the instant rate of change at a given age; however, it is negatively related with the acceleration rate. This suggests that the effect of physical activity cumulates over time such that health differences due to physical activity become explicit as time goes time.

Results from model (4) and (5) suggest that people's health risk behaviors do change over time, and such change results in different health trajectory. These results also suggest that it is better to treat time variant variables as they are in a longitudinal study. Otherwise, findings (based on treating health risk behaviors as time invariant) may be misleading.

[TABLE 4.6 ABOUT HERE]

Control Variables and Health

Race, employment status, gender are considered as control variables in this study. One interesting finding in Model (1) and (2) in table 4.5 is that women have lower instant rate of change at a given age and higher acceleration rate. This suggests that women stay on healthy life longer than men do, although the health difference between them diminishes at late life. This leads to the need to explore social and biological factors, which are responsible for gender differences in health trajectories over the life course. However, this is beyond the scope of this study.

3. SUMMARY

This part of study explores Age, SES and Health, using functional limitation as measurement of health. Two types of measurement of time are applied, which include passage of year since first wave interview and ages at interview waves. Financial stress and health risk behaviors as mediators of the effect of SES over health are explored. The Multi Level Model for Change and Multi Cohort Accelerate Longitudinal Design are applied to explore health trajectory over 8-year study period and over the life course.

Results from models using passage of years since first wave indicates that people's health are different at the beginning of ACL study; moreover, the rates of change in health are also different across different people. In general, higher SES (measured by income) groups are better off than low SES groups from the snapshot point of view; they are aging slower over time as well. Furthermore, the relationships between age cohorts and initial health at first wave, and the rate of change in health are curvilinear. This suggests that the process of aging is not linearly downward. People's health may actually be getting better to some age point. After that age point, their health starts to decline. However, the age point where people's health starts to decline varies across SES groups. For lower SES groups their health start to decline at early age, while higher SES groups have longer active life expectancy. People of different SES are aging in different ways. The cohort effect at first wave largely reflects the effect of age, which is supported by the results of Multi Cohort Accelerate Longitudinal Design.

Previous researchers propose health risk behaviors as the major mediators of the relationship between SES and health. This study partially supports previous research in

that health risk behaviors, together with financial stress, significantly mediate part of the effect of SES.

Models treating income as time variant variable indicates that increased income is associated with better health; however, the effect of increased income diminishes over time. All time variant health risk behaviors are significantly associated with health, which suggests that people's health behaviors do change over time and such changes lead to corresponding change in health. The effect from time variant health risk behaviors mediates about 50% effect of time variant income, which suggests that health risk behaviors are major mediators for the link of SES and health. Findings about the effect of time variant health risk behaviors suggests that it is more optimal to treat health risk behaviors as time variant in a longitudinal study.

Results from the model using age as measurement of time are largely consistent with those from the model using the passage of years since first wave interview as measurement of time. Health trajectories across different SES groups are different over the life course. For lower SES groups, their health tends to decline linearly since age 25, while for mid and higher SES groups their declination on health are postponed to late 30's. Therefore, at early life stage, the effect of SES over health is not explicit; however, its effect cumulates to be significant in middle age, while it diminishes (but not disappear) in late life when the fragility of human body becomes predominant.

Income has different effect over health at different life stage. The effect of income on health is not significant at young age; it cumulates to be significant during middle age

and diminishes in the old age. Thus, SES disparities in health over the life course largely reflect the effect of income during different life stages.

Financial stress and health risk behaviors are two major mediators of SES over health. In general, higher level of financial stress is associated with worse health. Higher income groups does not have better health than low SES groups if they face same level of financial stress and health risk behaviors. It is obvious that low SES groups are more likely to experience financial stress and to adopt health risk behaviors. Therefore, day-to-day financial stress faced by low SES and bad habits channel them into an accelerated downward health path from that of high SES groups.

Table 4.4: Mixed-effects Regression of Physical Health on Age, Education, Income, and Interaction Terms

		Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Fixed Effects							
At age 25 (π_0)	Intercept	1.136***	1.133***	1.309***	1.324***	1.130***	1.335***
	Mid income[a]		0.044		0.06		0.013
	High income[a]		-0.034		-0.011		-0.086
	High school[b]			-0.157	-0.206	0.035	-0.170
	College[b]			-0.169	-0.217	0.067	-0.176
	Income($10,000)					0.201	
	Highsch*income					-0.212	
	College*income					-0.228	
	Financial stress						-0.074*
Rate of change (age-25)/10	Intercept	-0.085***	0.108**	-0.049	0.041	0.165	-0.049
	Mid income		-0.247***		-0.255***		-0.017**
	High income		-0.214**		-0.223**		-0.008
	High school[b]			-0.029	0.075	-0.134	0.006
	College[b]			-0.073	0.076	-0.293*	0.006
	Income($10,000)					-0.214*	
	Highsch*income					0.172	
	College*income					0.229*	
	Financial stress						0.013***
Rate of acceleration (age-25)^2/100	Intercept	0.047***	0.018**	0.042***	0.027**	0.01	0.041***
	Mid income		0.033***		0.036***		0.026**
	High income		0.024*		0.029*		0.011
	High school[b]			0.001	-0.014	0.015	-0.011
	College[b]			0.005	-0.015	0.042*	-0.011
	Income($10,000)					0.030*	
	Highsch*income					-0.025†	
	College*income					-0.035**	
	Financial stress						-0.016***
Variance Components:							
At age 25	σ_0^2	.3331***	0.2995***	0.3155***	.295***	.3021***	.2793***
Within-person	σ_ε^2	.3714***	0.3734***	0.3726***	.3738***	.3727***	.3718***
Deviance (-2 log likelihood)		20873.7	20655	20765.1	20626.6	20663.2	20463.3
Degree of freedom		5	11	11	17	20	20
AIC		20883.7	20677	20787.1	20660.6	20703.2	20503.3
BIC		20914.7	20745.1	20855.2	20765.9	20827.1	20627.2

Note:
a: low income is reference category
b: elementary school is reference category
† $p<0.10$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

Table 4.5: Mixed-effects Regression of Physical Health on Age, SES, Health Risk Behaviors, and Control Variables

		Model 1	Model 2
Fixed Effects			
At age 25 (π_0)	Intercept	0.801**	0.733**
	High school[b]	0.134	0.179
	College[b]	0.222	0.257
	Income ($10,000)	0.223	0.254
	Highsch*income	-0.227	-0.251
	College*income	-0.257	-0.283
	Financial stress	-0.057†	-0.041
	No drink		0.004
	Heavy drink		0.112
	Under weight		-0.007
	Over weight		-0.01
	Physical activity		0.026
	Smoking		-0.069
	Sleep<7 hours		0.033
	Sleep>9 hours		0.054
	Black	-0.022	0.018
	Employed	0.07	0.087
	Retired	2.601***	2.328***
	Other	0.201	0.177
	Female	0.134†	0.131†
Rate of change (age-25)/10	Intercept	0.379*	0.361*
	High school[b]	-0.134	-0.136
	College[b]	-0.252	-0.232†
	Income ($10,000)	-0.181†	-0.204*
	Highsch*income	0.161	0.181†
	College*income	0.207*	0.023*
	Financial stress	0.108***	0.087***
	No drink		0.021
	Heavy drink		-0.127
	Under weight		0.077
	Over weight		0.099
	Physical activity		-0.058*
	Smoking		0.087
	Sleep<7 hours		-0.028
	Sleep>9 hours		-0.007
	Black	0.044	-0.008
	Employed	-0.165	-0.184†
	Retired	-1.242***	-1.099***
	Other	-0.052	-0.039
	Female	-0.154**	-0.152**
Rate of acceleration $(age-25)^2/100$	Intercept	-0.035	-0.040
	High school[b]	0.016	0.017
	College[b]	0.034†	0.035†
	Income ($10,000)	0.028*	0.032**
	Highsch*income	-0.024†	-0.026*
	College*income	-0.032*	-0.035**
	Financial stress	-0.012**	-0.009*
	No drink		0.003
	Heavy drink		0.0204
	Under weight		-0.012
	Over weight		-0.009
	Physical activity		-0.001
	Smoking		-0.009
	Sleep<7 hours		0.009
	Sleep>9 hours		0.0072
	Black	-0.007	-0.002
	Employed	0.021	0.0219
	Retired	0.147	0.126***
	Other	0.012	0.004
	Female	0.030***	0.026***
Variance Components:			
At age 25	σ_0^2	0.2626***	.1986***
Within-person	σ_ε^2	0.3699***	0.3707***
Deviance (-2 log likelihood)		20288.6	19708.6
Degree of freedom		38	62
AIC		20364.6	19832.6
BIC		20600	20216.6

Note:
a: low income is reference category
b: elementary school is reference category
† $p<0.10$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

Table 4.6: Mixed-effects regression of Physical Health on Time variant Income, Financial Stress, and Health Risk Behaviors

	Model 1	Model 2	Model 3	Model 4	Model 5
Fixed Effects					
At age 25 (π_0)	1.136***	1.176***	1.109***	1.019***	1.056***
Rate of change(age-25)/10	-0.085***	-0.071**	-0.066**	-0.071**	-0.034
Rate of acceleration $(age-25)^2/100$	0.047***	0.044***	0.046***	0.039***	0.025***
Income ($10,000)		-0.017***	-0.008**	-0.001	-0.003
Financial stress			0.121***	0.098***	0.099***
No drink				0.168***	0.012
No drink by (age-25)/10					0.043
No drink by (Age-25)2/100					0.001
Heavy drink				0.009	0.096
Heavy drink by (age-25)/10					-0.073
Heavy drink by (Age-25)2/100					0.007
Under weight				0.255***	-0.109
Under weight by (age-25)/10					0.195*
Under weight by (Age-25)2/100					-0.020†
Over weight				0.158***	-0.022
Over weight by (age-25)/10					0.109†
Over weight by (Age-25)2/100					-0.012
Physical activity				-0.195***	-0.009
Physical activity by (age-25)/10					-0.031
Physical activity by (Age-25)2/100					-0.006*
Non-smoking				-0.033	-0.049
Non-smoking by (age-25)/10					0.066
Non-smoking by (Age-25)2/100					-0.016†
Variance Components:					
At age 25	0.3331***	0.3241***	0.2983***	0.2243***	0.2023***
Within-person	0.3714***	0.3733***	0.3747***	0.371***	0.3677***
Deviance (-2 log likelihood)	20873.7	20841.3	20669.7	19965.6	19687.6
Degree of freedom	5	6	7	13	25
AIC	20883.7	20853.3	20683.7	19991.6	19737.6
BIC	20914.7	20890.5	20727.1	20072.1	19892.4

† $p<0.10$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

Chronic Diseases as Measurement of Health

Chronic diseases are another major components of health. This part of study explores how health (measured by chronic diseases) is associated with Age and SES from the perspective of longitudinal study.

I break down this part into three chapters: Chapter 4-II-4 uses the number of chronic diseases (Natural logged count to reduce positive skewness) at each wave as the measurement of health over time, and explore the relationship of health trajectories to age cohorts and SES during the 8-year study periods in ACL. The passage of year since first interview is used as measurement of time. This chapter mainly provides base for my critique that using count of chronic disease is not optimal for a longitudinal study, because this measurement doesn't actually tap change of health over time if the number of chronic diseases is constant.

Chapter 4-II-5 uses the combination of count and duration of chronic diseases (Natural logged to reduce positive skewness) as measurement of health over time. The passage of year since first interview will be used as measurement of time. The results from this chapter are compared with those from chapter 4-II-4. It is suggested that the combination of count and duration of chronic diseases is an optimal measurement of health over time. The combination measurement taps the actual change of health even the number of chronic diseases may keep constant over time.

Chapter 4-II-6 expands the findings from chapter 4-II-5 in that the Multi Cohorts Accelerated Longitudinal Design (MCALD) is used to explore health trajectory over the life course, instead of only 8-year study period.

