A LONG SHADOW OF CHILDHOOD DISADVANTAGE ON LATE-LIFE HEALTH TRAJECTORIES IN CHINA

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ABSTRACT

Zhenhua Jessica Xu: A Long Shadow of Childhood Disadvantage on
Late-Life Health Trajectories in China
(Under the direction of Yang Claire Yang)

Childhood disadvantage casts a long shadow on health trajectories over the life course, but little is known on how these associations vary by birth cohorts. This paper examines whether childhood disadvantage is associated with health trajectories and how cohort effects moderate the associations among Chinese elderly. Drawn data from the Chinese Longitudinal Healthy Longevity Survey (CLHLS), I find inadequate access to health care, poor nutrition and no schooling during childhood are associated with poor health in late life, while birth in rural areas and having a father with lower occupational status are associated with better health. Cross-over health trajectories by birth cohorts indicate that being born in rural areas and having father in low SES are advantageous for older cohorts but disadvantageous among younger cohorts. Residential stratified analysis suggest that China's rapid economic growth and unprecedented social inequality since late 1970s shaped late-life health distinctively for rural and rural elderly.

TABLE OF CONTENTS

LIST OF TABLES v

LIST OF FIGURES vi

LIST OF ABBREVIATIONS vii

INTRODUCTION 1

Childhood Disadvantage and Health in Late Life 3

Cohort Effects 5

Frailty 6

Hypothesis 8

DATA AND METHODS 12

Analytic Methods 14

RESULTS 18

Childhood Disadvantage Differences within Cohorts 20

Childhood Disadvantage Differences across Cohorts 24

Stratified Sample by Residential Areas and Gender 26

CONCLUSION AND DISCUSSION 33

APPENDIX 41

REFERENCES 44

LIST OF TABLES

Table 1. Sample Descriptions CLHLS 2002 - 2011 (N= 14,617) 19

Table 2. Growth Curve Model Estimates of Childhood Disadvantage with Age Trajectories of Frailty in China 21

Table 3. Estimates of Childhood Disadvantage with Age Trajectories of Frailty by Residential Areas and Sex 29

LIST OF FIGURES

Figure 1. Predicted age trajectories of frailty (N=14,617) 23

Figure 2. Cohort variations in predicted mean levels of frailty index among Chinese elderly 25

Figure 3. Predicted age trajectories of frailty within cohorts by nutrition in rural and urban subsamples 28

Figure 4. Predicted age trajectories of frailty for each birth cohort by nutrition in rural areas. 31

Figure 5. Predicted age trajectories of frailty for each birth cohort by nutrition in urban areas. 32

LIST OF ABBREVIATIONS

ADL	Activities of daily living
CLHLS	The Chinese Longitudinal Health Longevity Survey
FI	Frailty Index
GCM	Growth curve model
IADL	Instrumental activities of daily living
MMSE	The Mini Mental State Examination
SES	Socioeconomic status

INTRODUCTION

Childhood disadvantage casts a long shadow on late-life health (Ferraro, Shippee, & Schafer, 2009; Haas, 2008; Herd, Goesling, & House, 2007; Smith, 2004). Disadvantageous childhood is associated with higher morbidity, disability and mortality risks through biopsychosocial mechanisms (Herd et al., 2007; Link & Phelan, 1995; Pollitt, Rose, & Kaufman, 2005; Poulton et al., 2002; Preston, Hill, & Drevenstedt, 1998; Pudrovska & Anikputa, 2014; Smith, Mineau, Garibotti, & Kerber, 2009). However, few studies have examined cohort variations in the long-term impacts of disadvantaged childhood on late-life health, particularly for less developed countries (McEniry, 2013). Comparing to developed societies, developing counterparts experienced distinctive causes and consequence of morality decline during the twentieth century, which may reflect cohort specific associations between childhood disadvantage and health trajectories in late life for these societies. Furthermore, the association between disadvantageous childhood and health trajectories may vary by gender and residential areas across birth cohorts. Prior studies from less developed countries suggest that life expectancy largely improved in at ages under five or above forty in the past century. Female gained larger life expectance than male, while urban areas gained more rapidly than rural areas (Preston, 1980). This evidence indicates heterogeneity of life expectance that highlight the importance of gender and residential differentials in cohort variations when examining long-term impacts of childhood disadvantage on health trajectories in less developed countries.

In this paper, I draw data from the Chinese Longitudinal Health Longevity Survey (CLHLS) to investigate whether childhood disadvantage is associated with late-life health

trajectories over the life course, and further examine how the associations vary by birth cohorts across gender and residence areas in China. China has been undergoing rapid social, political and economic changes during the past century, providing unique social contexts to examine how childhood conditions exhibit a long-term influence on health trajectories in later life since early1910s. Most previous China-based studies usually treat birth cohorts of early twentieth century as a single birth cohort (Wen & Gu, 2011; Zeng, Gu, & Land, 2007), thus cohort variations regarding the long term effects of dynamic societal changes are less explored. For instance, China experienced a critical public health transition due to modern medical knowledge spreading and accessible clean water in big cities around 1920s. Beijing and other big cities became healthier places to live than the countryside during that period, partially due to improved medical infrastructure, hygiene regulations, sanitation and public health such as renovated sewage system, water supply during 1920s – 1930s that significantly increased life expectancy in urban areas (Campbell, 1997). Medical centers funded by western countries, such as the Rockefeller Foundation's China Medical Board, introduced western institutional health infrastructures, public health programs, and sanitary interventions. These medical centers contributed to the reducing risk of infectious diseases and mortality in big cities (Campbell, 1997).

Considering the long term effects of early life conditions on late health may vary across birth cohorts, this study aims to distinguish cohort variations in the association between childhood disadvantage and health trajectories among Chinese population in early twentieth century. This paper first reviews conceptual life course mechanisms that link childhood disadvantages with later-life health outcomes. Then I discuss the importance of examining cohort variations in long term effects of childhood disadvantage on health, particularly for China.

Finally, I explain measurements of health, frailty index, and advantage of using frailty to illustrate cohort variations in the association of childhood disadvantage with late health trajectories.

Childhood Disadvantage and Health in Late Life

Early socioeconomic status (SES) fundamentally influences health disparities in late life (Dannefer, 2003; Merton, 1988; Phelan, Link, & Tehranifar, 2010; Willson, Shuey, & Elder, 2007). Early life disadvantage as "the first life course risk" and "initial injustice" provoke cumulative effects on health via biological, psychological, social and ecological pathways (Ferraro et al., 2009; Gortmaker & Wise, 1997; Herd et al., 2007; O'Rand, 2006). Extensive evidence suggests that early socioeconomic conditions influence later health trajectories through biopsychosocial mechanisms over the life course (Campbell & Lee, 2009; Elder, 1998; Elo & Preston, 1992; Ferraro et al., 2009; Finch & Crimmins, 2004; Merton, 1968; O'Rand, 2006; Smith et al., 2009). The "long arm" of disadvantaged socioeconomic conditions during childhood are positively associated with physical, psychological impairments and mortality risks in late life (Herd et al., 2007; Link & Phelan, 1995). For instance, low parental education is associated with a high likelihood of physical limitations and impairments, poor mental health, heart disease, hypertension, and increase level of allostatic load, systolic blood pressure and inflammation in late life (Poulton et al., 2002). Childhood residential stability in family and neighborhood is positively associated with good mental health in later life (Bures, 2003). China-based study using the CLHLS data find low childhood SES is associated with higher risk of functional limitations, cognitive impairments, poor self-reported health and mortality (Huang & Elo, 2009; Shen & Zeng, 2014; Wen & Gu, 2011; Zeng et al., 2007).

Hypothetical mechanisms of childhood disadvantage on health in life course paradigm. Life course paradigm provides a framework to understand changing lives in changing contexts. Life course as a concept refers to a sequence or temporal pattern of age-graded events and roles revealing sociological contours of biography, emphasizing dynamics of social contexts and their interaction with human development from birth to death (Elder, Shanahan, & Jennings, 2015). Life course paradigm emphasizes dynamics of socially constructed meaning of age, birth and death over time and place.

Hypothetical mechanisms underlying links between early-life conditions and health outcomes over life course include: critical period model, accumulation of risks model, pathway model, and mobility model (Pollitt et al., 2005; Pudrovska & Anikputa, 2014). Critical period model elaborates a window of time - biological and epidemiological origins of health outcomes deriving from very early stage of life and even before birth – that exerts long lasting, permanent, irreversible, and direct influence on health outcomes in late life (Barker, 1998; Ben-Shlomo & Kuh, 2002; Braveman & Barclay, 2009; Pudrovska & Anikputa, 2014).

Accumulation of risks model emphasizes persistent, additive, overall, and synergistic effects of risks on later health outcomes across multiple life stages (Pudrovska & Anikputa, 2014) rather than a single life stage as critical period model documents. Specifically, initial risks during childhood make one more vulnerable to adverse situations and lasting risk exposures throughout adulthood. At the same time, the adverse adulthood may also increase risks of poor health outcomes in late life. Therefore, both childhood and adulthood disadvantage additively or accumulatively increases the health disparities over the life course. A China-based study shows that inadequate access to medical care during both childhood and late life cumulatively decreases survivorship of oldest old people (Gu, Zhang, & Zeng, 2009).

Pathway model posits that childhood disadvantage is associated with health in late life through mediating factors, for instance, SES and health behaviors in adulthood, such that early-life conditions link health outcomes indirectly. Studies have found that associations of childhood SES with health at advanced ages are indirect and mediated by adulthood occupation, marital status, and health behaviors such as smoking using Chinese population (Shen & Zeng, 2014; Zeng et al., 2007).

Mobility model describes dynamics of socioeconomic positions change over life course, indicating an upward or downward mobility moderates effects of childhood disadvantage on health in late life. In sum, four hypothetical life course models represent potential mechanisms on how childhood conditions are associated with health disparities in late life. I expect the hypothetical models of life course mechanisms may vary by different birth cohorts in China, which may attribute to the rapidly changing Chinese society and increasing social inequality among urban and rural areas over past few decades,

Cohort Effects

From concepts to mechanisms. A cohort is defined as a group of people experiencing life events or social changes at the same period such as a birth cohort or marriage cohort. Cohort effects describe enduring effects of specific life events or social changes among some cohorts that distinguish them from other cohorts. In 1960s, Ryder (1965) asserts cohort as a concept to study social changes, for human being interact with social changes and historical contexts over their life course and the social and historical environments shape features of certain birth cohorts, for example, baby boom cohorts (1946-1962). During the past half-century, research on cohort effects has developed from concepts into mechanisms that how early life experience matters over the life course (Finch & Crimmins, 2004; Preston & Walle, 1978; Ryder, 1965). These

theoretical and methodological developments of cohort analysis address two kinds of research questions: (1) Across birth cohorts, what lead to health disparities, and (2) Within birth cohorts, how the health disparities can happen over life course. In this study, I integrate the two research questions aiming to uncover how early life environments link to late health trajectories within and across birth cohorts.

