

Childhood Disadvantage and Age Trajectories of Frailty in China: A Cohort Analysis

Zhenhua (Jessica) Xu

Department of Sociology

University of North Carolina – Chapel Hill

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Abstract

Childhood disadvantage casts a long shadow on health trajectories over the life course. Few studies, however, have investigated how these associations vary by birth cohorts, especially in developing countries. This paper examines whether childhood disadvantage is associated with age trajectories of health and how cohort effects moderate the associations among Chinese elderly. We draw data from the Chinese Longitudinal Healthy Longevity Survey (CLHLS) and measure health using frailty index. Results from growth curve modeling show that inadequate access to health care, poor nutrition and no schooling during childhood are associated with poor health in late life, while being born in rural areas and having a father with lower occupational status are associated with better health. Educational and nutritional disparities in health trajectories converge in successive birth cohorts, while effects of birth place and father's occupational status on age trajectories of health diverge in successive birth cohorts. Birth cohorts born before 1920s in urban areas have greater frailty than those born in rural areas, whereas cohorts born after 1920s in urban areas have lower frailty than those born in rural areas. These findings suggest that being born in urban areas is disadvantageous for older cohorts, whereas it exerts protective effects among younger cohorts, which is partially due to long term effects of clean water supply and public health around 1920s on later health outcomes in big cities of China. We also find differentials of age trajectories of frailty in nutrition vary by birth cohorts and residential areas, suggesting social changes after 1970s influence health trajectories in late life and these social contexts work distinctively for rural and rural areas.

Key word: childhood disadvantage, frailty trajectories, cohort, China

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Childhood disadvantage may cast a long shadow on late life health (Ferraro et al. 2009, Haas 2008, Herd et al 2007, Smith 2004). Through social, biological and behavioral mechanisms, disadvantageous childhood is associated with high risks of chronic diseases, disability, mental distress, and mortality in later life (Herd et al 2007, Link & Phelan 1995, Pollitt et al 2005, Poulton et al 2002, Pudrovska & Benedicta 2014, Preston et al 1998, Smith et al 2009). How the long-term impacts of disadvantaged childhood on health trajectories vary across birth cohorts in early twentieth century is less discussed, particularly for less developed countries (McEniry 2013). Distinctive causes and consequence of mortality decline in less developed countries, comparing to developed countries, during the twentieth century may lead to cohort specific associations between childhood disadvantage and health trajectories in late life are different for developed western countries and less developed countries during twentieth century. Furthermore, the impact of disadvantageous childhood on health trajectories may vary by gender and residential areas across birth cohorts. Prior works from less developed countries suggest that life expectancy largely improved in at ages under five or above forty in the past century, while female gained larger life expectancy than male and urban areas gained more rapidly than rural areas (Preston 1980). This evidence indicates heterogeneity of life expectancy which highlights the gender and residential differentials in the cohort variations when examining impacts of childhood disadvantage on health trajectories in less developed countries.

This paper uses Chinese Longitudinal Health Longevity Survey (CLHLS) to investigate whether childhood disadvantage is associated with frailty trajectories over the life course, and further examine how the associations vary by birth cohorts in China. Chinese society has changed rapidly in past century, which provides a unique opportunity to examine how childhood conditions in

early 1910s exhibit a long-term influence on health trajectories in later life. Most China-based studies usually treat birth cohorts in early twentieth century as a single birth cohort (Wen and Gu, 2011, Zeng et al 2007), thus cohort variations related to the long term effects of dynamic societal changes during that period are less explored in previous study. For instance, modern medical knowledge spreading and accessible clean water in big cities around 1920 led a critical public health transition in China. Beijing and other big cities in China 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, and reduced 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 advantages with later-life health outcomes. Then I will explain why we need investigate cohort variations in understanding the associations, particularly for China. At last, I explain measurements of health – frailty and discuss the possible advantages using frailty to describe the cohort variations in long shadow of childhood disadvantage on 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 et al. 2010, Willson et al. 2007). Advantaged or disadvantaged early

SES during childhood significantly shapes health trajectories over life course. 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 and 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 and Lee 2009; Elder 1998; Elo and Perstun 1992; Ferraro et al. 2009; Finch and Crimmins 2004; Merton 1968; O’Rand 2006; Smith et al. 2009). Research indicates that “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 and 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 biological markers such as allostatic load, systolic blood pressure and inflammation in late life (Poulton et al., 2002). Childhood residential stability in neighborhood and family is positively associated with good mental health in later life (Bures 2003). Chinese population study using the survey data (CLHLS) find low childhood SES is associated with higher risk of functional limitations, cognitive impairments, poor self-reported health and mortality (Huang and Elo 2010; Shen and Yi 2014; Wen and Gu 2011; Zeng, Gu and Land 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 and 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 models (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 and Barclay 2009; Pudrovska and Anikputa 2014).