4. Age, SES and Chronic Diseases (Count) in the 8-year Study Period

The results from mixed-effects model are presented in table 4.7 on page 134. Regression coefficients in the initial status indicate the association between health (measured by count chronic diseases) and predictors at the beginning of the study (in 1986). Regression coefficients in the rate of change indicate the rate of health change during the 8-year study interval and factors responsible for such change.

Model (1) is unconditional growth model, in which time (passage of years since first interview in ACL) is the only predictor of health. The intercepts in both the initial status and rate of change are positively significant, which suggest that on average people have some chronic diseases at the beginning of the study; furthermore their health decline over time. The variances both in initial status and rate of change are significant at $p<0.001$, suggesting variability in both the health at the beginning of the study, and the rate of change over time across individuals. The covariance between initial status and rate of change is negatively significant, which suggests that healthier people at the beginning of the study decline in a faster rate over time than those having high level of chronic diseases at the beginning of the study. In sum, results from model (1) suggest variation in people's health trajectories over 8-year study period.

The unconditional growth model provides significant variation in initial status and the rate of change, for which level-2 predictors are responsible. Model 2 through 12 provide stepwise regression results, exploring how level-2 predictors, such as age, SES, health risk behaviors, and control variables, are associated with health trajectories in the 8-year study period.

Age cohort and health trajectories

Results in table 4.7 suggest curvilinear relationships between age cohorts and health at the beginning of the study, between age cohorts and the rate of change in health over time. Results in table 4.7 also indicate interactive effect of age cohort and SES on health at the beginning of the study. Health gap between lower and higher income is getting larger as age cohorts increase, although the rate diminishes. The interactive effect of education and age cohort is largely through that of age cohort and income. At the rate of change, age cohort does not interact with SES.

Age cohort and health at the beginning of the study

The fixed effect at initial status in Model 2 (table 4.7) includes age (centered at age 25), age square. The coefficients indicate the overall effect of age cohort (including both direct and indirect effect of age cohort) on health at the beginning of the study.

The coefficients associated with age and age square indicates that the the number of chronic diseases increases as age cohort increases; however, the rate diminishes. Therefore, at the beginning of the study, old age cohorts have worse health (in term of the number of chronic diseases) than younger cohorts; however, the gap between adjunction cohorts diminishes as age cohort increases.

Model 3 through model 12 add SES, health risk behaviors, gender, race, and employment status as mediators of age. The coefficients of age and age square are consistently significant across all models, which suggest that the effect of age on chronic diseases is independent from the above mediators. This suggests that chronic diseases are largely related to age in that old people are more likely to experience chronic diseases

than the younger. This, to some extent, also suggests that chronic diseases may be due to the accumulation of health declination over time.

Does the relationship between age cohorts and health at the beginning of the study actual represent the effect of age on health in the process of aging? In other words, is the effect of age cohort on health actually reflecting the effect of aging in a life course? The Multi Cohort Accelerated Longitudinal Design (MCALD) (Tonry, Ohlin, and Farrington, 1991) provides a valid inference about the effect of age on health over time. Chap 4-II-6 applies MCALD to explore health trajectory in a life course. The results from MCALD test if cohort effect actually reflects the effect of age.

Age cohort and health at the rate of change

Model 2 through 12 in table 4.7 include age, SES, health risk behaviors and control variables in predicting the rate of health change in the 8-year study period. These models aim to explore if the above variables are responsible for health change over time.

Age cohorts are consistently related with the rate of change, and such relationship is curvilinear. The gap in rate of change between two adjunction age cohorts is larger among younger age cohorts; however, it diminishes as age increases.

Coefficients in the rate of change indicate the average health trajectory during the 8-year study period. On average, older cohort ages faster than younger cohorts; however, the acceleration rate diminishes as age increases.

SES and Health Trajectory

Model 3 include age, age square and income (categorized into low income, mid income and high income) to examine the effect of income on health trajectory over 8-year

study period. Model 4 examines the effect of education on health trajectory over 8-year study period. Model 5 is to test if education and income compound to affect health trajectory; Model 6 explores the interactive effect between income and education.

At the beginning of the study, results from the above models suggest that both income and education are negatively associated with the number of chronic diseases. In general, people from high income or high education groups have less the number of chronic diseases. The effect of income doesn't interact with that of education, which suggests an accumulative effect of income and education. This suggests that people with high income and high education are most likely to be among the healthiest groups and those with low income and education will be at the bottom of health stratum.

At the rate of change, income is positively associated with the rate of change in chronic diseases. However, education doesn't significantly impact the rate of change in the number of chronic diseases. These results suggest that the health of higher SES groups will eventually catch up with lower SES groups due to their higher rate of change.

In chapter 3-II "Measurement of Health in Term of Chronic Diseases", I argue that the number of chronic diseases of a respondent is likely to be stable over time unless she/he experiences new chronic diseases. People from low income groups are more likely to have more chronic diseases at the beginning of the study, and this high level of chronic diseases may likely stay stable over the 8-year study period. There is no much space for low income people to exceed the ceiling of total six chronic diseases, thus, their rate of change in chronic disease will be small over time. This is called "ceiling effect". Zimmer and House (2003) also identify "ceiling effect" in their study that people with severe

functional limitations at the baseline have zero coefficient in slope over time because they can not get worse. On contrary, people from higher income groups have less chronic diseases; they have more space for further chronic diseases. Thus, they have higher rate of change in chronic diseases over time.

In sum, I argue that the higher rate of change among high income groups is largely due to a ceiling effect of using the number of chronic diseases as a measurement of health. This also suggests that using the number of chronic diseases as measurement of health over time may lead to biased results.

Age Cohort, SES and Health Trajectory

Model 2 through model 10 provide stepwise results about the effect of age, SES, financial stress, and the interactive effect between age and SES on the initial health and rate of change over the 8-year study period.

Results from the initial status provide cross-sectional view about the relationship between health and age cohorts, SES groups at the beginning of the study.

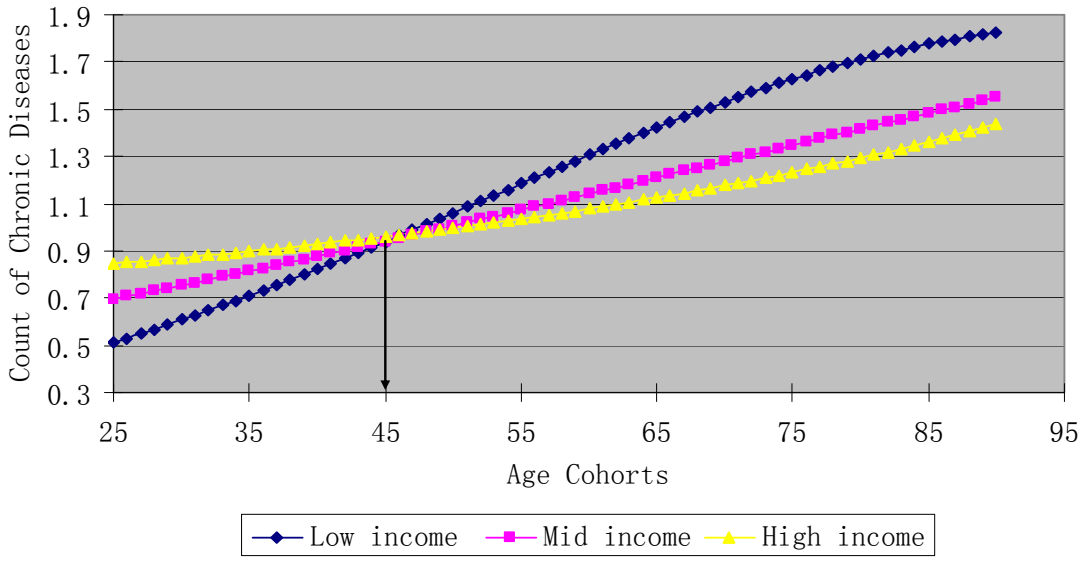
Results indicate that people from higher income or higher educated groups have fewer counts of chronic diseases at the beginning of the study. The effect of education on health is constant across different income groups, which suggest an accumulative effect of education and income. Income is interactive with age cohorts on health. The health gap across different SES groups gets large as age cohort increases; however, the acceleration rate of such gap decreases. There is an interactive effect between education and age cohorts at model 8; however, this interactive effect is not significant at model 9 when income and education are included in the same model. Thus, it is obvious that the

interactive effect between education and age cohorts is actually compounding with income.

Model 10 includes financial stress as mediator of SES. The effect of income is partially mediated by financial stress; however, it is consistently significant. This suggests that higher level of financial stress may be partially responsible for poor health among lower SES people. However, much is still remained to be identified as mediators of SES and health link.

Figure 4.7 on page 127 illustrates the interactive effect between age cohort and income at the beginning of the study. Before age 45 cohort, people from lower income groups are actually better off than those from higher income; however, low income cohorts have steeper slope than mid and high income cohorts. There is a turnover after age 45 cohort in that low income cohorts have higher level of chronic diseases than mid and high income cohorts.

Figure 4.7: Health and Age Cohorts by Income at the beginning of the study



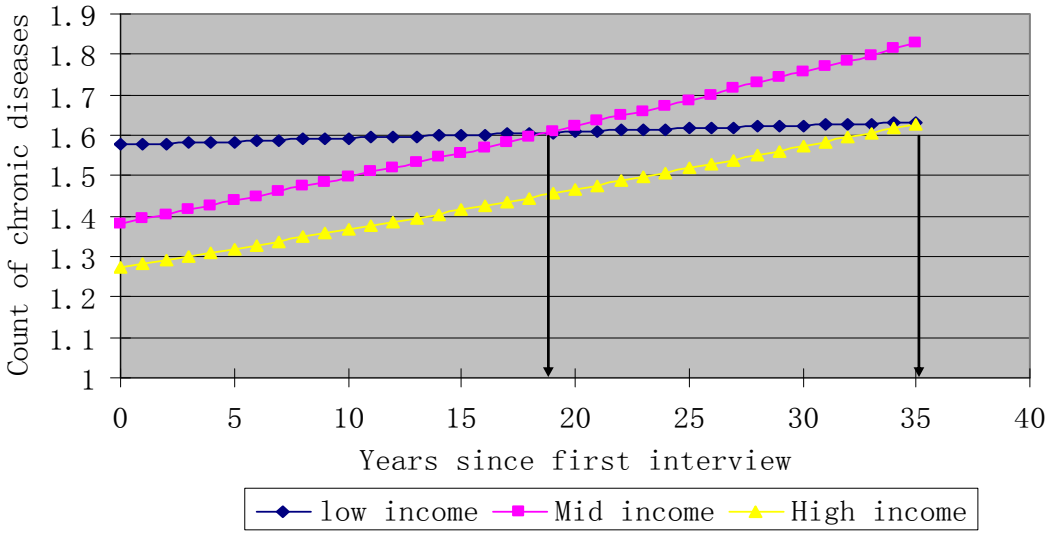
In the rate of change, older age cohorts have higher instant rate of change at a given age; however, the acceleration rate diminishes as age cohort increases. People from higher income groups have higher rate of change over time, which suggests that health of higher SES groups will eventually catch up with that of lower SES groups. Education does not relate to the rate of change. Financial stress does not mediate the effect of income on the rate of change.

Figure 4.8 on page 129 is the predicted health trajectories by income over 30 years since the beginning of ACL study. The Figure indicates that low income groups have higher level of chronic diseases at the beginning of ACL study, and they have relatively flat rate of change over time. Mid and high income groups have higher rates of change over time, although they have fewer the number of chronic diseases at the beginning of the study. The predicted health trajectories suggest that there is a turnover between mid and low income groups in the number of chronic diseases after 19 years since the beginning of the study. Mid income group will have higher level of chronic diseases than low income group 19 years later. The converge between high and low income groups will occur later in about 35 years.

The above results may lead to an "optimistic" view about SES disparities in health over the life course in that SES disparities in health will eventually diminish at later life. However, the big issue regarding SES disparities in health is that low income groups have experienced higher level of chronic diseases 35 more years than high income groups. Does this 35-year gap indicate some social issue regarding the SES disparities in health?