Cohort mechanisms interpreting health disparities. Cohort effects elaborate formative and cumulative impacts of both early-life conditions and lasting exposures to historical and socioeconomic factors over the life course on subpopulation (Elo & Preston, 1992; Yang, 2009), which may contribute to distinctive health trajectories in late life. Birth cohorts not only acquire coherence from structured social contexts in early life, they also adapt to distinctive societal developments that shape ecological processes of birth cohorts by time and place (Elder & Shanahan, 2006). Research on cohort mechanisms of health disparities emerged in 1970s when mortality was a major health measure. Investigators discovere a cohort specific mortality based on a French urban population study in nineteenth century, suggesting hygienic improvements increase life expectancy of younger birth cohorts rather than whole age groups of the population (Preston & Walle, 1978). Existing literature finds that Chinese birth cohorts of the Great Famine (1959-1962) endure worse health and earn less income than other birth cohorts in later life (Chen & Zhou, 2007).

Frailty

Frailty is clinically defined as a failure to repair damaged cells that lead to tissue and organ damages (Kirkwood, 2005; Rockwood & Mitnitski, 2011). Frailty describes susceptibility to disease and death in aging process of human being. Frailty is depicted as a physiological accumulation of deficits, reducing health reserves as one ages through behavioral, functional,

clinical and biological pathways (Fried, Ferrucci, Darer, Williamson, & Anderson, 2004; Rockwood & Mitnitski, 2011). Conventionally, theoretical concept of frailty as a risk factor of mortality is obtained from many variables using mathematical techniques to investigate patterns of its association with mortality at the population level. The early definition of frailty is fixed at birth and constant throughout the life course (Vaupel, Manton, & Stallard, 1979), thus the variation of frailty as an aging process is unobserved. For the past few decades, however, frailty has integrated clinical perspectives by involving systemic measures of symptoms, disability, disease classification, and physiological disorder among community dwelling older people (Rockwood & Mitnitski, 2011). The recent concept of frailty highlights an entity of health reserves and cumulative deficits, especially the long-term effects of childhood conditions on health outcomes over the life course.

Frailty Index. Frailty index (FI) is widely used to measure the degree of frailty by counting total numbers of self-reported symptoms related to chronic diseases, functional limitations, and mental impairments (Searle, Mitnitski, Gahbauer, Gill, & Rockwood, 2008). FI is regarded as a valid and generalized composite measure of cumulative health disorders and deficits in biological aging processes. FI integrates aging related physiological functions that effectively track heterogeneity of aging processes across individuals and dynamic trends of health reserves across population due to changing environments over time. FI presents a more comprehensive understanding of aging trajectories than a signal dimension of health measure such as self-reported health (Yang & Lee, 2010).

FI as a reliable and valid predictor of mortality risk has its advantages in measuring aging processes. First, accumulative deficits (by summing up total numbers of symptoms related to biological aging) can capture multiple domains of health trajectories within each person over

time. Second, FI represents environment factors relevant to both recovery rate and damage rate of human bodies. Third, age distributions of FI are insensitive to survey design and number of variables composing this index (Mitnitski et al., 2005). FI is widely used to measure frailty cross different populations due to its good availability in health survey. In sum, FI provides a standardized comprehensive geriatric assessment of impairments, disability and comorbidities and offers a valid, reliable and sensitive clinical measure for health (Jones, Song, & Rockwood, 2004).

Age trajectories of Frailty. Age trajectories describe a temporal pattern of roles, experiences and statuses that vary as one ages. The onset and duration exposures to risk factors jointly shape age trajectories over the life course. Risk accumulations, available resources, social contexts, and human agencies also influence age trajectories (Elder & Shanahan, 2006; Ferraro et al., 2009; Hitlin & Elder, 2007). Age trajectories of frailty represent age-graded patterns of vulnerability to cumulative deficits such as chronic diseases, functional limitations, physical disabilities, and mental impairments which may vary across birth cohorts. Age trajectories of frailty are embedded in peculiar historical contexts and social institutions for each birth cohort. This paper gives special attention to integrating cohort variations into the biological aging process in social contexts of China.

Hypothesis

Social institutions and historical contexts. The importance of social institutions is a basic principle of life course theory. Sociologists and social epidemiologists have emphasized that socially structured conditions are key to understand how aging processes of individuals are related to macrostructural circumstances over time and place (Mayer, 2004; Mayer, 2009; Moen, 2013). Chinese unique historical, social, economic and political environments institutionalize

early life conditions of population and shape potential mechanisms on how childhood disadvantage are associated with health trajectories over the life course. Few studies, however, focus on the association between childhood disadvantage and health trajectories over the life course in Chinese society. Given China's unique social contexts, I purpose the first hypothesis:

> *Hypothesis 1. Childhood disadvantage increases mean level of frailty index across all ages within each cohort.*

Prior longitudinal studies have found that educational differentials in health disparities converge across successive cohorts (Chen, Yang, & Liu, 2010). Income gap in self-reported health trajectories diverges across age for birth cohorts in 1930s, but it converges for birth cohort in 1960s (Chen et al., 2010). For earlier birth cohorts (1900 – 1930), however, the association between early-life conditions and health trajectories is less discussed. Birth cohorts before 1910s experienced the republican revolution and early residential inequality from 1901 to 1911. The economic inequality between urban and villages had increased during 1910s in China, partially due to economic growth in newly opened treaty ports around costal, accelerated urban developments around riverine areas, and renovated public sanitary infrastructures such as water supply, estimations of Health Stations in big cities (Campbell, 1997; Fairbank & Goldman, 2006), whereas the self-reproductively agricultural economy in villages was left behind. Birth cohorts after 1910s experienced establishment of the Republic of China in their childhood and exposed to implications of economic and educational policies on individual, family, residential regions and even entire country. Cohort variations in age trajectories of frailty may associate with the long arm of differential social conditions during childhood. The second hypothesis examines whether the impacts of early childhood conditions on frailty in late life vary across birth cohorts.

Hypothesis 2. The gap in age trajectories of frailty between disadvantaged and advantaged childhood converges in recent birth cohorts.

Health inequalities in urban and rural areas. Household registration (*Hukou*) system launched in 1950s is a residential permit system in China. *Hukou system* divides Chinese population into agricultural and non-agricultural identity, which is still in place for nowadays. *Hukou* system initially intended to limit the demographic mobility from rural to urban side since the first Five -Year Plan (1953 – 1957) of industrialization. Rural residents have fewer opportunities to receive formal education and public health service than urban counterparts. Rural residents not only means spatial differentials in dwellings but also means less life chances in upward social mobility than those in urban side (Wu & Treiman, 2004). In traditional agrarian Chinese society, administration and market in rural areas created hierarchal social structures, including gentries, peasants, inter-middle merchants (Skinner, 1985). In modern China society (after 1950s), the gentry-peasant hierarchal structure has no longer existed. However, as China's precedential economic growth since 1978, the social inequality between rural and urban areas increases as well. Scholars describe China as "one country but two societies" (Whyte, 2010). For instance, education and health care resource become more privatized and fragmented, which is unaffordable for people with rural *Hukou* (Blumenthal & Hsiao, 2005). Even for those birth cohorts born before emergence of *Hukou system*, their life trajectories are significantly shaped by this residential system in adulthood and late life. Being rural residents is a cumulative disadvantage over the life course, for they have higher risk in school dropout, lower chances to seek for higher education in early life. During the adulthood, they work on farm or nonfarm jobs for low payment. The rural-urban income gap have widened after economic reforms since 1980s. Given the urban-rural segregation and gender roles in social institutions, the last two hypothesis

examines whether the association between childhood disadvantage and health trajectories vary across residential areas and gender.

Hypothesis 3. Patterns of associations between childhood disadvantage and age trajectories of frailty are different between rural and urban areas.

Hypothesis 4. Patterns of associations between childhood disadvantage and age trajectories of frailty are different between male and female.

DATA AND METHODS

I draw data from the Chinese Longitudinal Healthy Longevity Survey (CLHLS) from 2002 to 2011. The CLHLS is a nationally representative survey that selects half of total counties and cities in 22 out of 31 provinces in China, which has covered 1.1 billion people and about 85 percent of total population (http://centerforaging.duke.edu/chinese-longitudinal-healthy-longevity-survey). The goal of this survey is to determine how social, behavioral, biological and environmental factors affect health and longevity in Chinese society. Baseline survey is in 1998, and respondents are followed up every two or three years. Nowadays, CLHLS has collected six waves: 1998, 2000, 2002, 2005, 2008 and 2011. In each wave, deceased or lost to follow-up people are replaced with new participants. In first two waves (1998 and 2000), CLHLS cohort is 80 years old and above. Since 2002, younger birth cohorts aged 65 – 79 have been included, and birth cohorts aged 45 – 64 have been included since 2008. In order to obtain a large age range and repeated measures of health in the longitudinal survey, this study chooses wave 2002, 2005, 2008 and 2011. 16,064 Chinese elderly aged 65 years and older participated into this survey in 2002. I exclude people aged less than 65 years old or above 105 years old (n=260) and drop individuals with missing value in frailty index (n=242), childhood disadvantage, demographic characteristics and health behaviors (n=1020). Finally, we have 14,617 respondents in analytic sample.

Frailty is defined based on the counts of deficits such as symptoms, signs, diseases, disabilities or other abnormalities of health conditions. Frailty Index (FI) is calculated by counting the number of deficits divided by the total number of potential deficits evaluated (Rockwood et al 2010). The range of FI is from 0 to 1. For instance, if a respondent reports 6 out of total 41 items of deficits listed on questionnaire, FI for this person equals to 6/41 = 0.146.

Higher value of FI represents poor health conditions. FI in this paper includes instrumental activities of daily living (IADLs), activities of daily living (ADLs), functional limitations, cognitive impairments (measured by the Mini Mental State Examination, MMSE), auditory/visual ability, heart rhythm, numbers of times suffering from serious illness in the past two years, and interviewer reported health (Appendix Table A1).

Cohort variable is separated into 8 categories with 5 year interval for each group: before 1900, 1900-1904, 1905-1909, 1910-1914, 1915-1919, 1920-1924, 1925-1929 and after 1930. Cohort coded from 1 to 8 is a continuous variable in model estimation. Childhood disadvantage includes four retrospective questions in 2002 survey, (1) No schooling: never went to school (yes=1, no=0), (2) Poor nutrition: arm length in the lowest quartile (yes=1, no=0), (3) Inadequate health care: unable to get access to health care service when being sick in childhood (yes=1, no=0), (4) Rural born: birth in rural areas and (5) Father in low SES: father in manual occupations before sixty years old (yes=1, no=0). Previous studies shows arm length indicates nutrition conditions in early life, which is a reliable measure of nutrition conditions in early life for old people than other physical signs such as height (Huang & Elo, 2009; Jeong et al., 2005). I define arm length in bottom quartile (1= arm length in bottom quartile, 0 = otherwise) as an indicator of poor nutrition in childhood. Inadequate access to health care is measured by asking "Were you able to get access to health care when you were sick in childhood". I code those who answered "Not sick in childhood" or "be able to get access to health care" as 0; those who were unable to get access to health care when sick as 1. Father's occupation in (1) professional and technical personnel, (2) governmental, institutional or managerial personnel is coded as 0; I code (3) staff/service works/industrial workers, (4) personnel in agriculture fishery forestry animal

husbandry, (5) housework, (6) military personnel, (7) unemployed and (8) others before sixty years old as 1, representing father in low SES.