Accumulation of risks model emphasizes persistent, additive, overall, and synergistic effects of risks on later health outcomes across multiple life stages (Pudrovska and 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 increase 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 et al. 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

and Zeng 2014, Zeng *et al.* 2007). Mobility model describes dynamics of socioeconomic positions change over life course, changing over life stages, 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. Considering the rapid changing Chinese society and increasing social inequality among urban and rural areas in China over past few decades, we expected the hypothetical models of life course mechanisms may vary by different birth cohorts.

COHORT EFFECTS

From Concepts to Mechanisms

The association has been empirically supported by longitudinal studies across distinctive birth cohorts and geographical areas during past few decades (Hayward & Gorman 2004, Herd 2006). 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 the 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 and Crimmins 2004, Preston and Van de 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. Cohort effects in this article try to integrate the two perspective to understand how early life environments link to late health trajectories over the life course. For instance, water system, public health and medical improvements reducing infections during childhood have lowered the inflammation levels and further diminished morbidity and mortality in later life among contemporary population (Finch and Crimmins 2004, Preston and Van de Walle 1978). Technology advances in human physiology have profoundly increased body size, vital functions of organ system, and longevity for recent birth cohorts (Fogel and Costa 1997).

Cohort Mechanisms in 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 and 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 and Shanahan 2006). Research on cohort mechanisms of health disparities emerge around 1970s, as majority of health measures are mortality during that period. Investigators have discovered a cohort specific nature of mortality trend based on a study of French urban population in nineteenth century, suggesting hygienic improvements increase life expectancy of younger birth cohorts since their initial life stage instead of whole age groups of the population in nineteenth century (Preston and Van de Walle 1978). Existing literature finds that Chinese birth cohorts born during great famine 1959 – 1962 endure worse health outcomes and earn less income than other birth cohorts in later life (Chen and Zhou 2007).

FRAILITY

Frailty is clinically defined as a failure to repair damaged cells that lead to tissue and organ damages (Kirkwood 2005, Rockwood and Mitnitski 2011). Frailty refers to 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 et al. 2004, Kirkwood 2005, Rockwood and Mitnitski 2011). Conventional theoretical concept of frailty as a risk factor of mortality was obtained from many variables using mathematical techniques to investigate pattern of its associations with mortality at the population level. Along this line of research, frailty was defined as fixed at birth and constant throughout the life course (Vaupel et al 1979). Among the early definition of frailty, the variation of frailty as an aging process is unobserved. In most recent decades, however, frailty as a concept has integrated clinical perspectives that have involved systemic measures of symptoms, disability, disease classification, and physiological disorder among community dwelling older people (Rockwood and Mitnitski 2011). Frailty highlights an entity of health reserves by counting for cumulative deficits. Clinical interpretation of frailty about cumulative deficits can be enriched by incorporating the long term effects of childhood conditions on health outcomes over the life course.

Frailty Index

The Frailty Index (FI) is regarded as a valid and generalized composite measure of cumulative health disorders and deficits in biological aging processes by counting total numbers of self-reported symptoms related to chronic diseases, functional limitations, and mental impairments (Searle et al. 2008). 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 and Lee 2010). FI as a reliable 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 represent environment factors that affect 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 2005). FI is widely used to measure frailty cross different populations due to its good validity and 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 frailty (Jones, Song and Rockwood 2004).

Age Trajectories of Frailty Index

Age trajectories describe a temporal pattern of roles, experiences and statuses that vary as one ages. The onset and duration exposing to risk factors jointly shape age trajectories over the life course. In addition, risk accumulations, available resources, social contexts, and human agencies also influence age trajectories (Elder and Shanahan 2006, Hitlin and Elder 2007, Ferraro et al. 2009).

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 which are unique to each birth cohort, this paper gives special attention to integrating cohort variations into the biological aging process under Chinese society.

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 individual aging processes are related to macrostructural circumstances over time and place (Mayer 2004, 2009, Moen 2013). A large proportion of previous studies on the association between childhood disadvantage and health trajectories over the life course mostly draw survey data from developed countries, however, few studies focus on Chinese society. Social institutional foundations between China and western countries are significantly distinct. 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. This leads to 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 et al, 2010). Income gap in self-reported health trajectories diverges across age for birth cohorts in 1903s, 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, along with newly opened treaty

ports around coastal, accelerated urban developments around riverine areas, and renovated public sanitary infrastructures such as water supply, establishments of Health Stations in big cities (Campbell 1997, Fairbank and Goldman 2006). The self-reproductively agricultural economy in villages, however, 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. People in rural *Hukou* have fewer opportunities to receive formal education and get access to health care than those with urban *Hukou*. 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 and Treiman, 2004). In traditional agrarian Chinese society, administration and market in rural areas created hierarchal social structures, including gentries, peasants, inter-middle merchants (Skinner 1964). 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 and Hsiao 2005). Even though birth cohorts in this paper were born before emergence of *Hukou system*, their life trajectories are significantly shaped by this residential system in adulthood and late life. Given the urban-rural divided social institutions, the third hypothesis examines whether the association between childhood adversity and health trajectories vary rural and urban areas.