This leads to further exploration of duration of chronic disease across SES groups, which will be covered in chapter 4-II-5.

Figure 4.8: Predicted Health Trajectories by Income Groups



Health risk behaviors and health trajectory

Health risk behaviors as mediators of SES and health have been examined by previous researchers. Model (12) in table 4.7 (Continued) explores the effects of time invariant health risk behaviors on health trajectory over the 8-year study period.

Results show that non-drinking people actually are worse off in health than moderate drinking at the beginning of the study; however, there is no significant difference in health between people of heavy drinking and moderate drinking. In the rate of change, people of non-drinking have marginally higher rate than those of moderate drinking; however, there is no significant difference between people of heavy drinking and moderate drinking at the rate of change.

Thus, non-drinking individuals have worse health over time, compared to moderate drinking ones. The reason that there is no difference in health trajectories between heavy drinking and moderate drinking may be due to the arbitrary way of categorizing drinking behaviors, which has been explained in chapter 3-I.

Overweight people have higher level of chronic diseases than normal weight or underweight people at the beginning of the study; however, there is no significant difference in the rate of change across different weight people.

People with higher level of physical activity are healthier than those with lower level of physical activities. However, physical activity does not significantly affect the rate of change over time. Thus, physically active people will be consistently healthier than physically inactive ones over time.

Smoking doesn't significantly affect people's health at the beginning of the study; however, an individual who smokes marginally has a higher rate of change over time. This result is consistent with the finding from chapter 3-I where health is measured by physical limitation. This indicates that the effect of smoking may be accumulative both to physical health and chronic diseases, the longer a person smokes, the more likely he/she will experience physical limitation or chronic diseases.

People sleeping less than 7 hours or more than 9 hours are marginally worse off than those sleeping between 7 and 9 hours at the beginning of the study; however, they are not significantly worse in the rate of change.

In summary, no drinking, over weight, less physical activity, and sleeping less than 7 hours or more than 9 hours are negatively associated with health at the beginning of the study; however, none of these factors is associated with the rate of change over time. Adding these health risk behaviors to models marginally mediate the effect of SES at the beginning of the study. This suggests a robust effect of SES over health.

Health risk behaviors (except smoking) do not have significant effects on the rate of change, which is consistent with those findings in chapter 3-I.

The above result suggests it is necessary to use time variant health risk behaviors in models to reflect the reality that people may change their health risk behaviors over time and such change may affect their health.

[TABLE 4.7 ABOUT HERE]

[TABLE 4.7 (Continued) ABOUT HERE]

Time Variant SES (measured by income) and Health

Models in table 4.8 on page 136 utilize time variant income, financial stress and health risk behaviors to explore how health trajectory changes correspondingly to change in the above variables.

Model (1) is an unconditional growth model, which indicates the average health at the beginning of the study (in 1986) and rate of change over the 8-year study period. Variance components indicate that people's health are different in 1986, and the rates of change in health are different over time as well.

Model (2) includes time variant income and interaction of income and time as level-one predictors. The result shows that the number of chronic diseases will decrease as income increases; however, the effect of income diminishes as time goes by.

Results from time variant income are consistent with findings in chapter 4-II-1, where health is measured by physical limitation. Findings from these two chapters indicate that people's health (either measured by physical health or chronic diseases) do correspond to their change in income. Thus, income is an important source for health.

Financial stress partially explains income disparities in health over time; however, income consistently to be a significant predictor on health. Thus, other factors remain to be explored in the SES and health link.

Time variant health risk behaviors and health

Results in table 4.8 indicate that people's health do change corresponding to the change in health risk behaviors. Moderate drinking benefits health, while no drinking is associated with worse health. Over weight is associated with worse health over time;

however, under weight does not significantly impact health compared to normal weight. Physical activity benefits health in that higher level of physical activities is associated with lower level of chronic diseases. Smoking is negatively associated with health over time.

The results in the above models show that health risk behaviors do impact health trajectory over time. Therefore, insignificant effect of time invariant health risk behaviors on the rate of change in table 4.7 is mainly due to the inappropriate way of treating time variant variables as time invariant. Thus, it is optimal to treat time variant variables as the way they are in a longitudinal study.

[TABLE 4.8 ABOUT HERE]

Control Variables and Health

Race, employment status, and gender are considered as control variables when exploring SES disparities in health. Model (11) in table 4.7 (continued) shows that Black is worse off than non-Black at the beginning of the study; however, there is no significant difference in the rate of change over time across race groups. When controlling health risk behaviors in model (12), the health gap between Black and non-Black decreases by 15%. This suggests that higher levels of health risk behaviors among Black is partially responsible for worse health of Black.

There is no significant difference in health between male and female at the beginning of the study; however, female has lower rate of change in health over time. This suggests that male ages faster than female in term of chronic diseases.

Employed are healthier than unemployed at the beginning of the study; however, there are no significant differences in the rate of change between them. Disable people have worse health both at the beginning of the study and rate of change on health.

Worse health among retired than unemployed at the beginning of the study may actually reflect the fragility of human being at the later stage of life.

Table 4.7: Mixed-effects Regression of Chronic Diseases (Natural Logged) on Age, Education, Income, and Interaction terms

		Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10
Fixed Effects											
Initial status (π_{0i})	Intercept	0.344***	-0.408***	-0.360***	-0.213**	-0.252***	-0.218**	-0.668****	-0.556*	-0.509*	-0.549***
	(Age-25)/10		0.179***	0.223***	0.169***	0.210***	0.185***	0.351***	0.305***	0.322***	0.308***
	(Age-25) ^{2}/100		-0.007***	-0.012***	-0.007**	-0.012***	-0.009***	-0.024***	-0.02**	-0.022***	-0.020***
	Mid income[a]			-0.132***		-0.104***		0.306*		0.232	0.290*
	High income[a]			-0.213**		-0.173***		0.502*		0.397	0.528**
	High school[b]				-0.128***	-0.087***	-0.122***		0.209	-0.069	-0.078***
	College[b]				-0.195***	-0.116***	-0.199***		0.382	-0.017	-0.113***
	Income($10,000)						-0.025*				
	Highsch*income						0.008				
	College*income						0.018				
	(Age-25)/10*midinc							-0.189**		-0.153**	-0.161**
	(Age-25) ^{2}/100*midinc							0.018***		0.015**	0.016**
	(Age-25)/10*highinc							-0.296***		-0.245**	-0.273***
	(Age-25) ^{2}/100*highinc							0.028***		0.024**	0.026***
	(Age-25)/10*highsch								-0.137	-0.006	
	(Age-25) ^{2}/100*highsch								0.013†	0.001	
	(Age-25)/10*college								-0.238**	-0.046	
	(Age-25) ^{2}/100*college								0.022**	0.004	
	Financial stress										0.048***
Rate of change (π_{1i})	Intercept	0.013***	-0.035***	-0.039***	-0.037***	-0.037***	-0.035**	-0.026	0.019	-0.041***	-0.039***
	(Age-25)/10		0.019***	0.018***	0.019***	0.018***	0.017***	0.013†	-0.006	0.019***	0.019***
	(Age-25) ^{2}/100		-0.002***	-0.002***	-0.002***	-0.002***	-0.002***	-0.001**	0.008	-.002***	-0.002***
	Mid income			0.007**		0.008**		-0.016		0.007**	0.006*
	High income			0.006*		0.007*		-0.009		0.006*	0.004
	High school[b]				0.002	0.001	0.006		-0.061		
	College[b]				0.001	-0.003	0.003		-0.064		
	Income($10,000)						0.004				
	Highsch*income						-0.003				
	College*income						-0.003				
	(Age-25)/10*midinc							-0.01			
	(Age-25) ^{2}/100*midinc							0.001			
	(Age-25)/10*highinc							0.005			
	(Age-25) ^{2}/100*highinc							-0.001			
	(Age-25)/10*highsch								0.023		
	(Age-25) ^{2}/100*highsch								-0.002		
	(Age-25)/10*college								0.023		
	(Age-25) ^{2}/100*college								-0.002		
	Financial stress										-0.002
Variance Components:											
Level_1: within-person	σ_{ε}^2	0.0604***	0.0604***	0.0604***	0.0604***	0.0604***	0.0604***	0.0604***	0.0604***	0.0604***	0.0604***
Level_2: In initial status	σ_0^2	0.1461***	0.111***	0.1048***	0.107***	0.1036***	0.1044***	0.104***	0.1065***	0.1028***	0.1009***
In rate of change	σ_1^2	0.0008***	0.0073***	0.0007***	-0.0016***	0.0007***	0.0007***	0.0007***	0.0007***	0.0007***	0.0007***
Covariance	σ_{01}	-0.0013*	-0.0016***	-0.0014**	0.007***	-0.0014**	-0.0014**	-0.00134***	-0.0015***	-0.0014**	-0.0013**
Deviance (-2 log likelihood)		8019.2	7128.3	6977.4	7022.9	6939.3	6986	6956.1	7010.3	6920.9	6871.2
Degree of freedom		6	10	14	14		20	22	22	24	22
AIC		8031.2	7148.3	7005.4	7050.9	6975.4	7026	7000.1	7054.3	6968.9	6915.2
BIC		8068.4	7210.2	7092.1	7137.6	7086.8	7149.9	7136.4	7190.6	7117.5	7051.4

Note:
a: low income is reference category
b: elementary school is reference category
† p<0.10; * p<0.05; ** p<0.01; *** p<0.001

Table 4.7 (Continued)

		Model 11	Model 12
Fixed Effects			
Initial status (π_{0i})	Intercept	-0.588***	-0.561***
	(Age-25)/10	0.285***	0.259***
	(Age-25) ^{2}/100	-0.019***	-0.017***
	Mid income	0.282*	0.303*
	High income	0.452*	0.431*
	(Age-25)/10*midinc	-0.136*	-0.143**
	(Age-25) 2/100*midinc	0.013**	0.013**
	(Age-25)/10*highinc	-0.216**	-0.206**
	(Age-25) 2/100*highinc	0.020**	0.020**
	High school	-0.063***	-0.036*
	College	-0.088***	-0.047*
	No drink		0.042**
	Heavy drink		-0.015
	Under weight		-0.024
	Over weight		0.170***
	Physical activity		-0.036***
	Smoking		-0.017
	Sleep<7 hours		0.029†
	Sleep>9 hours		0.035†
	Financial stress	0.042***	0.036***
	Black	0.079***	0.050***
	Employed	0.006	-0.012
	Retired	0.102**	0.086**
	Other	0.149***	0.119***
	Female	0.001	-0.021
Rate of change (π_{1i})	Intercept	-0.036**	-0.038**
	(Age-25)/10	0.020***	0.018***
	(Age-25) ^{2}/100	-0.002***	-0.002***
	Mid income	0.006*	0.006*
	High income	0.004	0.005
	No drink		0.005*
	Heavy drink		0.002
	Under weight		-0.003
	Over weight		0.002
	Physical activity		0.001
	Smoking		0.004†
	Sleep<7 hours		0.002
	Sleep>9 hours		0.004
	Financial stress	-0.001	-0.002
	Black	0.004	0.002
	Employed	-0.002	-0.002
	Retired	-0.001	-0.001
	Other	-0.008†	-0.008†
	Female	-0.004*	-0.004†
Variance Components:			
Level_1: within-person	σ_{ε}^2	0.0604***	0.0604***
Level_2: In initial status	σ_{0}^2	0.09762***	0.0907***
In rate of change	σ_{1}^2	0.0007***	0.0007***
Covariance	σ_{01}^2	-0.0012**	-0.0013**
Deviance (-2 log likelihood)		6763.8	6531.1
Degree of freedom		32	48
AIC		6827.8	6627.1
BIC		7026	6924.4

Note:
a: low income is reference category
b: elementary school is reference category
† p<0.10; * p<0.05; ** p<0.01; *** p<0.001

Table 4.8: Mixed-effects Regression of Chronic Diseases on Time Variant Income, Financial Stress, and Health Risk Behaviors

		Model 1	Model 2	Model 3	Model 4
Fixed Effects					
Initial status (π_{0i})		0.344***	0.394***	0.389***	0.337***
Time (π_{1i})		0.013***	0.009***	0.010***	0.009***
Income			-0.022***	-0.020***	-0.018***
Income*time			0.002***	0.001***	0.001***
financial stress				0.013**	0.012**
No drink					0.073***
Heavy drink					-0.014
Under weight					0.01
Over weight					0.102***
Physical activity					-0.041***
No smoking					-0.066***
Variance Components:					
Level_1: within-person	σ_{ε}^2	0.0604***	0.0612***		0.0641***
Level_2: In initial status	σ_{0}^2	0.1461***	0.1369***		0.1179***
In rate of change	σ_{1}^2	0.0008***	0.0007***		0.0007***
Covariance	σ_{01}^2	-0.0013*	-0.0011*		-0.0011**
Deviance (-2 log likelihood)		8019.2	7915.8		7630.5
Degree of freedom		6	8		15
AIC		8031.2	7931.8		7660.5
BIC		8068.4	7981.3		7753.4

† $p<0.10$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

5. Age, SES and Chronic Diseases (Combination of Count and Duration as the Measurement of Health) in the 8-year Study Period

This chapter explores the association between age, SES and health by using the combination of count and duration of chronic diseases as the measurement of health. Models from chapter 4-II-4 are applied parallel to indicate the advantages associated with the combination measurement.