Demographic characteristics include age, gender, marital status, occupation, and residential areas. Age (65-105 years old), gender (female=1, male=0), marital status (devoiced/widowed/never married=1; currently married=0), residential areas (rural=1, urban=0) and occupation (manual=1, otherwise = 0) were collected in 2002. I create dummy variables to adjust for attrition issue: death (died=1, survival=0) and lost to follow up (yes=1, no=0). Health behaviors include: (1) did you smoke in the past (Yes=1, No=0), (2) did you drink alcohol often in the past (Yes=1, No=0), and (3) did you take exercise often in the past (Yes=1, No=0).

Analytic Methods

This paper uses growth curve model (GCM) to examine the association of childhood disadvantage with age trajectories of frailty in late life. Level 1 model (equation 1) estimates frailty index trajectories with age, named age trajectories of frailty, within each person. Level 2 model (equation 2) examines whether variations of childhood disadvantage (CH_{ij}) and cohort ($Cohort_i$) are associated with the changes of intercept (β_{0i}) and slope (β_{1i}) of age trajectories of frailty between persons. Dependent variables in level 2 model are parameters of level 1 model, which calculates to what extent the variance of mean and slope for age trajectories of frailty within each person can be explained by variance of childhood disadvantage and cohort variables. Interaction terms between childhood disadvantage and cohorts ($CH_{ij} \times Cohort_i$) examine whether the association of age trajectories of frailty with childhood disadvantage varies by birth cohorts, as hypothesis 2 states.

In level 1 model, FI_{ti} represents repeated measures of individual i's frailty index at time t. In our analysis, $t = 1, 2, 3, 4$ refer to each time point of survey year 2002, 2005, 2008, and

2011. Age_{ti} is age of the respondent i at time t. For an easier interpretation of intercept (β_{0i}), I use centered age, for it allows us to interpret the intercept (β_{0i}) as mean levels of frailty index when a person is at median age rather than at age of 0. In addition, centered age has two merits in term of robustness of statistical estimates: (1) centered age eliminates confounding effects of age and cohort variables; (2) centered age minimizes the correlation between age and quadratic age and gains a robust estimation (Chen et al., 2010). Age_{ti} is centered by median age of each birth cohort by subtracting a median age - $m(Age)$ within each birth cohort at time t. For instance, there are 894 respondents in birth cohort before 1900 with a median age of 103 years old. For each person in this birth cohort, their centered age equals to their actual age in survey year of 2002 subtracting 103. Therefore, for those older than median age, their centered ages are positive, whereas, for those younger than median age, they centered ages are negative. For other notations in level 1 model, β_{1i} is the linear growth rate of age trajectories of frailty; β_{2i} is the quadratic growth rate of age trajectories of frailty. e_{ti} represents residual of each individual in age trajectories of frailty at time t. In sum, FI_{ti} and $Age_{ti} - m(Age)$ and e_{ti} are time-variant sections in growth curve model.

Level 1 Model

$$FI_{ti} = \beta_{0i} + \beta_{1i}[Age_{ti} - m(Age)] + \beta_{2i}[Age_{ti} - m(Age)]^2 + e_{ti} \qquad (1)$$

Level 2 Model

for the intercept:

$$\beta_{0i} = \gamma_{00} + \sum_{j=1}^{5} \gamma_{01j} CH_{ij} + \gamma_{02} Cohort_i + \sum_{j=1}^{5} \gamma_{03j} CH_{ij} \times Cohort_i + \sum_{j=1}^{5} \gamma_{04j} Z_{ij} + u_{0i} \qquad (2)$$

for the linear growth rate:

$$\beta_{1i} = \gamma_{10} + \sum_{j=1}^{5} \gamma_{11j} CH_{ij} + \gamma_{12} Cohort_i + \sum_{j=1}^{5} \gamma_{13j} CH_{ij} \times Cohort_i + \sum_{j=1}^{5} \gamma_{14j} Z_{ij} + u_{1i} \qquad (3)$$

In level 2 model, γ_{00} is the expected value of frailty at median ages; $\gamma_{01j}$is the coefficient of each domain of childhood disadvantage (j = inadequate access to health care, poor nutrition,

no schooling, rural born and father in low SES) on intercept of age trajectories of frailty across individuals; γ_{02} is the main effects of birth cohort on the mean of frailty; γ_{03j} is the coefficient of interactions between childhood disadvantage and cohorts, indicating how much mean effects of childhood disadvantage on frailty trajectories vary across birth cohorts. γ_{04j} is the coefficient for time invariant covariates Z_{ij} on mean level of frailty trajectories, such as sex, residential areas, marital status, occupation, health behaviors and attrition. For the linear growth rate of age trajectories of frailty, γ_{11j} is coefficient of interaction term between age and childhood disadvantage - $CH_{ij} \times Age_{ti}$ (substitute β_{1i} using equation 3), which indicates whether the effect of childhood disadvantage on frailty vary by age. γ_{12} is coefficient of interaction term between age and cohort - $Cohort_i \times Age_{ti}$ γ_{13j} is coefficient of three-way interaction term between age, cohort and childhood disadvantage - $CH_{ij} \times Cohort_i \times Age_{ti}$, which indicates whether the growth rate of age trajectories of frailty in childhood disadvantage varies across birth cohorts. γ_{14j} is the coefficient of interaction term between age and covariates - $Z_{ij} \times Age_{ti}$.

The CLHLS dataset span 9 years from 2002 to 2011. The number of observations declined from 14,617 in 2002 to 2,347 in 2011 due to the increasing numbers of the deceased and nonresponses (see Figure S1). The length of observation time for each respondent is unbalanced across four waves. For instance, about 35 percent of respondents from 2002 survey deceased and about 13 percent from 2002 survey were lost to follow up in 2005 survey, suggesting about 48 percent of total respondents have only 1 time point observed value that contributes to the changes of age trajectories of frailty over the 9 year observation period. Unbalanced observation period across persons may be associated with the respondents' health conditions, such as it is highly possible that those with higher frailty index are more likely to decease and lost to follow up than others with lower frailty index. In other words, the missing

cases due to the deceased and nonresponses are not randomly distributed, which may result into biased estimates if ignoring attrition issue in datasets. In our analysis, unbalanced data raise two questions: (1) whether growth curve model (GCM) can obtained unbiased estimates from incomplete or unbalanced number of repeated measures in longitudinal data and (2) if assuming repeated measures are not missing at random, what strategies can diminish influence of nonrandom missing due to attrition on coefficient estimates effectively. For question (1), the advantage of GCM allows for incomplete and unbalanced data, in other words, the number of FI_{ti} can vary over subjects. GCM can obtain consistent and asymptotically normal resulting estimates without estimating the missing values, particularly in large sample and under normality assumptions of fixed covariates, such as $CH_{ij}, Cohort_i$ and Z_{ij} in our analysis (Raudenbush & Bryk, 2002; Vonesh & Carter, 1987, 1992). For question (2) on nonrandom missing issue, we create a dummy variable for attrition in level 2 modeling to distinguish subjects deceased and nonresponse from survival ones. We account for attrition as a dummy variable in second level modeling, assuming health conditions of the deceased and nonrespondents are different from other survival individuals, which is a simple but effective way to control attrition issue in longitudinal data (Chen et al., 2010).

RESULTS

Table 1 describes characteristics of whole sample and stratified subsamples by residential areas and gender. Average frailty index of total sample is 0.16 (s.d.= 0.15). Urban elderly have higher frailty than rural elderly; female have higher frailty than male. Average frailty level declines in recent survey years, partially because of early birth cohorts with high frailty index deceased in the observational time period from 2002 to 2011. Average age of whole sample is around 86 (s.d.= 11.37) years old. About 6 percent respondents were born before 1900, about 58 percent were born between 1900 and 1919; and 37 percent were born after 1920. About 30 percent of female were born before 1904, while about 12 percent of male were born before 1904. About half of respondents didn't get adequate access to health care in childhood. The percentage of inadequate access to health care is 14 percent higher for people in rural areas than those in urban areas; is 3 (=50-47) percent higher for female than male. About 27 percent people had poor nutrition during childhood. Living in rural areas is more likely to have poor nutrition in childhood than living in urban areas; female have poor nutrition than male. Education in childhood follows the same pattern of difference between residential areas and gender. About 81 percent of female had never went to school, whereas male had 34 percent. People living in rural areas has 15 percent higher in no schooling than those living in urban areas. 71 percent of urban elderly were born in rural areas, where 96 percent of rural elderly were rural born. There are about 96 percent of respondents whose father worked in manual occupations before sixty years old.

Table 1. Sample Descriptions CLHLS 2002 - 2011 (N= 14,617)

	Whole Sample (*N* = 14,617)		Rural (*n* = 7,857)		Urban (*n*= 6,760)		Female (*n*= 8,239)		Male (*n*= 6.378)	
Variable	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Frailty Index										
Frailty index (2002 - 2011)	.16	.15	.15	.14	.17	.15	.18	.15	.12	.13
Frailty index 2002	.14	.15	.13	.15	.14	.15	.16	.16	.11	.13
Frailty index 2005	.14	.15	.13	.14	.15	.16	.16	.16	.11	.14
Frailty index 2008	.12	.14	.10	.13	.13	.15	.13	.15	.10	.13
Frailty index 2011	.13	.14	.12	.13	.14	.15	.14	.14	.11	.14
Age	85.84	11.37	85.88	11.55	85.81	11.17	87.59	11.66	83.59	10.57
Birth Cohort										
Before 1900 (cohort 1)	.06	-	.06	-	.06	-	.09	-	.03	-
1900 - 1904 (cohort 2)	.16	-	.16	-	.15	-	.20	-	.09	-
1905 - 1909 (cohort 3)	.13	-	.13	-	.12	-	.13	-	.13	-
1910 - 1914 (cohort 4)	.14	-	.14	-	.14	-	.13	-	.15	-
1915 - 1919 (cohort 5)	.15	-	.13	-	.17	-	.13	-	.17	-
1920 - 1924 (cohort 6)	.11	-	.11	-	.11	-	.10	-	.13	-
1925 - 1929 (cohort 7)	.10	-	.11	-	.10	-	.09	-	.12	-
After 1930 (cohort 8)	.16	-	.16	-	.15	-	.14	-	.18	-
Childhood Disadvantage										
Inadequate access to health care	.49	.50	.55	.50	.41	.49	.50	.50	.47	.50
Poor nutrition	.27	.45	.29	.45	.25	.44	.38	.49	.14	.34
No schooling	.61	.49	.68	.47	.53	.50	.81	.39	.34	.47
Rural born	.84	.37	.96	.20	.71	.46	.84	.36	.84	.37
Father in low SES	.96	.20	.98	.15	.94	.24	.96	.19	.95	.21
Covariates										
Female	.56	.50	.57	.50	.56	.50	-	-	-	-
Rural (urban=0)	.54	.50	-	-	-	-	.54	.50	.53	.50
Non-married (Married=0)	.68	.47	.69	.46	.67	.47	.82	.39	.50	.50
Low occupation	.91	.28	.97	.18	.85	.36	.97	.18	.84	.37
Attrition										
Died	.59	.49	.64	.48	.53	.50	.60	.49	.57	.50
Nonresponse	.25	.43	.18	.38	.34	.47	.25	.43	.25	.44
Health behaviors										
Smoking (never = 0)	.34	.48	.34	.47	.35	.48	.15	.35	.60	.49
Alcohol (never = 0)	.32	.47	.32	.47	.31	.46	.18	.38	.50	.50
Exercising (never = 0)	.38	.49	.29	.45	.49	.50	.32	.47	.46	.50

Table A2 shows descriptive statistics by each birth cohort. Average frailty level is lower in recent birth cohorts than early birth cohorts. Within each birth cohort (column), frailty index increases from 2002 to 2008. Recent birth cohorts are more likely to report inadequate access to health care in childhood than early birth cohorts. Early birth cohorts were mostly rural born, more vulnerable to poor nutrition, and had no schooling in childhood (see Table A2). As covariates, recent birth cohorts are more likely to smoke, drink alcohol; less likely to take exercise than early birth cohorts.