Since no information about the duration of chronic diseases is available at the beginning of the study in ACL (in 1986), the number of chronic diseases is used as measurement of health at wave 1. Therefore, results from models at the beginning of the study are same as those findings in chapter 4-II-4. Briefly, older age cohorts have higher level of chronic diseases at the beginning of the study; however, the gap between two cohorts diminishes as age cohort increases. In general, people from higher SES (measured by either income or education) groups are healthier than those from lower SES groups. Furthermore, the effect of education and income are accumulative. The effect of income and age cohort is interactive such that health gap between higher income groups and lower income ones diminishes as age cohort increases. Financial stress is positively associated with the number of chronic diseases, and it mediates part of income's effect on health. Moderate drinking and physical activity benefit health; over weight is associated with high level of chronic diseases.

The major tasks in chapter 4-II-5 are to explore how the new measurement (using combination of count and duration of chronic diseases as measurement of health) leads to

different findings about the rate of change in health over time, compared to count measurement in chapter 4-II-4.

Unconditional growth model

Model (1) in table 4.9 on page 144 is unconditional growth model, which suggests the overall health trajectory over 8-year study period. Coefficients at the initial status and the rate of change suggest that on average people have some level of chronic diseases at the beginning of ACL study and their health decline over time as well. These findings are consistent with those in model (1) in table 4.7 (where the number of chronic diseases was used as measurement of health). The variance in initial status suggests varieties in health across individuals at the beginning of the study. The variance in the rate of change indicates that people have different rates of change in health over time. Findings in variances are consistent with those in model (1) in table 4.7. However, covariance (.0296***, $p<.001$) between the initial status and the rate of change suggests a different story about the relationship between the health at the beginning of the study and the rate of change over time. The positive covariance suggests that high level of chronic diseases at the beginning of the study is associated with higher rate of change in health over time. That is, people of poor health at the beginning of the study tend to decline faster than healthy people over time.

In chapter 3, I argued that the declination of health is gradual over time. The occurrence of a chronic disease is actually the result of accumulative declination of health. Will people's health continue to decline after a chronic disease occurs? In other words, is the occurrence of a chronic disease the end of declining process? Using the number of

chronic diseases as measurement of health suggests that people's health will not get worse if no new chronic disease occurs. However, it is common sense to argue that people's health will continue to decline after the occurrence of a chronic disease if people do not do any thing about it. Findings from the unconditional growth model suggest that people's health is likely to continue declining even though their the number of chronic diseases may be constant over time.

Age cohort, SES and the rate of change over time

Model (2) through model (12) in table 4.9 examines the effect of age cohort, income, education, financial stress, and various combinations between income, education and age cohorts.

There is a curvilinear relationship between age cohort and the rate of change, which suggests that the rate of change in health will increase as age cohort increases; however the acceleration rate of change decreases. Does the age cohort effect at the beginning of the study actually reflect the effect of age? This would be tested in Chapter 4-II-6, where age at a specific study wave is used as the measurement of passage of time.

Both income and education are negatively associated with the rate of change in chronic diseases over time, and their effects are cumulative, instead of interactive. High income is interactive with age cohort, while mid income is not. This suggests that the gap between mid and low income in the rate of change is constant across age cohorts; however, the gap between high and low income in the rate of change increases as age cohorts increases, although this increase rate diminishes. This result suggests that income may have different effect on health at different age stages. The effect of education on the

rate of change is constant across age cohorts, which suggests a consistent effect of education on health at different life stage.

Financial stress is positively associated with the rate of change over time. People with higher financial stress have higher rate of change in health. Moreover, financial stress mediates part effect of income on the rate of change. The gap in the rate of change between mid and low income decrease by 23.1% (based on the comparison between model (3) and model (10)), and the gap between high and low income decreases by 23.3% after including financial stress.

The above findings suggest that people with high income and education will have lowest rate of change in health over time, while people with low income and education will have highest rate of change in health over time. In another words, people from higher SES will age slower than those from lower SES groups. This finding is consistent with that in chapter 4-II-2 in that people from different SES groups have different health trajectories. The health among low SES groups declines at faster speed over time, while high SES groups age at a slower speed. For the perspective of life course, the health gap between low and high SES groups will get larger until the fragility of human body becomes predominant.

Health risk behaviors, control variables and the rate of change

Model (11) and (12) explore the effect of control variables and health risk behaviors over the rate of change. As we can see from model (11) that income doesn't significantly affect the rate of change, the effect of financial stress on the rate of change becomes marginally significant.

Black have higher rate of change than non-Black, which suggests that Black age faster than non-Black. Employment status does not associate with the rate of change over time; however, the disable, and retired have higher rate of change. The higher rate of change of retried may reflect the effect of fragility of human body on health.

Model (12) includes health risk behaviors. The effect of income and financial stress become insignificant. No drink, over weight is positively associated with the rate of change, while physical activity is negatively associated with the rate of change. The effect of race decreases by 26.1% after controlling the effect of health risk behaviors.

The findings from model (12) suggest that SES disparities in the rate of change are largely due to the inequality in the distribution of health risk behaviors. Lower SES groups are more likely to adopt health risk behaviors, which harm their health over time.

[TABLE 4.9 ABOUT HERE]

Time variant SES (measured by income), health risk behaviors, and health over the life course

Table 4.10 on page 146 presents results based on time variant income and health risk behaviors. It is very likely that people's income and health risk behaviors may change over time. However, it is not clear whether changes in income and health risk behaviors will lead to changes in health. Models in table 4.10 explore this issue.

Results suggest that change in income is associated with the change of health. Increased income is related with better health, which is consistent with previous findings in chapter 3 where functional limitation is the measurement of health. However, the

effect of income doesn't interact with time, which suggests that the effect of income is constant over time.

Model (5) in table 4.10 indicates that changes in health risk behaviors are associated with changes in health. People who drink (whether moderate or heavily) are better off in health than those who do not drink at all. Underweight people are not significantly different from normal weight ones in health; however, overweight people are worse on health than normal weight ones. This suggests that people, who used to be overweight, will have better health if they become normal weight.

Physical activity is positively associated with health. Increasing physical activity is related with better health. Smoking is associated with worse health. People, who used to smoke, will have better health if they quit smoking.

The effect of income on health decreases by about 13% when health risk behaviors are added in the model. This suggests that health risk behaviors partially mediate the effect of income; however, large part of effect of income remains.

[TABLE 4.10 ABOUT HERE]

SUMMARY

This chapter explores SES disparities in health during the 8-year study period in ACL. The combination of count and duration of chronic diseases as measurement of health over time has been employed. The effects of both time invariant and time variant SES measurement and health risk behaviors have been explored.

Findings from this chapter suggest that people with higher SES (measured either by income or education) are generally healthier than those with lower SES across all age

groups at the beginning of ACL study; moreover, higher SES people have slower rate of change in health over time than lower SES ones, although the gap in the rate of change diminishes over time. Therefore, SES disparities will become large in the middle life and diminish to some extend at the late stage of life due to the fragility of human body.

Financial stress and health risk behaviors partially mediate the effect of SES on health at the beginning of the study. Findings from the initial status suggest the robust effect of SES over health. In the rate of change, the effect of SES (measured by income) is completely mediated by health risk behaviors and race. This suggests that health risk behaviors are responsible for the accelerated aging process among low SES groups.

Findings, in chapter 4-II-4 using the number of chronic diseases as measurement of health, suggest that lower SES groups have worse health at the beginning of the study; they, however, have lower rate of change in health over time. As a result, SES disparities in health over the life course will diminish at the late stage of life.

However, findings in this chapter suggest a different landscape about SES disparities in health over time. Compared to their higher SES counterparts, lower SES groups have worse health at the beginning of the study (which is consistent with those in chapter 4-II-4), they also have higher rate of change over time. Thus, lower SES groups face a double jeopardy over the life course in that their health are worse on a daily base and they are aging faster. Findings about SES disparities in health over time in this chapter are consistent with those in chapter 4-II-2 (where functional limitation is the measurement of health).

Table 4.9: Mixed-effects Regression of Chronic Diseases (Combination of Count and Duration) on Age, Education, Income, and Interaction Terms.

		Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10
Fixed Effects											
Initial status (π_{0i})	Intercept	0.3625***	-0.434***	-0.383***	-0.222**	-0.261***	-0.555*	-0.699***	-0.251***	-0.593*	-0.696***
	(Age-25)/10		0.192***	0.238***	0.181***	0.223***	0.318***	0.369***	0.222***	0.362***	0.340***
	(Age-25) ^{2}/100		-0.007**	-0.013***	-0.008**	-0.012***	-0.021**	-0.025***	-0.012***	-0.026***	-0.022***
	Mid income[a]			-0.141***		-0.110***		0.306†		0.186	0.283†
	High income[a]			-0.226***		-0.181***		0.536*		0.266	0.463*
	High school[b]				-0.140***	-0.097***	0.193		-0.102***	0.051	
	College[b]				-0.213***	-0.131***	0.371		-0.206***	0.091	
	Income($10,000)								-0.051**		
	Highsch*income								0.002		
	College*income								0.031†		
	(Age-25)/10*midinc							-0.193**		-0.143*	-0.170**
	(Age-25) ^{2}/100*midinc							0.018**		0.014*	0.016**
	(Age-25)/10*highinc							-0.318***		-0.201*	-0.026**
	(Age-25) ^{2}/100*highinc							0.030***		0.020*	0.025**
	(Age-25)/10*highsch						-0.141			-0.057	
	(Age-25) ^{2}/100*highsch						0.013†			0.005	
	(Age-25)/10*college						-0.248*			-0.083	
	(Age-25) ^{2}/100*college						0.023***			0.008	
	Financial stress										0.052***
Rate of change (π_{1i})	Intercept	0.073***	-0.163***	-0.161***	-0.135***	-0.143***	-0.150*	-0.223***	-0.142***	-0.157***	-0.162***
	(Age-25)/10		0.074***	0.081***	0.072***	0.078***	0.079***	0.104***	0.079***	0.080***	0.079***
	(Age-25) ^{2}/100		-0.005***	-0.006***	-0.005***	-0.006***	-0.006**	-0.008***	-0.006***	-0.006***	-0.006***
	Mid income			-0.013**		-0.009†		0.053		-0.014**	-0.010*
	High income			-0.030***		-0.022***		0.121*		-0.030***	-0.023***
	High school[b]				-0.015**	-0.010†	-0.022		-0.008		
	College[b]				-0.030***	-0.020**	0.031		-0.024**		
	Income($10,000)								-0.052		
	Highsch*income								-0.001		
	College*income								0.002		
	(Age-25)/10*midinc							-0.024			
	(Age-25) ^{2}/100*midinc							0.002			
	(Age-25)/10*highinc							-0.060**			
	(Age-25) ^{2}/100*highinc							0.006*			
	(Age-25)/10*highsch						0.002				
	(Age-25) ^{2}/100*highsch						-0.001				
	(Age-25)/10*college						-0.026				
	(Age-25) ^{2}/100*college						0.003				
	Financial stress										0.005**
Variance Components:											
Level_1: within-person	σ_{ε}^2	0.0702***	0.0702***	0.0702***	0.0702***	0.0702***	0.0702***	0.0702***	0.0702***	0.0702***	0.0702***
Level_2: In initial status	σ_0^2	0.1701***	0.1315***	0.1246***	0.1267***	0.123***	0.1261***	0.1237***	0.1231***	0.1221***	0.1215***
In rate of change	σ_1^2	0.0081***	0.0067***	0.0067***	0.0066***	0.0065***	0.0066***	0.0066***	0.0065***	0.0066***	0.0066***
Covariance	σ_{01}^2	0.0296***	0.0228***	0.0219**	0.0221***	0.02166***	-0.022***	0.0066***	0.0216**	0.0217**	0.02157***
Deviance (-2 log likelihood)		12412.5	11576	11430.9	11473.2	11395.2	11460.8	11408.5	11396.2	11390.8	11367.8
Degree of freedom		6	10	14	14	18	22	22	20	24	20
AIC		12424.5	11596	11458.9	11501.2	11431.2	11504.8	11452.5	11436.2	11438.8	11407.8
BIC		12461.6	11657.9	11545.6	11587.9	11542.6	11641	11588.7	11560	11587.4	11531.6