Childhood Disadvantage Differences within Cohorts

Table 2 shows the growth curve model estimates of age trajectories of frailty. Model 1 to Model 4 partially support hypothesis 1 that three indicators (inadequate health care, poor nutrition, and no schooling) of childhood disadvantage increase the mean level of age trajectories of frailty within birth cohorts, however, being born in rural areas decreases the mean level of age trajectories of frailty within birth cohorts. Model 1 shows that overall mean of age trajectories of frailty is 0.216 ($p<0.001$) with a growth rate of 0.127 ($p<0.001$) and a quadratic growth rate of -0.720 ($p<0.001$), indicating frailty trajectories follow a nonlinear trend over ages (see Figure 1 based on the estimation of Model 7). Indicators of childhood disadvantage are added in Model 2, suggesting that poor nutrition and no schooling are positively associated with age trajectories of frailty, whereas inadequate health care and rural born are negatively associated with frailty trajectories. Model 3, after adjusting covariates, shows the significant effects of poor nutrition and no schooling on frailty still remain, but being rural born and inadequate health care are not significant. The estimates of covariates suggest that elderly living in rural areas tend to have a

Table 2. Growth Curve Model Estimates of Childhood Disadvantage with Age Trajectories of Frailty in China

	Whole Sample (*N*=14,617)						
Fixed Effects	(1)	(2)	(3)	(4)	(5)	(6)	(7)
For Intercept							
Intercept	0.216***	0.183***	0.074***	0.420***	0.457***	0.467***	0.466***
	(0.002)	(0.009)	(0.010)	(0.010)	(0.020)	(0.021)	(0.021)
Childhood disadvantage							
Inadequate access to health care		-0.019***	-0.005	0.009***	0.020**	0.021**	0.020**
		(0.003)	(0.003)	(0.003)	(0.007)	(0.007)	(0.007)
Poor nutrition		0.042***	0.026***	0.016***	0.050***	0.049***	0.049***
		(0.004)	(0.004)	(0.003)	(0.007)	(0.008)	(0.008)
No schooling		0.084***	0.034***	0.009**	0.048***	0.044***	0.043***
		(0.004)	(0.004)	(0.003)	(0.008)	(0.008)	(0.008)
Rural born		-0.013**	-0.004	-0.009*	-0.052***	-0.054***	-0.055***
		(0.005)	(0.005)	(0.004)	(0.010)	(0.011)	(0.011)
Father in low SES		-0.012	-0.008	-0.001	-0.048**	-0.051**	-0.050*
		(0.008)	(0.008)	(0.007)	(0.018)	(0.019)	(0.019)
Cohort				-0.048***	-0.056***	-0.058***	-0.058***
				(0.001)	(0.003)	(0.004)	(0.004)
Cohort × Childhood disadvantage							
Cohort × Inadequate access to health care					-0.002	-0.002	-0.002
					(0.001)	(0.001)	(0.001)
Cohort × Poor nutrition					-0.007***	-0.007***	-0.007***
					(0.001)	(0.002)	(0.002)
Cohort × No schooling					-0.007***	-0.007***	-0.007***
					(0.001)	(0.001)	(0.001)
Cohort × Rural born					0.009***	0.009***	0.009***
					(0.002)	(0.002)	(0.002)
Cohort × Father in low SES					0.009**	0.009**	0.009**
					(0.003)	(0.004)	(0.004)
For Linear Growth Rate							
Intercept	0.127***	0.115***	0.082***	0.104***	0.104***	0.148**	0.242***
	(0.010)	(0.010)	(0.009)	(0.008)	(0.008)	(0.051)	(0.058)
Childhood disadvantage							
Inadequate access to health care						-0.020	-0.007
						(0.020)	(0.021)
Poor nutrition						-0.013	-0.017
						(0.023)	(0.023)
No schooling						0.034	0.017
						(0.021)	(0.022)
Rural born						-0.036	-0.038
						(0.028)	(0.028)
Father in low SES						-0.008	-0.007
						(0.050)	(0.050)
Cohort							-0.016***
							(0.005)

For Quadratic Growth Rate							
Intercept	-0.720***	-0.646***	-0.361***	0.076	0.073	0.073	0.099
	(0.051)	(0.050)	(0.047)	(0.043)	(0.043)	(0.053)	(0.053)
Covariates							
Female			0.027***	0.036***	0.035***	0.036***	0.036***
			(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Rural			-0.030***	-0.022***	-0.022***	-0.022***	-0.022***
			(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Non-married (Married = 0)			0.078***	-0.000	0.001	0.000	0.001
			(0.004)	(0.003)	(0.003)	(0.004)	(0.004)
Low occupations			-0.002	-0.010	-0.007	-0.008	-0.008
			(0.006)	(0.005)	(0.005)	(0.006)	(0.006)
Attrition							
Died			0.133***	0.032***	0.033***	0.035***	0.035***
			(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Nonresponse			0.050***	-0.003	-0.002	-0.001	-0.000
			(0.005)	(0.004)	(0.004)	(0.005)	(0.005)
Smoking			-0.007	0.005	0.004	0.005	0.005
			(0.004)	(0.003)	(0.003)	(0.004)	(0.004)
Alcohol			-0.003	-0.008**	-0.009**	-0.010**	-0.010**
			(0.004)	(0.003)	(0.003)	(0.003)	(0.003)
Exercising			-0.028***	-0.030***	-0.029***	-0.031***	-0.031***
			(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Random Effects - Variance Components							
Level 1: Within person	0.161**	0.161**	0.162**	0.164**	0.164**	0.152**	0.152**
Level 2: In intercept	0.161**	0.154**	0.130**	0.100**	0.100**	0.107**	0.107**
In Growth Rate	0.052*	0.055*	0.071*	0.053*	0.054*	0.507*	0.506*
Goodness of Fit							
BIC	-7874.0	-8560.6	-10728.7	-13866.0	-13849.7	-13118.5	-13113.0

Standard errors in parentheses
*** *p<0.001*, ** *p<0.01*, * *p<0.05*

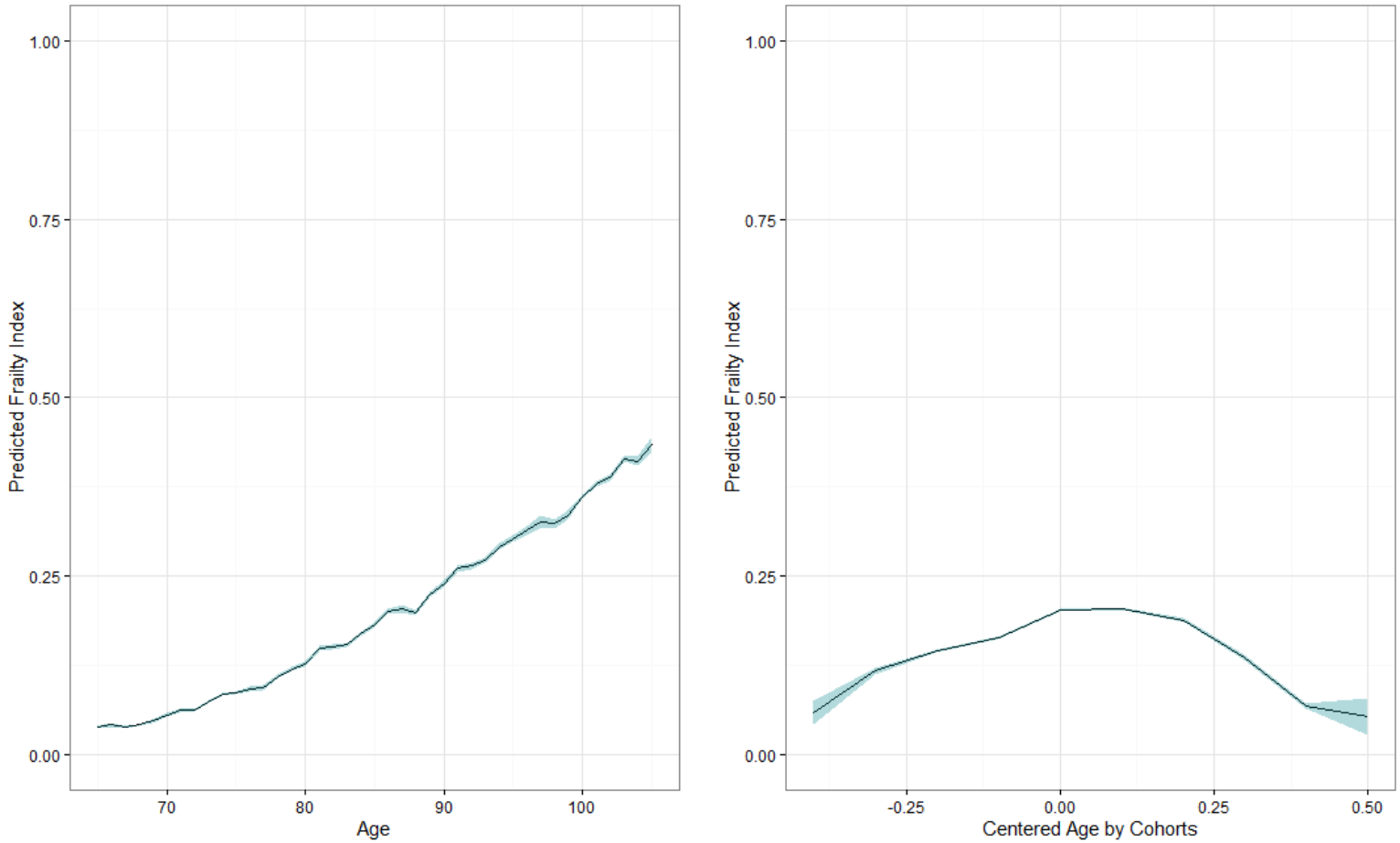


Figure 1. Predicted age trajectories of frailty (N=14,617)

lower frailty than those living in urban areas. Female and unmarried old people are positively associated with greater mean level of frailty trajectories than men and non-married old people. It is noted that died and nonresponse individuals are likely to report higher frailty than survival counterparts.

Model 4 controls for cohorts. The negative coefficient of cohort (-0.048, $p<0.001$) indicates the mean level of frailty trajectories decreases in younger birth cohorts. The estimates of childhood disadvantage in Model 4 suggest that, within each birth cohort, inadequate access to health care, poor nutrition and no schooling are associated with greater mean level of age trajectories of frailty. Being born in rural areas, however, exhibits protective effects against elevated frailty trajectories with age.

Childhood Disadvantage Differences across Cohorts

(Figure 2 is about here)

Model 5 to Model 7 examine hypothesis 2 that whether the gap in the long-term effects of childhood disadvantage on frailty trajectories vary across birth cohorts. The gap of age trajectories of frailty in nutrition and education converge in successive birth cohorts, which is as we expected and supports hypothesis 2. However, the gap of age trajectories of frailty birth place and father's SES diverge in successive cohorts, which does not support hypothesis 2. Model 5 includes interaction terms between cohort and each indicator of childhood disadvantage to examine whether the associations between childhood disadvantage and age trajectories of frailty vary by birth cohorts. Negative coefficients of poor nutrition and no schooling (-0.007, $p<0.001$) indicate the gap of frailty trajectories in these two domains declines in younger birth cohorts, whereas positive coefficients (0.009, $p<0.001$) of rural born and father in low SES indicate the gap of frailty trajectories increases in younger birth cohorts. Figure 2 shows the gaps of predicted

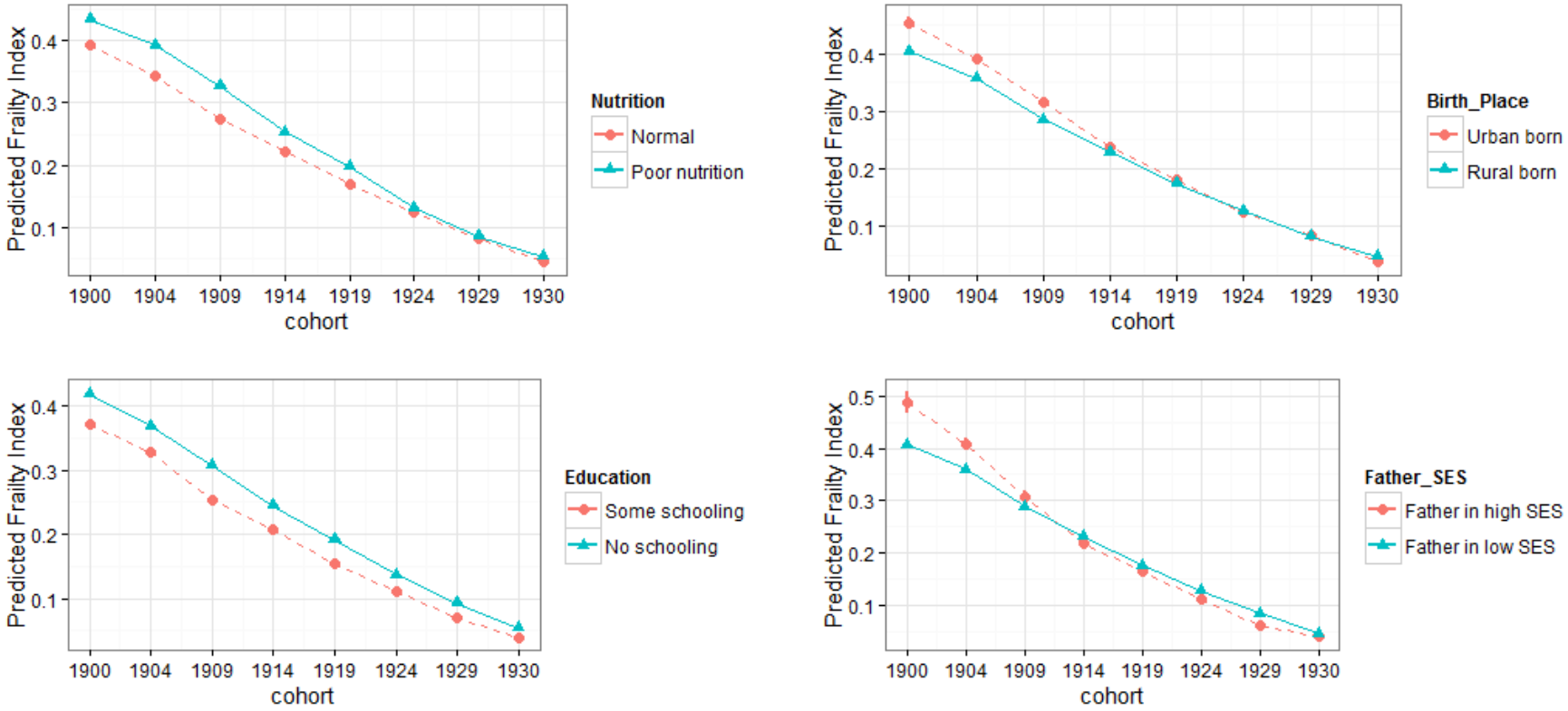


Figure 2. Cohort variations in predicted mean levels of frailty index among Chinese elderly

age trajectories of frailty by each indicator of childhood disadvantage are not uniform across cohorts. Gaps of frailty trajectories in education and nutrition decrease in successive birth cohorts. Gaps of frailty trajectories in birth place and father's SES have a crossover effect over birth cohorts. The predicted frailty index by birth place shows that older people born in urban areas before 1920s have a greater frailty index than those born in rural areas before 1920s in later life, however, those born in urban areas after 1920s have a lower frailty index than those born in rural areas after 1920s. The crossover effects of birth places suggest that urban born origin was disadvantageous for cohorts born before 1920s, however, it was advantageous for cohorts born after 1920s. People having father in high SES have greater frailty for those born before 1910s, whereas they have lower frailty for those born after 1910s. Figure 2 shows that impacts of childhood disadvantage on frailty trajectories differ across birth cohorts and differ among each domain of childhood disadvantage. Model 6 and 7 control for interaction terms of $age \times childhood\ disadvantage$ and $age \times cohort$. The coefficients of interactions terms between childhood and cohorts have little changes after controlling them.

Stratified Sample by Residential Areas and Gender

The estimates of Table 3 support hypothesis 3 and 4 that the association of childhood disadvantage with frailty trajectories vary by residential areas and gender. Results of rural subsample show that inadequate health care is positively associated with higher mean of frailty, whereas there are no significant associations between inadequate health care and frailty in urban subsample. In urban subsample, rural born people are likely to have lower mean level of frailty index than urban born elderly, however, being rural born is not significantly associated with mean level of frailty in rural subsample. In urban areas, differences of mean level of frailty trajectories in rural born and father in low SES diverge in successive birth cohorts, yet these

associations are insignificant in rural areas. The direction of coefficients (γ_{11j}) in interaction age × poor nutrition are positive for rural areas and negative for urban areas, suggesting that the effects of poor nutrition on frailty trajectories increase as ages in rural areas, but decrease as age in urban areas. A three-way interaction term $Age \times Poor\ Nutrition \times Cohort$ is considered in rural and urban subsample analysis. γ_{14j} is the coefficient for this item. $\gamma_{14j} = -0.027$ ($p<0.001$) in rural subsample suggests the rate of poor nutrition gap diverging with ages lessened in successive birth cohorts in rural areas (Figure 4). Figure 4 depicts poor nutrition gap with ages by each birth cohort in rural areas. The poor nutrition gap diverges in early birth cohorts (cohort before 1900, 1900-1904, 1905-1909 and 1910-1904), and converges in later recent birth cohorts (after 1930) in rural areas. Such trend of age trajectories of frailty in rural areas suggests that the effect of poor nutrition weakens in more recent birth cohorts. By contrast, $\gamma_{14j} = 0.047$ ($p<0.001$) in urban subsample suggests the rate of poor nutrition gap converging (-0.036, $p<0.001$) with age strengthened in successive birth cohorts in urban areas (see Figure 5). Figure 5 shows that gaps of age trajectories of frailty in nutrition decrease as ages among early birth cohorts: before 1900, 1900-1904, 1905-1909 and 1910-1914; however, the gaps increase in recent birth cohorts: after 1930 in urban areas. Growth rate of convergence of poor nutrition gap with age increase in successive birth cohorts in urban areas, suggesting the effects of poor nutrition on frailty trajectories strengthen in recent birth cohorts for urban areas.

Table 3 also shows the effects of childhood disadvantage on mean levels of frailty trajectories differ by female and male, which supports hypothesis 4. Inadequate access to health care increases the mean level of frailty for female, however there exists no significant effects on male. No schooling increases the mean level of frailty for male, but not for female. Being born in rural areas increases the mean level of frailty for both female and male. The difference of mean

level of frailty trajectories in birth place diverges in successive birth cohorts for female only and no significant difference exists for male.

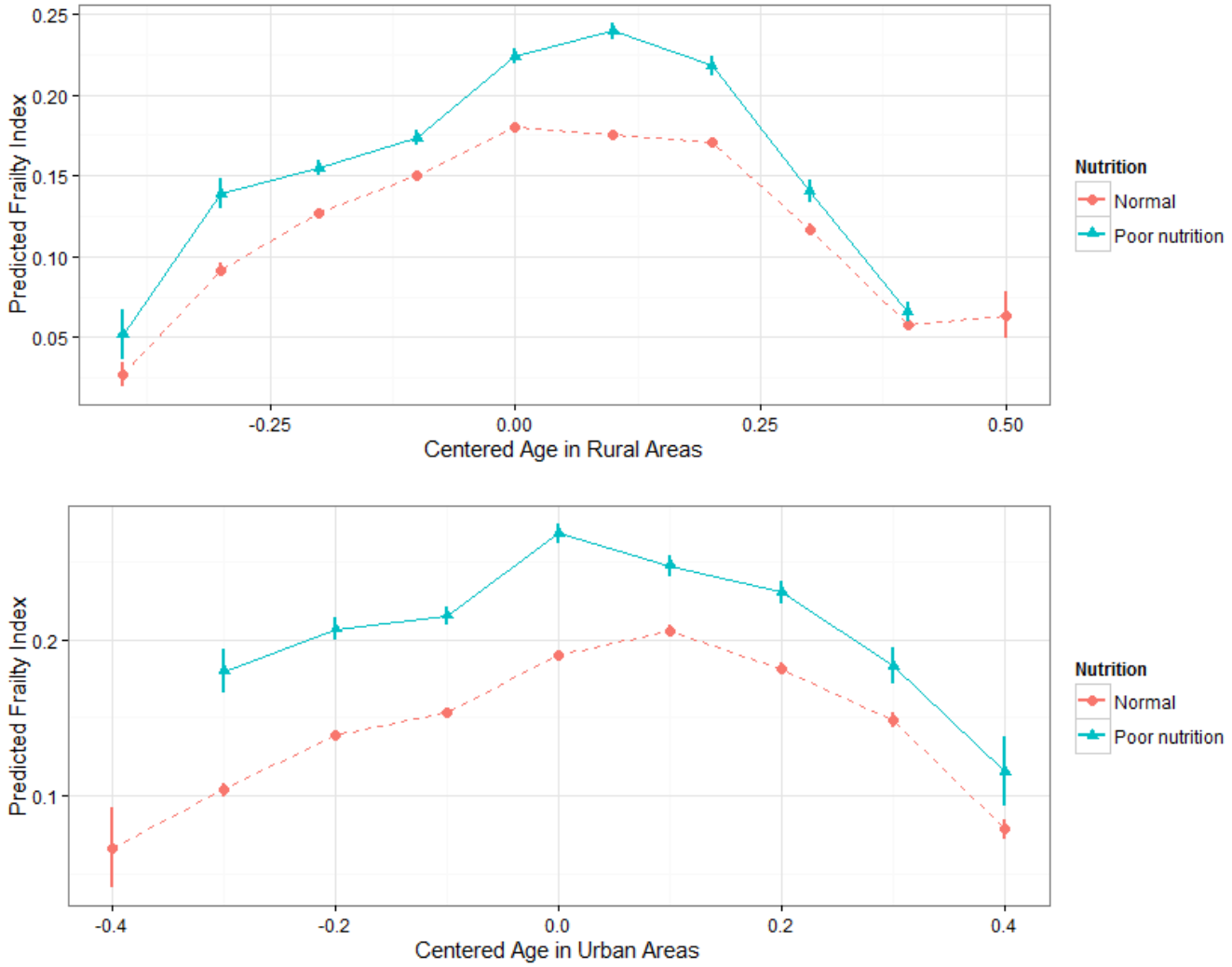


Figure 3. Predicted age trajectories of frailty within cohorts by nutrition in rural and urban subsamples