Note:
a: low income is reference category
b: elementary school is reference category
† p<0.10; * p<0.05; ** p<0.01; *** p<0.001

Table 4.9 (Continued)

		Model 11	Model 12
Fixed Effects			
Initial status (π_{0i})	Intercept	-0.582***	-0.552***
	(Age-25)/10	0.288***	0.260***
	(Age-25) ^{2}/100	-0.019***	-0.017***
	Mid income	0.254	0.274†
	High income	0.348†	0.343†
	(Age-25)/10*midinc	-0.132*	-0.138*
	(Age-25) 2/100*midinc	0.013*	0.013*
	(Age-25)/10*highinc	-0.180*	-0.175*
	(Age-25) 2/100*highinc	0.017**	0.017*
	High school	-0.075***	-0.048*
	college	-0.094***	-0.054*
	No drink		0.044**
	Heavy drink		0.017
	Under weight		-0.029
	Over weight		0.187***
	Physical activity		-0.034***
	Non-smoking		-0.018
	Sleep<7 hours		0.031†
	Sleep>9 hours		0.034†
	Financial stress	0.043***	0.037***
	Black	0.082***	0.053***
	Employed	0.016	-0.004
	Retired	0.114**	0.096**
	Other	0.174***	0.142***
	Female	0.004	-0.017
Rate of change (π_{1i})	Intercept	-0.157***	-0.152***
	(Age-25)/10	0.076***	0.071***
	(Age-25) ^{2}/100	-0.006***	-0.005***
	Mid income	-0.003	0.001
	High income	-0.08	-0.005
	High school	-0.007	-0.002
	college	-0.016	-0.008
	No drink		0.012**
	Heavy drink		-0.003
	Under weight		-0.006
	Over weight		0.036***
	Physical activity		-0.005**
	Non-smoking		-0.003
	Sleep<7 hours		-0.003
	Sleep>9 hours		0.012*
	Financial stress	0.003†	0.002
	Black	0.023***	0.017***
	Employed	-0.002	-0.005
	Retired	0.019*	0.015†
	Other	0.009	0.003
	Female	0.001	-0.004
Variance Components:			
Level_1: within-person	σ_{ε}^2	0.0701***	0.0701***
Level_2: In initial status	σ_0^2	0.1158***	0.1085***
In rate of change	σ_1^2	0.0064***	0.0061***
Covariance	σ_{01}^2	0.0209***	0.019**
Deviance (-2 log likelihood)		11219.3	11015.6
Degree of freedom		32	46
AIC		11283.3	11115.6
BIC		11481.5	11425.2

Note:
a: low income is reference category
b: elementary school is reference category
† p<0.10; * p<0.05; ** p<0.01; *** p<0.001

Table 4.10: Mixed-effects Regression of Chronic Diseases (Combination of Count and Duration) on Time, Variant Income, Financial Stress, and Health Risk Behaviors.

		Model 1	Model 2	Model 3	Model 4	model 5
Fixed Effects						
Initial status (π_{0i})		0.3625***	0.396***	0.399***	0.392***	0.354***
Time (π_{1i})		0.073***	0.075****	0.073***	0.075***	0.073***
Income ($10,000)			-0.015***	-0.016***	-0.015***	-0.012***
Income by Time				0.001		
Financial stress					0.014**	0.012*
No drink						0.052***
Heavy drink						-0.009
Under weight						-0.005
Over weight						0.076***
Physical activity						-0.038***
No smoking						-0.051***
Variance Components:						
Level_1: within-person	σ_{ε}^2	0.0702***	0.0709***	0.0710***	0.0709***	0.0724***
Level_2: In initial status	σ_0^2	0.1701***	0.1633***	0.1627***	0.1629***	0.1458***
In rate of change	σ_1^2	0.0081***	0.0079***	0.0079***	0.0079***	0.0079***
Covariance	σ_{01}^2	0.0296***	0.0289***	0.0289***	0.0289***	0.0269***
Deviance (-2 log likelihood)		12412.5	12353	12351.9	12341.7	12196.3
Degree of freedom		6	7	8	8	14
AIC		12424.5	12367	12367.9	12357.7	12224.3
BIC		12461.6	12410.3	12417.4	12407.2	12311

† $p<0.10$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

6. Age, SES and Health over the life course

Prior chapter (Chapter 4-II-5) explores Age, SES and health within 8 year study period, using a combination of count and duration of chronic diseases as the measurement of health over time. This chapter expands the findings in chapter 4-II-5 by applying the Multi Cohorts Accelerated Longitudinal Design (MCALD) to explore Age, SES and health from the perspective of life course. This chapter aims to explore if findings about age, SES and health in chapter 4-II-5 actually represent health trajectory over the whole life course.

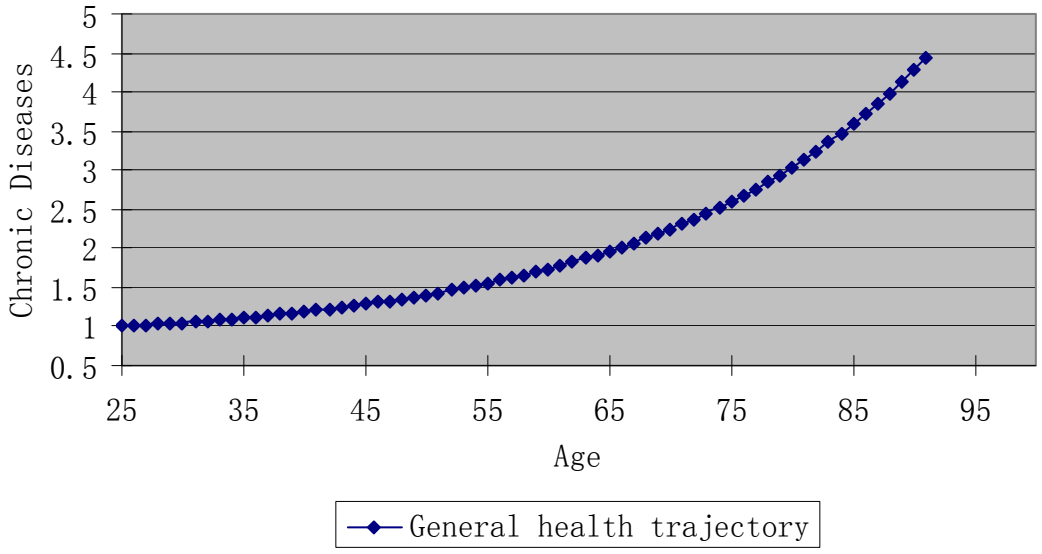
The Multi Cohorts Accelerated Longitudinal Design (MCALD) has been explained in the chapter 3, and the application of MCALD has been shown in chapter 4-II-2, where functional limitation over time is the dependent variable. In this chapter, the combination of count and duration of chronic diseases as a measurement of health is treated as the dependent variable, age at specific study period is the measurement of passage of time, and all principles and assumptions about MCALD are applied.

Age and Health Trajectory

Model (1) in table 4.11 on page 161 is unconditional growth model, in which age is the only predictor for health. The unconditional growth model presents a general health trajectory over the life course. The instant rate of change is positively significant at $p<0.001$, which suggests that the instant rate of change will increase by 8.1% as age increases by 10 years. Positive acceleration rate at $p<0.001$ suggests that the acceleration rate in health will increase by 2.2% as age increases by 10 years. The above coefficients suggest that people's health will decline as they age, and such declination process is accelerated. The unconditional growth model indicates an accelerating downward trend in health over the life course.

Figure 4.9 on page 150 illustrates the predicted health trajectory over the life course based on unconditional growth model. As we can see the declination in health is accelerated over the life course. In general, people's health decline by 55% from age 25 to 55; however, from age 55 to 85, the declination in health is about 130.9% (The comparison is calculated by the predicted health at the given age). As illustrated in the figure, the aging process starts at young age, and the decline rate is accelerated until the late stage of life, which suggests that aging process accelerates toward the late life stage.

Figure 4.9: Health Trajectory over the life course



SES and Health Trajectory over the life course

Model (2) through (9) in table 4.11 on page 162 explores the effect of income, education, financial stress, health risk behaviors, and various combinations of variables on health trajectory over the life course. As we can see that there is no SES disparities in health at age 25. However, there are differences in both the instant rate of change and the acceleration rate across different SES groups. Insignificant effect of SES at age 25 suggests the robustness of human body at early age such that the effect of SES on health becomes latent. This finding is consistent with that in chapter 3 when functional limitation is dependent variable.

Model (2) includes categorized income as the only predictor of health trajectory, which explores the overall effect (both direct and indirect) of income on health trajectory.

People from high income have significantly lower instant rate of change at $p<0.001$ than those from low income. With each ten years' aging, low income groups will have 15.9% higher in the instant rate of change than those from high income. However, there is no significant difference between mid and low income in the instant rate of change. There is continuous debate in sociology about gradient versus threshold effect associated with SES (Blane 1995; Siegrisst and Marmot 2006; Marmot and Wilkinson 1999; Adler and Ostrove 1999). Social gradient hypothesis suggests that the association between SES and health is gradient so that people's health will change corresponding to each dollar's change in SES (Blane 1995; Siegrist and Marmot 2006; Marmot and Wilkinson 1999; Adler et al. 1994), while threshold hypothesis argues that increased income below the poverty line would contribute to improve health, however, above the poverty line, increasing income will not significant contribute to improve health (Adler and Ostrove 1999). One advantage of categorized income is that we can identify threshold effect. The above result suggests that there is a threshold effect of income on the instant rate of change associated with age in that the effect of increased income will not significantly impact health until it reaches high level of income, which is $30,000 in ACL study.

In the acceleration rate, people from higher income have higher acceleration rate than those from low income groups. As we can see that the gap in the acceleration rate between mid income and low income is 0.015 at significant level of $p<0.05$, and that between high income and low income is 0.036 at $p<.001$ The gap in the acceleration rate will increase as income gap increases, which indicates a gradient effect of income on the acceleration rate.

The above findings are illustrated in Figure 4.10 on page 153 (which is based on model (2)). As we can see from the figure, the health trajectories between mid and low income are very close to each other for most of the life course; however, these two trajectories start to diverge after age 75 due to higher acceleration rate for mid income groups. People from mid income groups will have worse health than those from low income groups in the late life stage.