Table 3. Estimates of Childhood Disadvantage with Age Trajectories of Frailty by Residential Areas and Sex

	Rural	**Urban**	**Female**	**Male**
	n=7,857	*n*=6,760	*n*=8,239	*n*=6,378
Fixed Effects				
For Intercept				
Intercept	0.362***	0.464***	0.532***	0.401***
	(0.039)	(0.025)	(0.028)	(0.029)
Childhood disadvantage				
Inadequate access to health care	0.021*	0.020	0.022*	0.012
	(0.009)	(0.011)	(0.009)	(0.011)
Poor nutrition	0.037***	0.068***	0.037***	0.046**
	(0.009)	(0.012)	(0.009)	(0.015)
No schooling	0.044***	0.056***	0.009	0.027*
	(0.011)	(0.012)	(0.013)	(0.011)
Rural born	0.006	-0.057***	-0.057***	-0.033*
	(0.023)	(0.012)	(0.013)	(0.016)
Father in low SES	-0.049	-0.041	-0.044	-0.025
	(0.031)	(0.023)	(0.025)	(0.027)
Cohort	-0.042***	-0.059***	-0.064***	-0.047***
	(0.007)	(0.004)	(0.005)	(0.005)
Cohort × Childhood disadvantage				
Cohort × Inadequate access to health care	-0.002	-0.002	-0.002	-0.001
	(0.002)	(0.002)	(0.002)	(0.002)
Cohort × Poor nutrition	-0.005**	-0.010***	-0.005**	-0.006*
	(0.002)	(0.002)	(0.002)	(0.003)
Cohort × No schooling	-0.006***	-0.009***	-0.001	-0.002
	(0.002)	(0.002)	(0.002)	(0.002)
Cohort × Rural born	-0.003	0.009***	0.010***	0.004
	(0.004)	(0.002)	(0.003)	(0.003)
Cohort × Father in low SES	0.007	0.009*	0.006	0.007
	(0.005)	(0.004)	(0.005)	(0.005)
For Linear Growth Rate				
Intercept	0.092	0.372***	0.186**	0.285***
	(0.090)	(0.066)	(0.071)	(0.064)
Childhood disadvantage				
Inadequate access to health care	-0.004	-0.003	0.008	-0.025
	(0.021)	(0.026)	(0.024)	(0.023)
Poor nutrition	0.193**	-0.360***	-0.022	-0.013
	(0.063)	(0.078)	(0.024)	(0.033)
No schooling	0.037	0.008	0.034	0.020
	(0.023)	(0.027)	(0.030)	(0.025)
Rural born	-0.044	-0.014	-0.057	0.006
	(0.055)	(0.028)	(0.033)	(0.031)
Father in low SES	0.042	-0.016	0.011	-0.025
	(0.071)	(0.051)	(0.062)	(0.053)
Cohort	-0.005	-0.037***	-0.012*	-0.028***

	(0.006)	(0.007)	(0.005)	(0.006)
Cohort × Poor nutrition	-0.027**	0.047***	-	-
	(0.011)	(0.014)	-	-
For Quadratic Growth Rate				
Intercept	0.021	0.219**	0.173**	0.044
	(0.057)	(0.068)	(0.062)	(0.059)
Covariates				
Female	0.037***	0.032***	-	-
	(0.005)	(0.006)	-	-
Rural	-	-	-0.023***	-0.020***
	-	-	(0.004)	(0.004)
Non-married (Married = 0)	0.006	-0.005	0.003	0.000
	(0.004)	(0.005)	(0.005)	(0.004)
Low occupations	-0.003	-0.009	-0.001	-0.007
	(0.010)	(0.006)	(0.011)	(0.006)
Attrition				
Died	0.034***	0.036***	0.037***	0.031***
	(0.005)	(0.006)	(0.006)	(0.005)
Nonresponse	0.000	-0.000	0.001	-0.001
	(0.006)	(0.006)	(0.006)	(0.006)
Smoking	0.009*	0.000	0.010	-0.001
	(0.004)	(0.005)	(0.006)	(0.004)
Alcohol	-0.008*	-0.009	-0.013*	-0.007
	(0.004)	(0.005)	(0.005)	(0.004)
Exercising	-0.030***	-0.026***	-0.032***	-0.022***
	(0.004)	(0.004)	(0.004)	(0.004)
Random Effects - Variance Components				
Level 1: Within person	0.157**	0.173**	0.154**	0.172**
Level 2: In intercept	0.098**	0.100**	0.091**	0.104**
In Growth Rate	0.043*	0.075*	0.053*	0.059*
Goodness of Fit				
BIC	-8702.8	-4750.1	-7677.6	-5934.9

Standard errors in parentheses
*** *p<0.001*, ** *p<0.01*, * *p<0.05*

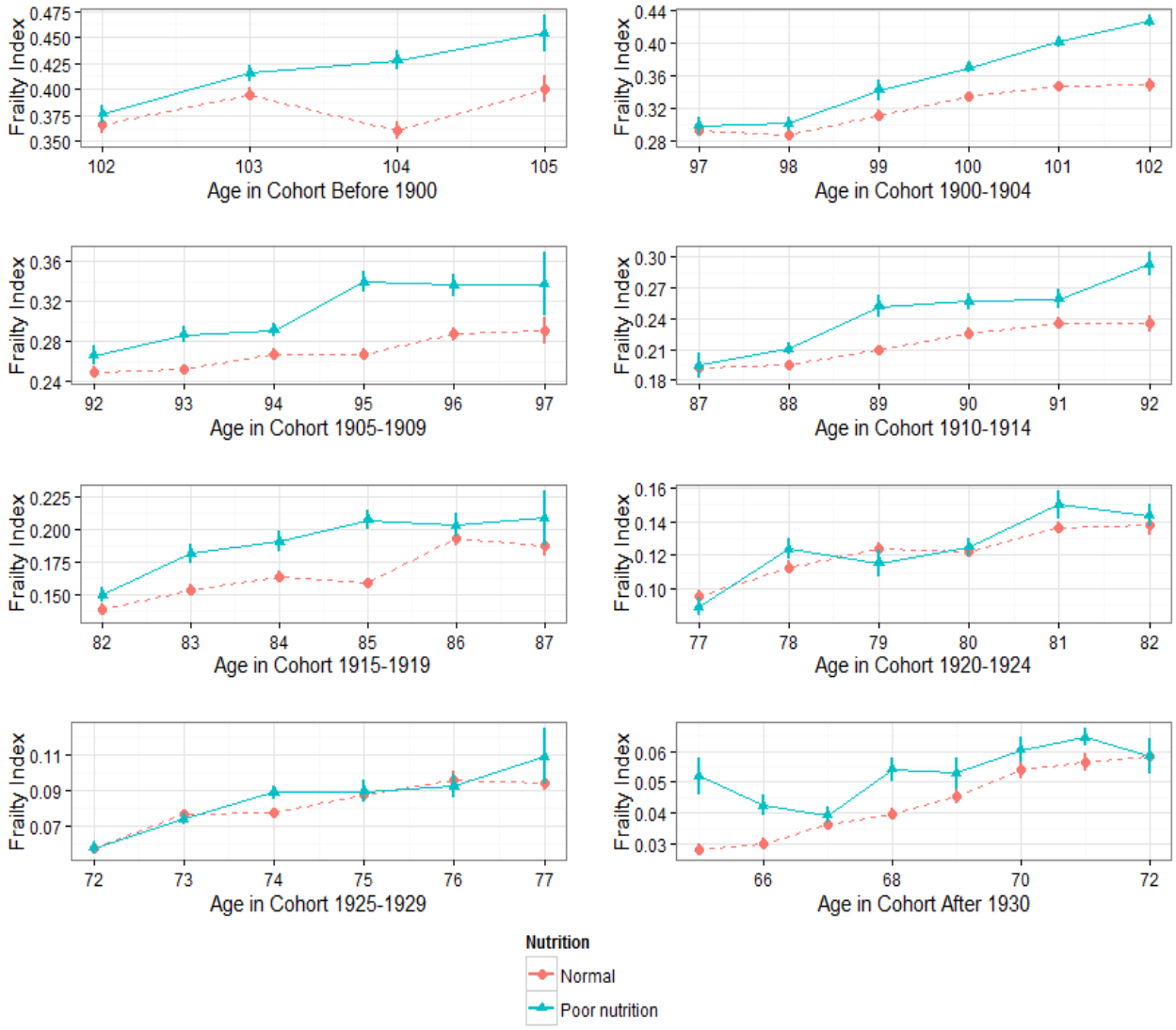


Figure 4. Predicted age trajectories of frailty for each birth cohort by nutrition in rural areas.