Health trajectories between high and low income indicate that the gap in health between them starts to cumulate ever since age 25 due to significant instant and acceleration rate of change in age; it becomes significant during age of 30's, peak during age of 50's, decline in late 60's, and cross over after age 70.

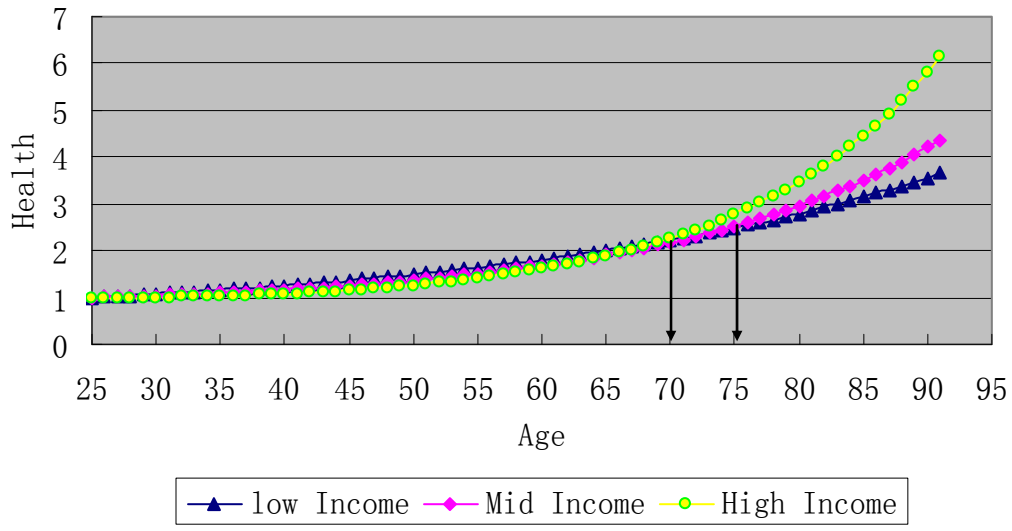
Findings from model (2) are partially consistent with those indicated by House et al. (1994) in that socioeconomic disparity in health becomes increasingly differential at middle age, while it diminishes until later old age (House et al. 1994). House et al. (1994) suggests no SES disparities in health in later old age; however, this study identifies a cross over in health between high and low SES groups. People from low SES groups are likely to have better health than those from high SES groups at later old age.

House et al. (1994) argued that the largest SES disparities in health in the middle life are largely due to the greater exposure of health risk behaviors among low SES groups. However, this study argues that the largest disparities in health between low and high SES groups in the middle age are largely due to timing differences in the aging process. Low SES groups start their declination process at young age, while high SES ones

postpone this process at late middle life. Such time differences lead to the observed SES disparities in health over the life course.

Thornton and Nam (1968) have suggested that the crossover is to be expected if mortality works differentially to eliminate those of a population who are less fit. For advantaged groups, mortality is lower than that in disadvantage groups in early years. It is presumed that it will consist of more less fit individuals in older age categories. In turn, advantaged groups are presumed to contain higher proportions of weak individuals at older ages.

Figure 4.10: SES Disparities in Health Trajectories

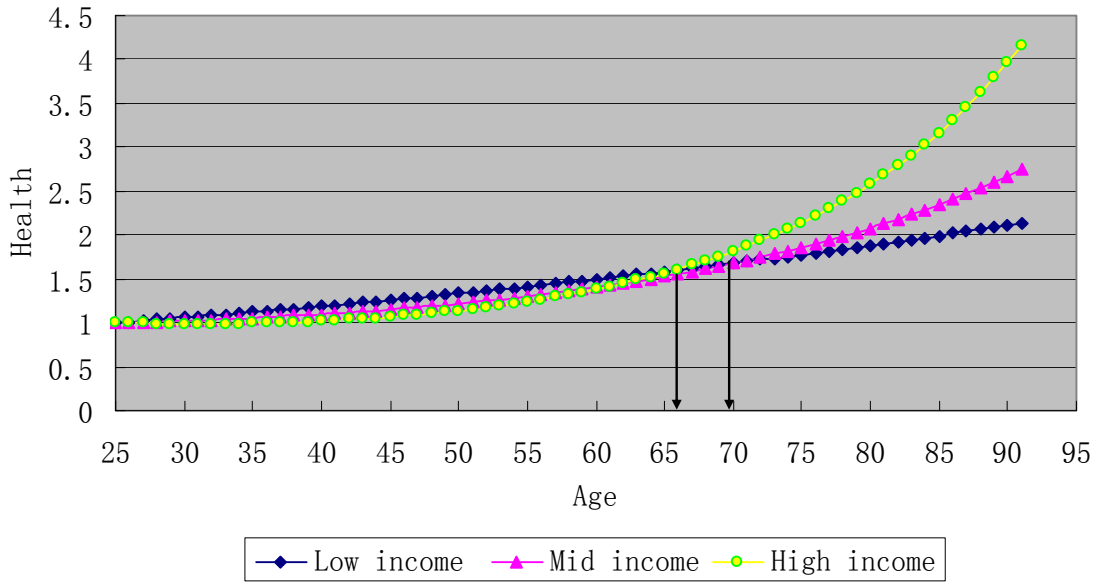


Model (3) through (5) examines various combinations of education and income as predictors of health trajectory over the life course. As we can see that education doesn't significantly affect either instant rate of change or acceleration rate (as shown at model (3) in table 4.10). It doesn't interact with income in affecting health trajectory either (as shown in model (5)). However, education does enhance the effect of income on health

trajectory. Model (4) indicates that the magnitude of income on both instant rate of change and acceleration rate goes up when education is held constant. The gaps in the instant rate of change get even large (in favor of high income) among different income groups with same education (the gap between mid and low income becomes significant and the magnitude between high and low income increases); However, the acceleration rate is in favor of low income groups such that the higher income a person has, the higher acceleration rate will be.

Figure 4.11 on page 154 illustrates the result from model (4). When education is controlled, SES disparities in health start to be significant at early 30's, peak during 40's, decline in age 50's and cross over in late 60's. We can see that the SES disparities in health (illustrated in Figure 4.11) shift to younger age when education is controlled.

Figure 4.11: SES (Income) Disparities in Health Trajectories (Controlling Education)



The results from the above model suggest that biological factors may be more important factors impacting health at both early and late life stage, while SES plays an important role on health in middle life stage. The robustness of human being at early life stage and the fragility of human being at late life stage are likely to cancel out the effect of SES at the corresponding life stages.

Model (6) adds financial stress as mediator of income. At age 25, people with higher financial stress are worse in health. Although financial stress does not directly impact the instant rate of change or acceleration rate, the magnitude of income's effect on the instant rate of change and acceleration rate both decreases. This suggests that SES disparities in health trajectories over the life course may partially due to the fact that people from low SES groups are more likely to experience financial stress in their daily life.

[TABLE 4.11 ABOUT HERE]

Health risk behaviors (time invariant) and health trajectory over the life course

Model (7) through (9) in table 4.11 (continued) on page 161 explores the main effects of health risk behaviors on health trajectory over the life course. Health risk behaviors in this model are treated as time invariant, which assume that people are likely to keep their health risk behaviors over time.

As indicated in these models, only physical activity is significantly associated with instant rate of change and acceleration rate. No other health risk behaviors are significantly related with health trajectory. In general, physical activity is beneficial to health over time; however, such benefit diminishes as time goes by. Therefore, the effect

of physical activity on health is more significant at younger age; it is less significant at late old age.

Drinking, BMI, smoking status, and time of sleep are not significant predictors for instant rate of change and acceleration rate. As indicated in chapter 4-I, these insignificant coefficients do not certainly mean that health risk behaviors are not related with health trajectory over time. It may be due to the fact that people may change their health risk behaviors over time and such changes lead to corresponding changes in health. Therefore, using time invariant variables in a longitudinal study may not be optimal, which may lead to misleading conclusions.

[TABLE 4-11 (Continued) ABOUT HERE]

Time variant variables and health trajectory over the life course

An advantage associated with longitudinal study is that variables are repeatedly measured during different study periods. Thus, we are able to track the changes in these variables and to examine how these changes are associated with health trajectory over time.

As we know that both income and health risk behaviors are likely to changeover the life course. This certainly leads to the question if these changes will lead to corresponding changes in health? The following part of this chapter aims to explore the correlation between changes in income or health risk behaviors and those in health.

Model (1) in table 4.12 on page 163 is unconditional growth, which presents the general health trajectory over the life course. Model (2) through (6) in table 4.12 explores the effect of time variant income and health risk behaviors on health trajectory over time.

Income is negatively associated with chronic diseases over time. As indicated in model (3), each $10,000 increase in income will improve people's health by 10% (this is calculated by combining coefficients of age, income and interaction term of them) as people age every 10 years; however, such benefit diminishes as age increases. This suggests that income is more important for health during young age than in the late stage of life. My theory about this finding is that income gap across SES groups decrease due to retirement in late life, and this decrease leads to less significant effect of income on health. Therefore, other factors in late life stage, such as wealth, may be more important predictors for SES disparities in health.

Including time variant income in to model (1) cuts the acceleration rate associated with age by 27.3% (from 0.022 in model (1) to 0.016). This suggests that income plays an important role in health as people age.

Model (4) includes financial stress as predictor of health. It shows that higher level of financial stress is associated with higher level of chronic diseases. Moreover, financial stress enhances the effect of income on health.

Model (5) and (6) explores the effect of health risk behaviors on health trajectory. As we can see that health will change correspondingly to changes in health risk behaviors. Specifically, people without drinking have worse health trajectory over time than those who drink. People of heavy drinking are worse off than moderate drinking people, although the gap diminishes over time.

Under weight individuals have worse health trajectory over time than those of normal weight; however, the gap between these two groups diminishes as they age. Thus,

people who used to be under weight will have better health trajectory if they become normal weight, although such benefit diminishes toward to late old life. This suggests that the earlier an underweight person change to normal weight, the more benefit he/she will get for their health. People of over weight consistently have worse health over the life course. People, who used to be over weight, will have better health trajectory if they become normal weight, and such benefit is consistent over the life course. Therefore, the longer a person stay over weight, the faster their health will decline.

Physical activity consistently benefits health over time. The harm associated with smoking get larger as people age, which suggests as accelerated aging process for those having long smoking history. As indicated in model (5), those, who stay on non-smoking trace, will be 4.6% healthier than those keeping smoking as they age every 10 years.

In sum, time variant income and health risk behaviors mediate 2.5% instant rate of change associated with age (from .081 in model (1) to 0.079 in model (6)) and 27.3% acceleration rate (from 0.022 in model (1) to 0.016 in model (6)). This suggests that income and health risk behaviors explain some of the decline in health over the life course; however, large part of the decline still remains to be the result of aging per se and other factors that are not explored in this study. Thus, further biological and social research is needed.

In the paragraph 'Health risk behaviors (time invariant) and health trajectory over the life course' in this chapter, health risk behaviors were treated as time invariant variables, and as such most health risk behaviors were not significantly related to health trajectory. However, when health risk behaviors were treated as time variant variables,

they are significantly related to health trajectory. Different findings regarding the effect of health risk behaviors on health trajectory suggest that it is optimal to treat time variant variables as they are; otherwise, findings based on time invariant health risk behaviors may be misleading.

[TABLE 4.12 ABOUT HERE]

Control Variables and Health

Model (7) in table 4.11 (continued) on page 163 includes race, sex, and employment status as predictors of health trajectory over time. Women have lower instant rate of change and higher acceleration rate, which suggests that gender difference in health increases as age increases and the rate of this increase diminishes with time.

Black have higher instant rate than non-Black; however, there is no difference in the acceleration rate across different race. This suggests that black people's health consistently decline faster than non-Black over the life course. Employed people have low instant rate of change and higher acceleration rate, which suggests that employed will have better health over the life course than non-employed.

SUMMARY

This chapter explores Age, SES and Health Trajectory over the life course by using the combination of count and duration of chronic diseases as measurement of health over time. Age at a specific study period is the measurement of passage of time. Both time invariant and variant income, health risk behaviors have been utilized in models.

At age 25, neither SES nor health risk behaviors are significantly associated with health, which largely reflect the robustness of human being at young age. However, SES disparities in health becomes significant during age of 30's, reach the peak during age of 50's, start to decline in late 60's, and cross over after age 70.

The effect of SES is differential on the instant rate of change and acceleration rate associated with age. A threshold effect of income on the instant rate of change is identified, while the effect of income on the acceleration rate is gradient.

Models, utilizing time invariant income and health risk behaviors, indicates that health trajectories between low and high income start to diverge at early age, become significant during middle age, diminishes in age 60's and cross over in age 70's. The SES disparities in the instant rate of change at a given age are largely due to unequal distribution of health risk behaviors among low income groups; however, health risk behaviors do not mediate the effect of SES in the acceleration rate. This suggests different mechanisms through which health risk behaviors mediate the link of SES and health in term of instant rate and accelerate rate associated with age.

Models, utilizing time variant income and health risk behaviors, indicate that health trajectory do change correspondingly to changes in income or health risk behaviors.

Increased income is associated with improved health trajectory over time, although the benefit of increased income diminishes as age increases. Health risk behaviors are associated with worse health trajectory. The effect of health risk behaviors is cumulative with that of income. Health risk behaviors enhance the effect of income on health.

Table 4.11: Mixed-effects Regression of Chronic Diseases (Combination of Count and Duration) on Age, Education, Income, and Interaction Terms

		Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Fixed Effects							
At age 25	Intercept	0.005	0.078	0.042	0.101	0.115	0.082
	Mid income[a]		-0.062		-0.056		-0.062
	High income[a]		-0.053		-0.048		-0.049
	High school[b]			-0.029	-0.036	-0.046	
	College[b]			-0.026	-0.021	-0.078	
	Income($10,000)					-0.035	
	Highsch*income					0.01	
	College*income					0.033	
	Financial stress						0.004*
Rate of change (age-25)/10	Intercept	0.081***	0.137***	0.104	0.115†	0.142	0.074*
	Mid income		-0.072		-0.081†		-0.039
	High income		-0.159**		-0.163**		-0.106†
	High school[b]			-0.003	0.047	0.019	
	College[b]			-0.067	0.014	-0.065	
	Income($10,000)					-0.062	
	Highsch*income					0.031	
	College*income					0.045	
	Financial stress						0.032
Rate of acceleration $(age-25)^2/100$	Intercept	0.022***	0.009**	0.018*	0.0134	0.006	0.019***
	Mid income		0.015*		0.018*		0.013†
	High income		0.036***		0.040***		0.033***
	High school[b]			-0.000	-0.009	-0.002	
	College[b]			0.009	-0.008	0.009	
	Income($10,000)					0.016	
	Highsch*income					-0.008	
	College*income					-0.011	
	Financial stress						0.001
Variance Components:							
At age 25		0.2838***	0.275***	0.2772***	0.2735***	0.2733***	0.2738***
Within-person		0.2035***	0.2047***	0.2048***	0.2047***	0.2047***	0.1998***
Deviance (-2 log likelihood)		16576.1	16529.8	16554.1	16513.2	16511.9	16364
Degree of freedom		5	11	11	17	20	20
AIC		16586.1	16551.8	16576.1	16547.2	16551.9	16392
BIC		16617	16619.9	16644.2	16652.5	16675.8	16478.7

Note:
a: low income is reference category
b: elementary school is reference category
† $p<0.10$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

Table 4.11 (Continued)

		Model 7	Model 8	Model 9
Fixed Effects				
At age 25	Intercept	0.003	0.096	-0.026
	Mid income[a]	-0.056	-0.069	-0.055
	High income[a]	-0.044	-0.059	-0.043
	Financial stress	0.015	-0.002	0.01
	No drink		0.008	-0.001
	Heavy drink		0.007	0.033
	Under weight		0.051	0.036
	Over weight		0.082	0.079
	Physical activity		0.028	0.027
	Non-Smoking		0.037	0.029
	Sleep<7 hours		-0.032	-0.001
	Sleep>9 hours		-0.001	0.021
	Black	-0.085		-0.075
	Employed	0.071		0.07
	Retired	-1.38***		-1.211***
	Other	0.033		0.053
	Female	0.097		0.111†
Rate of change (age-25)/10	Intercept	0.187*	-0.008	0.176†
	Mid income	-0.016	-0.011	-0.008
	High income	-0.071	-0.062	-0.063
	Financial stress	0.015	0.034+	0.015
	No drink		-0.027	-0.027
	Heavy drink		-0.006	-0.095
	Under weight		-0.033	-0.038
	Over weight		0.084	0.073
	Physical activity		-0.041*	-0.036†
	Non-Smoking		-0.008	-0.001
	Sleep<7 hours		0.039	0.029
	Sleep>9 hours		0.066	0.038
	Black	0.117**		0.089*
	Employed	-0.180*		-0.177*
	Retired	0.352*		0.266
	Other			-0.017
	Female	-0.114*		-0.127**
Rate of acceleration $(age-25)^2/100$	Intercept	-0.01	0.026***	-0.012
	Mid income	0.011	0.009	0.009
	High income	0.027**	0.027**	0.026*
	Financial stress	0.003	-0.001	0.002
	No drink		0.011	0.011
	Heavy drink		0.002	0.021
	Under weight		-0.003	0.001
	Over weight		0.002	0.003
	Physical activity		0.007*	0.007*
	Smoking		0.004	0.004
	Sleep<7 hours		-0.007	-0.006
	Sleep>9 hours		-0.013	-0.01
	Black	-0.009		-0.007
	Employed	0.047**		0.046**
	Retired	-0.014		-0.005
	Other	0.001		0.006
	Female	0.021**		0.021**
Variance Components:				
At age 25	σ_0^2	0.2755***	0.2543***	0.2598***
Within-person	σ_ε^2	0.1931***	0.1961***	0.1895***
Deviance (-2 log likelihood)		16164.7	16044.7	15886.1
Degree of freedom		26	32	51
AIC		16222.7	16120.7	15992.1
BIC		16402.3	16356.1	16320.4

Note:
a: low income is reference category
b: elementary school is reference category
† $p<0.10$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

Table 4.12: Mixed-effects Regression of Chronic Diseases (Combination of Count and Duration) on Time Variant Income, Financial Stress, and Health Risk Behaviors

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Fixed Effects						
At age 25 (π_{0i})	0.005	0.003	-0.006	-0.043	-0.068	-0.044
Rate of change(age-25)/10	0.081***	0.080***	0.103***	0.117***	0.096***	0.079*
Rate of acceleration $(age-25)^2/100$	0.022***	0.022***	0.016***	0.015***	0.015***	0.016**
Income ($10,000)		0.001	0.007	0.015	0.017†	0.018†
Income by (Age-25)/10			-0.013†	-0.016*	-0.018**	-0.018**
Income by $(Age-25)^2/100$			0.003**	0.004***	0.004***	0.004***
Financial stress				0.045***	0.041***	0.040***
No drink					0.079***	-0.037
No drink by (age-25)/10						0.064†
No drink by (Age-25)2/100						-0.006
Heavy drink					-0.029	-0.08
Heavy drink by (age-25)/10						0.151†
Heavy drink by (Age-25)2/100						-0.033*
Under weight					-0.021	-0.064
Under weight by (age-25)/10						0.115†
Under weight by (Age-25)2/100						-0.021*
Over weight					0.224***	0.029
Over weight by (age-25)/10						0.093†
Over weight by (Age-25)2/100						-0.008
Physical activity					-0.032***	0.029
Physical activity by (age-25)/10						-0.035*
Physical activity by (Age-25)2/100						0.004
Non-smoking					-0.046*	0.107*
Non-smoking by (age-25)/10						-0.095*
Non-smoking by (Age-25)2/100						0.011
Variance Components:						
At age 25 σ_0^2 σ_0^2	.2838***	.2844***	.2846***	0.2776***	0.2536***	0.2453***
Within-person σ_ε^2 σ_ε^2	.2035***	.2033***	.2024***	0.2036***	0.2065***	0.2058***
Deviance (-2 log likelihood)	16576.1	16575.9	16552.5	16519.2	16366.7	16256.9
Degree of freedom	5	6	8	9	14	27
AIC	16586.1	16587.9	16568.5	16537.2	16394.7	16310.9
BIC	16617	16625	16618.1	16592.9	16481.4	16478.1

† $p<0.10$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

CHAPTER 5: HEALTH AND SES

Selection hypothesis suggests a flow from health to SES, they argue that people's health actually impacts their socioeconomic status (Adler and Ostrove 1999; Bartley et al. 1999; Marmot et al. 1997; Ross and Mirowsky 1995; Stern 1983).

The ideal model to examine selection hypothesis is to select individuals without any chronic diseases at the beginning of a study, follow them over time to collect their health and income. If selection hypothesis is true, those, with occurrence of chronic diseases in following study waves, will significantly have lower income than they did in previous study waves.

Since ACL study does not track respondent individual's annual income over time, and family income in ACL would not be a good variable to test the link from health to individual's SES, I use the data set of "Aging, Status, and Sense of control (ASOC) 1995, 1998, 2001" to test selection hypothesis.

"Aging, Status, and Sense of control (ASOC) 1995, 1998, 2001" is a three-wave panel survey conducted by Mirowski and Ross. It is a survey with fixed 3-year intervals and repeated assessments of the same variables (Mirowski and Ross 2005). The sample size at first wave is 2593; it is 1378 at wave two and 1144 at wave three.

Those respondents without any life threatening chronic disease at wave 1 are selected for the purpose of testing selection hypothesis. Further filter excludes those with missing value in individual annual income at either wave 2 or wave 3. As a result, total 679 respondents are used for analyses.

The purpose of this chapter is to explore if the occurrence of any life threatening chronic diseases at wave two will lead to corresponding income decrease at wave 3. If the answer is yes, then selection hypothesis is supported in that health actually impacts individuals' SES (measured by income). Otherwise, selection into lower SES may not be significant.

Dependent variable is decrease in income. A dummy variable indicating income decrease is created by compared individuals' income at wave 2 with that at wave 3. If income at wave 3 is less than that at wave 2, then income decreases; otherwise, income remains same or increase. There are 146 respondents reporting income decrease at wave 3. The dummy variable of decrease in income represents the mobility of individuals' SES from wave 2 to wave 3. Using decrease in income as the dependent variable helps to examine if health declination leads to downward mobility of SES.

Independent variables include health at wave 2, race, education, age, employment status, and gender.

Health at wave two is measured by any occurrence of heart disease, diabetes, high blood pressure, lung cancer, breast cancer, or other cancer. Total 79 respondents reported that they had been diagnosed or told by doctor that they had one or two of the above chronic diseases. 600 respondents reported no chronic diseases at all. A dummy variable is created as an indicator of health declination. 1 refers to declination, 0 refers to no change.

Race, education level, age, employment status, and gender are measured at wave 3. Race is dichotomized as White versus non-White. Education level is measured by the

years of education. Age is the actual year at wave 3. Employment status is trichotomous, which includes unemployed, retired, or other. Employed is treated as reference. Gender is dichotomized into female versus male.

A logistic regression model is used to explore the log-odds of declination in income based on various combinations of independent variables.

The model could be expressed as follows:

$$Logit(p) = \log(\frac{p}{1-p}) = \beta_0 + \sum_{i=1}^{7} \beta_i * X_i$$

Where p is the probability of income declination at wave 3, X_i are independent variables, including health declination at wave 2, race, education, age, employment status, and gender. This model aims to test if occurrence of any life threatening chronic disease at wave 2 will lead to income decrease at wave 3.