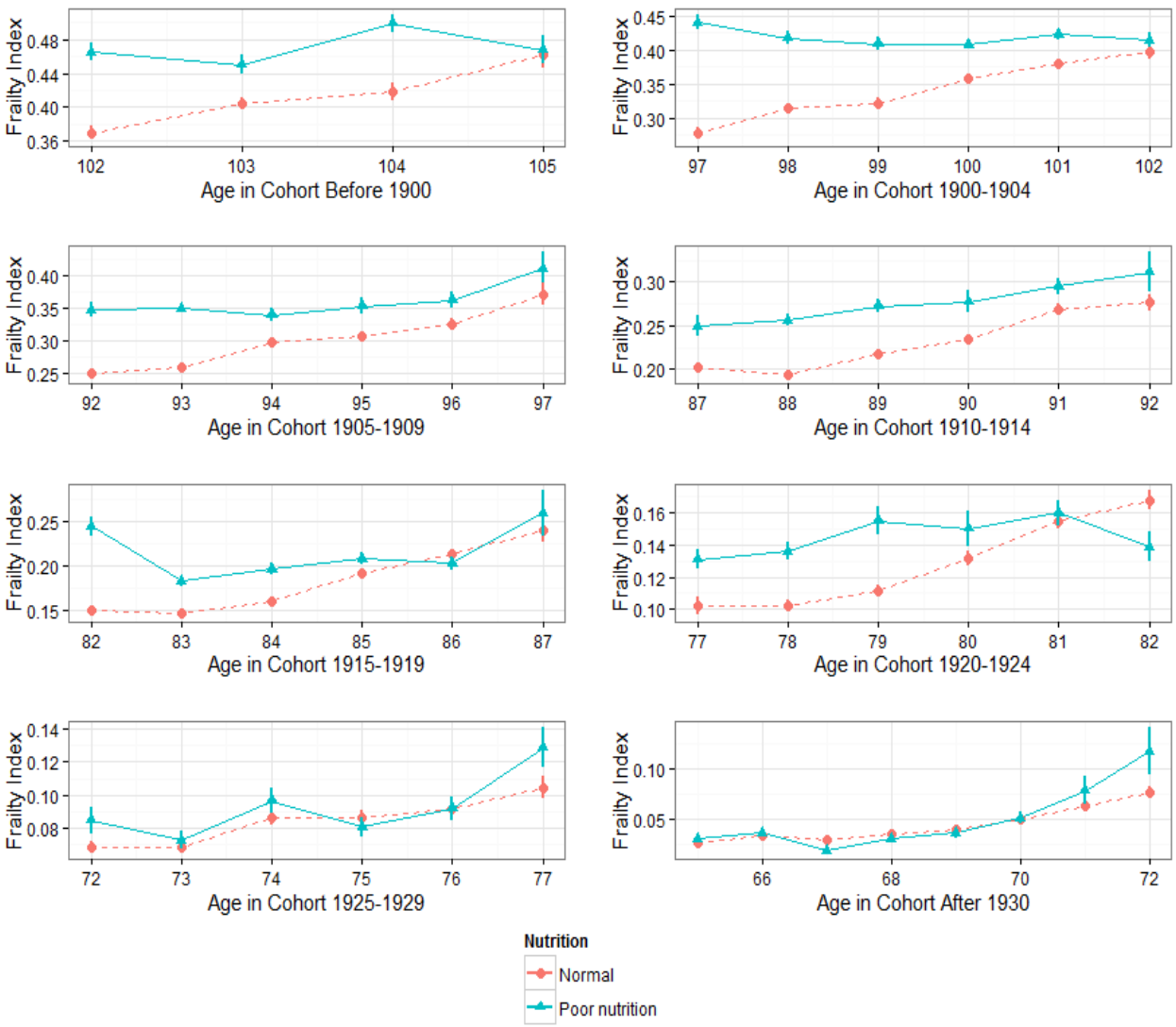


Figure 5. Predicted age trajectories of frailty for each birth cohort by nutrition in urban areas.

CONCLUSION AND DISCUSSION

This study investigates long-term effects of childhood disadvantage on late health trajectories among children who were born in the turn of the twentieth century, and examines whether these associations vary across birth cohorts. Using CLHLS 2002 to 2011, we find childhood disadvantage is associated with elevated mean levels of frailty trajectories in late life. Inadequate access to health care, no schooling and poor nutrition during childhood increase mean levels of frailty trajectories, whereas being rural born and having father in low SES decrease mean levels of frailty trajectories in late life. Frailty differentials in education and nutrition converge in successive birth cohorts, but frailty differentials in birth place and father's SES reverse in successive birth cohorts. A crossover effect of birth place on frailty trajectories occurs near 1920s. Being born in rural areas before 1920s is advantageous for late health outcomes, being born in rural areas after 1920s is disadvantageous for later health, however. Similarly, a cross-over trend also exists in the association between father's SES and frailty trajectories across birth cohorts. Having father in low SES is with lower mean levels of frailty trajectories for those born before 1910s, whereas it increases mean levels of frailty trajectories for those born after 1910s. Rural subsample suggests that for early birth cohorts before 1920s, gap of age trajectories of frailty in poor nutrition widens as ages, but the gap diminishes as ages for birth cohorts after 1930s. In contrast, urban subsample tells a different story that for early birth cohorts before 1925, the gap of age trajectories of frailty diminished with ages, however, slightly widens as people age among birth cohorts after 1925. The long-term effect of childhood poor nutrition on age

trajectories of frailty supports both cumulative (dis)advantage process and age-as-leveler hypothesis in a cohort and institutional-dependent way for Chinese older population.

People of poor nutrition, inadequate health care and illiterate in childhood experience increased levels of frailty index in late life, which is consistent with previous findings (Gu et al., 2009; Huang & Elo, 2009). Being born in rural areas and having father in low SES decrease the meaning levels of frailty in later life, which is inconsistent with most studies on birth cohorts. Evidence from U.S. population, however, to some extent, supports the negative associations among birth cohorts in the early twentieth century. Data from U.S. Census of 1900 and 1910 show that living on farm and having father working on farm had a greater postchildhood survival chances (Preston et al., 1998). One may argue that farmers in U.S. was different from those in China in early twentieth century considering their distinctive social status in different societies. For instance, Chinese peasants were on the bottom of social ladders and hired as labors works for landlord. Though our sample don't allow us to distinguish peasants from landlord based on the self-reported occupational status in analysis, it is noted that rural born children were less likely to expose to polluted water, infectious diseases, shorter life expectancy than urban born counterparts in early twentieth century in China (Campbell, 1997). This may explain that rural born and having father in low SES tend to have healthier later life than others, particular for earlier birth cohorts before 1920s. Results from urban subsample also support that being rural born has a lower mean level of frailty trajectories than those born in urban areas in early twentieth century.

Other studies using the same datasets have similar results and explain the differentials due to selective mortality, arguing that rural born people can survive to old ages partially because they are the "selected" hardest individuals with genetic traits which may enhance their health

outcomes and survival probability over their whole life compared to urban counterparts (Shen & Zeng, 2014). For the mortality selection, I argue the selection issue cannot fully interpret the health disparities in birth place, because both rural and urban born people expose mortality selection over their life. If life expectancy is a reliable indicator that reflects the sanitary conditions in urban and rural areas in early twentieth century, urban born people are more likely to be healthy elites than those rural born in their childhood. Whereas, considering the social segregation occurred in their middle life, rural residence are more likely to be heathy elites than urban residence. Selection trends are time-department across their life course. Without sufficient evidence, we are unable to argue the mortality selection can fully interpret the health inequality in later life.

We also find differentials of frailty trajectories in education and nutrition converge in successive birth cohorts in the twentieth century. CLHLS data show that about 80 percent of Chinese born before 1905 were illiterate. Among recent birth cohorts, percentage of people who had schooling increased from 20 to 50 percent during 1910s to 1930s. The overall educational level of Chinese was very low during that period. Only social elites and higher class people had chances to go to school in early twentieth century of China. Health disparities between schooling and no schooling were high in early birth cohorts, but decreased in successive birth cohorts. Two reasons may contribute to the convergence: the history of public health before 1937 of China and policy implication in 1950s. In the early twentieth century, China didn't accept Western medicine and Western public health concepts until very late around 1910s when the devastating epidemic of pneumonic plague took 60,000 lives in China (Bowers, 1973). During the turn of the twentieth century, prevalence of most infectious diseases and deaths were due to unclean water supplies, unhygienic housing conditions, maltreatment, famine, flood and ignorance and poverty.

To control the increasing prevalence of serious infectious diseases, construction of public health programs was a major interest of the League of Nations since 1910s in China. During that period, peasants, about ninety percentage of total population, cannot read in early twentieth century, it was difficult for public health programs to research major percentage of people during that period (Stampar, 1936). Therefore, education gaps made a great difference in accessing to public health programs before 1937 (Bowers, 1973), which may explain the larger gap in health disparities among early birth cohorts before 1920s. China experienced a phrase of hostilities and civil war from 1937 to 1949. The initial public health program in 1910s was replaced by health programs of Communist party in 1950s. The Chinese Communist Party (CCP)'s health policy "Prevention first; serve workers, farmers, and soldiers; combine traditional and Western medicine; mobilize the masses" effectively increased the life expectancy and improved public health conditions and finally leveled the health disparities in 1950s. These two aspects may interpret the gap of frailty trajectories in education and nutrition converge in successive birth cohorts.

To interpret crossover effects of birth place on differentials in age trajectories of frailty across birth cohorts, disparities in life expectancy and public health development in 1920s of urban and rural China should be considered. In early twentieth century, densely populated cities of China had greater prevalence of infectious diseases than rural areas, resulting into higher mortality risk in cities than the countryside, as the cases of Europe in nineteenth century. Cross-country comparisons show that life chances measured by average life expectancy at birth were about 1.5 times better in countryside than that in larger towns in nineteen century (Woods, 2003). In 1900s, Chinese people born in big cities have shorter life expectancy than those born in the countryside in early twentieth century. Explicit records of life expectancy in the imperial China

were limited. Studies using household registration records of a northern village in China have estimated life expectancy at Age 6-10 was 37.2 for female and 43.8 for male in Daoyi village, rural areas in Liaoning (North China) in late nineteenth century (Lee & Campbell, 2007). Cities of high population density, such as Beijing, did not appear to be healthier places to live than the countryside until improved water infrastructure, public health programs, and sanitation system emerged in urban areas around 1920s (Campbell, 1997). Urban health care facilities and medical training institutions, such as missionary hospitals, care agencies, Peking Union Medical College (PUMC), developed dramatically through central, provincial and municipal authorities during the decade from 1928 to 1937. However, rural areas received little attention in establishing modern medicine and public health (Chen, 1989). During 1920s, the public health improvements in urban areas of China increased the life expectancy, provided better living and housing conditions, and decreased the risks of infectious diseases that benefited health conditions of urban born population over the life course.

China's rapid economic growth has increased total resource of a society and overall Chinese people has gained better nutrition and health conditions than decades ago, however, health disparities increase between rural and urban areas, which is partly due to resources that determine health trajectories are not evenly distributed across social class, regions and birth cohorts. Stratified sample by residential areas shows differentials in growth rate for age trajectories of frailty in nutrition vary by birth cohorts for rural and urban areas. In rural areas, gaps of age trajectories of frailty in nutrition diverge as ages among early birth cohorts (born before 1914), whereas gaps of age trajectories of frailty in nutrition converge as age among recent birth cohorts (born after 1930). However, in urban areas, these trends are opposite. We interpret differential effects of poor nutrition on age trajectories of frailty in rural and urban areas

in light of China's historical and institutional contexts such as household registration (*Hukou*) system, considerably distinct institutional arrangements and distributional channels in rural and urban areas, increased socioeconomic inequality and changing social stratification during the Chinese reform era. Post Mao's reform started in rural areas in 1978 provided peasant household opportunities to free from their land and increase household income from nonagricultural business activities (Bian, 2002; Nee, 1989). Recent birth cohorts (born after 1930) benefit more in social and medical source from market reform of 1970s than early birth cohorts (born before 1914), because the timing of economic reform occurred at 40 to 50 years old of recent birth cohorts, which allow them more involved and benefit from this economic transitions around 1980s. In addition, the universal health care system in late 1970s may level the health disparities related to early childhood conditions. Our findings, converging effects of childhood poor nutrition on late health trajectories, can support that, during late 1970s and early 1980s, the growth household income and universal health care system buffered the long-term adverse effects of poor nutrition on later health outcomes among younger birth cohorts within rural areas (Nee, 1989), whereas the childhood nutrition attainments still matters the late health trajectories for early birth cohorts and the effect cumulate as ages in rural China. The findings from early birth cohorts in rural areas support the cumulative disadvantage hypothesis that the effects of childhood disadvantage on health increase with age. For urban areas, however, differentials of health trajectories in nutrition tell a different story.