Table 5 on page 168 shows the results from a logistic regression model. Coefficients represent log odds ratio. Model 1 represents the overall effect of health declination at wave 2 (both direct and indirect) on the odds of income decrease. The coefficient is not significant. This suggests that declination in health at wave 2 does not significantly affect the probability of income declination at wave 3. Model 2 adds race, education level, employment status, age, and gender to model 1. Coefficients show that only employment status affects the probability of income decrease at wave 3. Unemployed and retired have higher odds to have lower income at wave 3 compared to employed. Education level, race, age, and gender are not significantly related to the log

odds of income decrease. I also test interaction terms between health declination at wave 2 and race, education, age, gender (not shown here), none of them is significant.

In sum, results from table 5 indicate that health does not significantly impact individuals' income. It is the status of being in or out of employment that impacts individuals' income. Selection hypothesis is not supported in this study.

Table 5: Logistic Regression of the Log-odds of the probability of Income Decrease at Wave 3 on Health at Wave 2, Gender, Race, Age, Education, and Employment Status

	Model 1 Coefficient	Model 2 Coefficient
Intercept	-1.32***	-1.91**
Declined health at wave 2	0.24	0.02
Education		0.03
Age		0.00
Unemployed		0.72**
Retired		0.80***
Other		-0.57
Gender		0.24
White		-0.29
Chi-SQR	706.14	686.61
DF	2	9

* $p<0.05$; ** $p<0.01$; *** $p<0.001$

CHAPTER 6: SUMMARY

SES disparities in health have been the most consistent findings in the United States. This study focuses on the effects of SES on the life course trajectories of health, and investigates the role of health risk behaviors in the association of SES and health. The traditional measurements of health in term of functional limitation and chronic diseases have been applied in this study; moreover, this study introduces a new method to measure health by combining the number and duration of chronic diseases that individuals experience as the measurement of health over time.

Two types of measurement of the passage of time have been used. The first measurement is the passage of years since first interview in ACL. This measurement reflects changes that occurred during the 8-year study period in ACL. The second measurement is the actual age at each interview point. This measurement links changes during the 8-year study period in ACL to the actual age at the corresponding interview point. By doing so, the changes in health over time are linked to the process of aging over the life course.

Both time invariant and variant SES, financial stress, and health risk behaviors are employed in this study. Time variant SES and health risk behaviors are valuable to explore how health trajectory changes correspondingly to the changes in SES or health risk behaviors.

Significance of this study

This study is an addition to the existing literature, and provides a new perspective about the relationship of SES and health over the life course. Five strongest elements are:

1) Exploring SES disparities in health across the adult life span, and providing empirical evidences supporting the statement that SES disparities in health are largest in the middle age.

2) The utilization of both the MLMFC and MCALD, which appear in the current literature. The MLMFC takes into account the effect of time on health, and provides a tool to explore the effect of SES, age, and health risk behaviors on health both at the baseline and rate of change during the study interval. The MCALD expands the findings of the MLMFC, and explores the effects of SES and health risk behaviors on the life course trajectories of health. It offers a tool to examine the age-related change on health over the life course based on data collected over a shorter interval of time. There are debates in the current literature if the age cohort effect on health actually reflects the effect of age. The MCALD provides an efficient method to separate cohort effect from that of age. The findings from this study suggest that the cohort effect on health may actually reflect the effect of age.

Results from both the MLMFC and MCALD identify different rates of change over time across different SES groups, which are largely ignored in the existing literature. Findings of this study suggest that individuals from different SES probably have different aging processes. People are largely "segregated" into different life trajectories by their SES. Lower SES groups not only have worse health at a given point of time (here in this study is at the beginning of the ACL study), they also have a higher rate of change in health over time. In short, lower SES people suffer poor health more often than higher

SES ones, and their health decline faster than their higher SES counterparts over the life course.

3) The combination of number and duration of chronic diseases as a measurement of health over time. In previous research, the number of chronic diseases has frequently been applied as a measurement of health over time. However, there are limitations of the count measurement as indicated in chapter 3. This is the first study that combines number and duration of chronic diseases as a measurement of health over time. Including duration of a chronic disease into the measurement of health provides additional information than the count measurement does.

The combination measurement is able to track the change of health over time even though the number of chronic diseases may remain stable, because the duration of a chronic disease may actually reflect the intensity of a chronic disease. The combination measurement in this study provides a significant addition to the existing literature.

Models using the number of chronic diseases as a measurement of health suggest that higher SES groups have higher rates of health decline than lower SES ones, even though they are healthier at the beginning of the ACL study. As a result, health across different SES groups will eventually converge at some point of life. However, models using the combination measurement indicate that lower SES groups not only have worse health than higher SES ones at the beginning of the ACL study but also have a higher rate of change over the 8-year study interval. Thus, different measurements of health provide different points of view about SES disparities in health over the life course. Results from this study indicate that combining number and duration of chronic diseases as a

measurement of health over time is better than the number of chronic diseases, it is able to capture changes in health over time even the number of chronic diseases may be stable.

4) Identification of "Ceiling effect" in using the number of chronic diseases as a measurement of health over time. Results from the models using the number of chronic diseases as a measurement of health indicate that individuals' health will eventually converge at the late old age, even though lower SES individuals have worse health at the beginning of the study. However, as indicated in this study, it is largely due to the "ceiling effect" of the count measurement, in which there is no much space for those with higher number of chronic diseases at the beginning of the study to develop over time due to the maximum number of chronic diseases is set. Therefore, people of higher SES who have less number of chronic diseases at the beginning of the study have a higher rate of change over time than their lower SES counterparts.

5) Utilization of time variant income and health risk behaviors. Previous studies mainly utilize time invariant income and health risk behaviors (e.g. House et al. 1994, 2005; Lantz et al. 1998, 2001). As Lantz et al. (1998) argued that health risk behaviors are highly stable over time. Based on this argument, They argue that health risk behaviors explain only a modest portion of the association of socioeconomic status and health (Brunner and Marmot 1999; Lantz et al. 2001). This study applies both time invariant and variant health risk behaviors to explore if health risk behaviors significantly mediate the effect of SES on health. Time invariant health risk behaviors don't significantly mediate the effect of SES on health, which is consistent with the argument of Lantz et al. (2001). However, time variant health risk behaviors significantly mediate the effect of SES on

health. Findings in this study suggest that results based on time invariant health risk behaviors are misleading in examining the relationship of health risk behaviors and health over time. The assumption that health risk behaviors are highly stable over time does not hold as Lantz et al. (1998) suggest. This study identifies that SES and health risk behaviors are likely to change over time, and such changes will lead to corresponding changes in health. Using time variant variables to explore how health trajectory changes accordingly to the changes in SES and/or health risk behaviors is another addition to the existing literature.

Major findings

1) People from different SES have different health trajectories over the life course. In the ACL study, lower SES groups generally have poor health at the beginning of the study; they also have a high rate of change over the 8-year interval in ACL. This pattern holds in both the functional limitation and chronic diseases (the combination of number and duration) as measurements of health.

Results from this study partially support the statement of previous studies that socioeconomic disparities in health become increasingly differential at the middle age, while it diminishes until later old age (House et al. 1995; Siegrist and Marmot 2006). House et al. (1995) argue that the greater exposure of lower SES individuals to health risk behaviors in the middle and early old age is a main reason responsible for such pattern. However, this study argues that SES disparities in health over the life course are largely due to the different health trajectories across SES groups. The exposure of lower SES persons to health risk behaviors in the middle age may not be greater than at younger age;

however, the timing differences in aging processes across SES groups lead to the observed SES disparities in health over the life course. Health of lower SES persons start to decline linearly since age 25, while the decline process of higher SES persons postpone to late 30s. As a result, SES disparities in health are not explicit at the early life stage; however, it continues to accumulate to be significant in the middle age, while it diminishes (but not disappear) in the late life when the fragility of human being becomes predominant. Although lower SES individuals are worse off most of their lives, they will have better health than higher SES ones if they could survive to age 70.

Fries (1980) proposed that morbidity could be compressed into a shorter period of time between the increasing age at the onset of disability and mortality. This study indicates that a compression of morbidity may occur among high SES groups because their health start to decline later than lower SES ones, moreover, their rates of change are significantly smaller than those of their lower SES counterparts. For low SES groups, however, compressed morbidity does not seem to exist.

2) The aging process of human beings is not linearly downward. It is curvilinear. Individuals' health may actually get better as they age before they hit the healthiest age point. After that age point, their health is on the downward trend. The healthiest age point varies across SES groups. For lower SES groups, their downward aging process starts at early age, while higher SES groups enjoy healthy life until early old age. Aging processes in the United States are "segregated" based on individuals' SES.

It needs, however, more research on the curvilinear health trajectory over the life course. Since this study imposes quadratic term in the model, it is unclear whether the

model really reveals the reality of health trajectory or whether it is simply due to the parameterization of the models.

3) Health risk behaviors and financial stress are major mediators for the association of SES and functional limitation. Worse health of lower SES groups is largely due to the fact that they are more likely to experience financial stress and to adopt health risk behaviors, which harms their health. In the MCALD models, time variant financial stress expresses 52% of the effect of time variant income on functional limitation, and time variant health risk behaviors express the rest effect of income. In the MLMFC models, time variant financial stress mediates about 15% main effect of time variant income, and completely mediates the interactive effect of income and time. Time variant health risk behaviors explain about 21% main effect of time variant income, and enhance the interactive effect of income and time.

However, findings about the effect of financial stress and health risk behaviors on chronic diseases are complicated. In the MCALD models, Time variant financial stress and health risk behaviors actually amplify the effect of time variant income. In the MLMFC models, time variant financial stress does not mediate the effect of income, it is accumulative with income. Health risk behaviors mediate about 20% of the effect of time variant income on health.

In chapter 2, I proposed that (Hypothesis 1): SES is positively associated with health at the beginning of the ACL study, but is negatively associated with the rate of change in health over time. Major health risk behaviors mediate the relationship between SES and

health both at the beginning of the study and the rate of change over time. This hypothesis is supported by the study.

I also proposed that (Hypothesis 2): People from different SES groups have different health trajectories over the life course in that higher SES groups age at a slower rate, compared to their lower SES counterparts. Major health risk behaviors significantly mediate SES's effect on health trajectories. This hypothesis is also supported.

Limitations of this study

1) No onset information is available for chronic diseases in ACL. As such, the combination measurement of health at the beginning of ACL study is actually a count measurement. Moreover, assigning time interval between two interview points as the duration of chronic diseases may not exactly reflect the actual duration of chronic diseases due to the left censoring. Nevertheless, the combination measurement of health does provide additional information about health over time, which suggests a reasonable way to capture health change given the constant number of chronic diseases.

2) Variables selected in this study may be limited. Some important health-related variables, such as nutrition, family illness history, dietary habits, and biological factors (such as gene), were not included in the ACL study, which limits the ability to exclude the compounding effects from these variables. This study mainly focuses on psycho-social factors to explore SES disparities in health. However, nutrition, family illness history, dietary habits, and biological factors may also play an important role in SES disparities in health. The biological and social factors may interact in influencing SES disparities in health over the life course.

3) Functional limitation and the number of chronic diseases are treated as continuous variables in this study; however, they are actually ordinary. The assumption of normality for Ordinary Linear Square (OLS) models is not completely held. Although OLS is a robust method, which can provide reliable results even though the normality is violated, the conclusions based on models using functional limitation and the number of chronic diseases as dependent variables should be used with caution.

4) This study suggests no cohort effect in the health trajectory over the life course. However, non-significant cohort effect may be due to a short follow-up time period in ACL, which may not be long enough to estimate the cohort effect.

Considerations for future research

More research is called to consistently improve the measurement of health over time. This study shows that different measurements of health in term of chronic diseases may lead to different conclusions. In a longitudinal study, it is crucial to have a measurement that could track health change as people age. Once the health change could be precisely tracked, we will be able to identify responsible factors for such change. By the same token, it is important to correctly measure and utilize the exploratory variables of health over time. Especially, when an exploratory variable changes over time, it is optimal to use it as time variant; otherwise, results may be misleading.

As we know that aging is a process that human beings cannot avoid. It is impacted by both biological and psycho-social factors. Thus, it is valuable that research can combine biological and social factors in explaining SES disparities in health over time.

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