Studies show that income inequality declines slightly at the first decade of economic reform era but dramatically increases in subsequent years in major cities of China (Bian & Logan, 1996). Political control in term of Communist party and work units have significant impacts on urban stratification systems in reform era. Early birth cohorts benefit more in public

medical insurance during early stage of economic transition, which may contribute to converging health disparity in late life. As market oriented reform became dominant in late 1990s, the significance of work units and the rank of state ownership declined (Bian & Logan, 1996). Previous public medical service paid by work units and state ownership gradually became difficult to maintain during 1990s. Younger birth cohorts (born after 1930s) are more vulnerable to "out of pocket" medical care than early birth cohorts, which leads to increasing health disparities in later life for urban elderly (Liu, Hsiao, & Eggleston, 1999). The diverging gap of age trajectories of frailty in nutrition for younger birth cohorts reflects the impact of social changes in late 1990s on the health outcomes of urban elderly. Overall, in urban areas, converging health gaps among early birth cohorts support "age-as-leveler" hypothesis that long term effects of childhood disadvantage (poor nutrition) on health disparities decline as ages in late life. For younger birth cohorts, the impacts of economic transitions exert addictive gains or losses on health outcomes for urban elderly in their late lives, which supports cumulative (dis)advantage hypothesis.

Our study has limitations. Less than 10 percent of respondents survive throughout the 10 year observation period (see Figure S1). The cumulative impacts of childhood conditions on health are influenced by premature mortality that people in low SES and/or in poor health are more likely to die early than others. The heterogeneity of mortality decreases as ages, for people who survive tend to have higher SES and good health. Potential mortality selection, on the other hand, underestimates effects of childhood disadvantage on late life health trajectories. Second, since adult SES may influence the trajectories of frailty in old age as much or even more than childhood conditions, limited information about adulthood don't allow us to sufficiently examine social mobility and pathway models over the life course, considering the associations between

socioeconomic position and health are reciprocal (Haas, 2008; Haas & Rohlfsen, 2010). Third, four of five measures of childhood disadvantage are retrospective (except poor nutrition measured by bottom quartile of arm length), which may increase measurement errors of childhood economic conditions. Overall, our findings suggest that childhood disadvantage exhibits long term effects on age trajectories of frailty and frailty trajectory differentials vary across birth cohorts and regions among Chinese elderly in the "long" twentieth century. Evidence from China implies the long-term effects of childhood disadvantage on later health inequality support both cumulative (dis)advantage and age-as-leveler hypothesis, suggesting differential social contexts fundamentally shape the long shadow of childhood conditions on later health through a cohort and institutional-dependent process over the life course.

APPENDIX

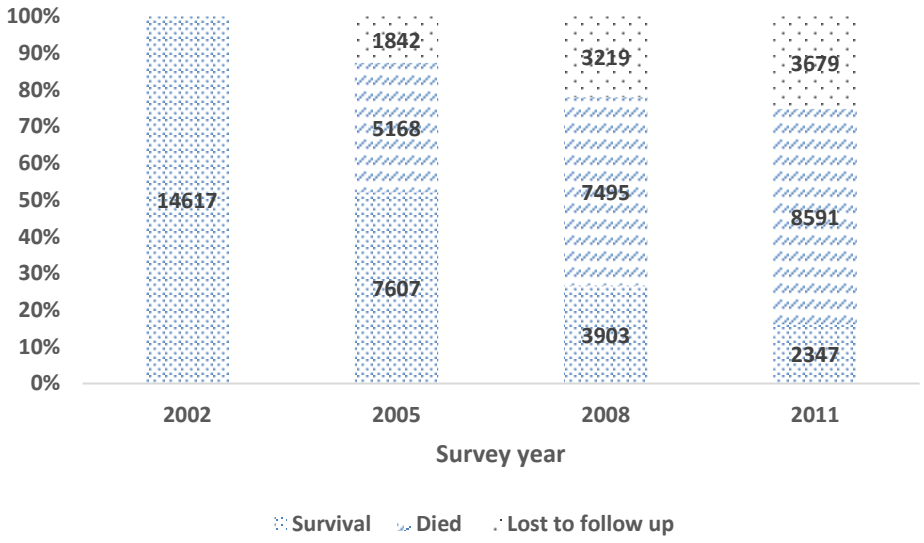
Table A1. Frailty Index Descriptive Statistics in CLHLS 2002 – 2011

Frailty Index Items	Coding	2002	2005	2008	2011
		(*N*=14,617)	(*N*= 7,525)	(*N*= 3,838)	(*N*= 2,149)
		%	%	%	%
IADLs					
Visit neighbors by oneself	0 = Yes; 0 = al little difficult; 1 = not able to do so.	15.76	15.92	12.45	14.33
Shop by oneself if necessary		27.55	24.4	20.21	22.62
Cook meals by oneself if necessary		28.65	26.14	21.57	22.66
Wash clothing by oneself		27.82	25.85	20.74	21.78
Walk continuously for 1 kilometer		31.15	27.65	23.71	25.87
Lift a weight of 5 kg		30.69	27.04	22.92	24.52
Continuously crouch and stand up three times		31.31	28.19	25.19	27.41
Use public transportation		40.39	35.79	31	33.5
Functional Limitations					
Put hand behind neck	0 = right; 0 = left; 0 = both; 1 = neither	5.13	5.57	5.37	4.28
Put hand behind lower back		5.35	6.94	5.89	4.93
Raise arm upright		5.62	5.91	5.63	5.11
Stand up from sitting in a chair	0 = Yes; 1 = No	8.33	8.6	6.93	5.72
Pick up a book from the floor		10.7	11.89	9.3	9.21
ADLs					
Bathing	0 = without assistance; 1 = need assistance	25.71	20.82	13.11	19.59
Dressing		11.66	10.7	7.42	10.28
Toileting		12.51	11.19	7.82	9.44
Transferring indoor		9.89	9.34	6.85	7.96
Feeding		6.53	5.14	3.54	5.72
Incontinence		7.18	6.12	5.05	6.1
Cognitively impaired (MMSE)	1 if MMSE<18; 0 if MMSE > = 18	21.73	21.08	17.4	16.15
Interviewer-rated health	0 = surprisingly healthy; 0 = relatively healthy; 1 = moderately ill; 1=very ill	16.05	17.8	16.46	17.4
Vision	0 = can see and distinguish the break in the circle; 1 = can see but not distinguish the break in the circle; 1= cannot see; 1 = blind	31.7	37.1	32.91	33.13

Hearing	0 = Yes; 1 = Yes, need hearing aid; 1 = Partly, using hearing aid; 1 = No	29.83	26.87	23.79	20.38
Heart rhythm	0 = regular; 1 = irregular	8.64	8.59	-	-
# of times suffering from serious illness in the past two years	0 = no serious illness; 1 = 1 time; 2 = 2 times and more				
	1 time	12.16	15.83	12.69	17.82
	2 times and more	5.7	6.9	5.91	9.73

Note.
1. Frailty Index is calculated by summing up all items and divided total numbers of items in each wave. Frailty Index range [0,1].
2. Frailty Index excludes self-reported health because of more than 10 percent of respondents missing this question in 2002.
3. Frailty Index excluded self-reported chronic diseases, considering potential underestimations of undiagnosed chronic diseases in remote villages or rural areas.

Figure A1. Attrition Descriptions in CLHLS 2002 – 2011



Note. Numbers of survival respondents in 2005 (*n*=7,607), 2008 (*n*=3,903) and 2011(*n*=2,347) are larger than analytical sample (in Table A1) because those with missing values in frailty index are excluded in model estimation.

Table A2 Description of Variables by Birth Cohorts in CLHLS 2002 - 2011

Variables	Before 1900 Cohort 1 (*n*=894) Mean (s.d.)	1900-1904 Cohort 2 (*n*=2,278) Mean (s.d.)	1905-1909 Cohort 3 (*n*=1,855) Mean (s.d.)	1910-1914 Cohort 4 (*n*=2,035) Mean (s.d.)	1915-1919 Cohort 5 (*n*=2,145) Mean (s.d.)	1920-1924 Cohort 6 (*n*=1,606) Mean (s.d.)	1925-1929 Cohort 7 (*n*=1,518) Mean (s.d.)	After 1930 Cohort 8 (*n*=2,286) Mean (s.d.)
Age [median(s.d.)]	103(.99)	100(1.36)	94(1.45)	89(1.53)	84(1.45)	80(1.55)	74(1.49)	68(2.14)
Frailty Index								
Frailty Index 2002	.39(.24)	.36(.24)	.28(.24)	.20(.22)	.13(.18)	.09(.14)	.05(.11)	.03(.08)
Frailty Index 2005	.46(.26)	.40(.26)	.31(.25)	.25(.24)	.18(.22)	.13(.19)	.09(.15)	.05(.11)
Frailty Index 2008	.47(.27)	.43(.27)	.36(.26)	.29(.24)	.22(.23)	.15(.20)	.09(.15)	.06(.13)
Frailty Index 2011	.40(.40)	.44(.27)	.35(.30)	.35(.27)	.30(.26)	.21(.22)	.15(.20)	.08(.14)
Childhood Disadvantage								
Inadequate access to health care	.43(.50)	.55(.50)	.41(.49)	.39(.49)	.35(.48)	.58(.49)	.60(.49)	.57(.49)
Poor nutrition	.39(.49)	.35(.48)	.28(.45)	.26(.44)	.25(.43)	.25(.43)	.23(.42)	.22(.41)
No schooling	.81(.40)	.81(.40)	.66(.47)	.62(.49)	.55(.50)	.56(.50)	.52(.50)	.43(.50)
Born in rural areas	.89(.31)	.88(.33)	.85(.36)	.83(.37)	.81(.39)	.83(.38)	.84(.36)	.83(.38)
Father in low SES	.97(.18)	.97(.17)	.96(.19)	.95(.22)	.95(.23)	.97(.18)	.96(.20)	.96(.20)
Covariates								
Female	.80(.40)	.74(.44)	.56(.50)	.54(.50)	.49(.50)	.49(.50)	.49(.50)	.49(.50)
Rural	.55(.50)	.56(.50)	.56(.50)	.52(.50)	.47(.50)	.54(.50)	.57(.49)	.55(.50)
Nonmarried	.97(.18)	.95(.22)	.87(.34)	.79(.41)	.68(.47)	.56(.50)	.41(.49)	.29(.45)
Low occupations	.97(.18)	.97(.17)	.94(.23)	.92(.27)	.89(.30)	.90(.30)	.88(.33)	.85(.36)
Died	.84(.36)	.81(.39)	.78(.41)	.70(.46)	.63(.48)	.47(.50)	.34(.47)	.22(.41)
Nonresponse	.15(.35)	.18(.38)	.20(.40)	.24(.43)	.25(.43)	.32(.47)	.32(.47)	.34(.47)
Smoked (never = 0)	.20(.40)	.22(.42)	.33(.47)	.35(.48)	.41(.49)	.38(.48)	.41(.49)	.39(.49)
Alcohol (never = 0)	.30(.46)	.27(.44)	.33(.47)	.32(.47)	.37(.48)	.32(.47)	.31(.46)	.31(.46)
Exercised (never = 0)	.32(.47)	.31(.46)	.37(.48)	.45(.50)	.49(.50)	.35(.48)	.34(.47)	.36(.48)

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