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 was in 1998, and the respondents were 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 were 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 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 adversity, 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 (Supplement Table S1).

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 can indicate 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 and 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 – 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 modeling to examine long term effects of childhood advantage on age trajectories of frailty in late life. The Level 1 model describes changes in age trajectories of frailty within person. The Level 2 model examines whether these childhood disadvantage indicators can

contribute to the changes of intercept and slope in age trajectories of frailty between persons.

Dependent variables in level 2 are parameters in the level 1 model, indicating heterogeneity of age trajectories of frailty within each person. Gap in age trajectories of frailty within and across birth cohorts are examined by adding interaction term between each domain of childhood advantages and birth cohorts.

FI_{ti} in the level 1 model represents repeated measures of individual i 's frailty index at time t . Age_{ti} is the age of the respondent i at time t . $t = 1, 2, 3, \dots T$; T represents the total number of repeated measures. For instance, when a respondent participates in all waves from 2002 to 2011, T equals 4. Age is centered at the median age of each birth cohort for obtaining an easier interpretation of intercept β_{0i} , where intercept β_{0i} indicates the mean level of frailty trajectories across all birth cohorts at median age. β_{1i} is the linear growth rate of age trajectories of frailty. β_{2i} is the quadratic growth rate of age trajectories of frailty.

Level 1 Model

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

Level 2 Model

for the intercept:

$$\beta_{0i} = \gamma_{00} + \sum_{j=1}^5 \gamma_{01j}CH_{ij} + \gamma_{02}Cohort_i + \sum_{j=1}^5 \gamma_{04j}CH_{ij} \times Cohort_i + \sum_{j=1}^5 \gamma_{05j}Z_{ij} + u_{0i} \quad (2)$$

for the linear growth rate:

$$\beta_{1i} = \gamma_{10} + \sum_{j=1}^5 \gamma_{11j}CH_{ij} + \gamma_{12}Cohort_i + \sum_{j=1}^5 \gamma_{14j}CH_{ij} \times Cohort_i + \sum_{j=1}^5 \gamma_{15j}Z_{ij} + u_{1i} \quad (3)$$

γ_{00} is the expected value of frailty when age equals the median; γ_{01j} indicates the mean of frailty trajectories by each domain of childhood disadvantage (j = inadequate access to health care, poor nutrition, no schooling, rural born and father in low SES); γ_{02} is the main effects of birth cohort on the mean of frailty; γ_{04j} 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_{05j} is the coefficient for 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_{14j} 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 vary across birth cohorts. γ_{15j} is coefficient of interaction term between age and covariates - $Z_{ij} \times Age_{ti}$.

RESULTS

(Table 1 is about here)

Descriptive statistics (see Table 1) shows the average frailty index of total sample is 0.16 (s.d.=0.15). Urban elderly have greater frailty than rural elderly. Female have elevated frailty index than male. Average frailty level decline 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 were not able to get adequate access to health care in childhood. The percentage of inadequate access to health care is 14 percent higher for people living in rural areas than those living 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 S2 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 S3). 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 is about here)

(Figure 1 is about here)

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 increases 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 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 decrease 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 cohort, 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 decline in younger birth cohorts, whereas positive coefficients (0.009, $p < 0.001$) of rural born and father in low SES indicates the gap of frailty trajectories increases in younger birth cohorts. Figure 2 shows the gaps of predicted 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×childhood disadvantage and age×cohort. The coefficients of interaction terms between childhood and cohorts have little changes after controlling them.

Stratified Sample by Residential Areas and Gender

(Table 3 is about here)

(Figure 3 is about here)

(Figure 4 is about here)

(Figure 5 is about here)

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 × Poor Nutrition × 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 suggest that the effect of poor nutrition weaken 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 diverge in successive birth cohorts for female only and no significant difference exists for male.

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 diverge in successive birth cohorts. A crossover effect of birth place on frailty trajectories occurred near 1920s. Being born in rural areas before 1920s is advantageous for late health outcomes, while 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 decreases mean levels of frailty trajectories for those born before 1910s, whereas it increases mean levels of frailty trajectories for those born after 1910s. Results from stratified sample indicate that long-term impacts of childhood disadvantage on frailty trajectories differ in residential areas and gender. Growth rates for the converging differentials in age trajectories of frailty increases in rural areas, whereas it decreases in urban areas. Three-way interaction terms among age, cohort and poor nutrition indicates that the growth rate of converging age trajectories of frailty lessens for recent birth cohorts in rural areas, whereas it strengthens for recent birth cohorts in urban areas.

Individuals with 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 and Elo 2009). Being born in rural areas and having father in low SES decrease the meaning levels of frailty in later life. Even though this association is opposite to most findings based on birth cohorts in late half of twentieth century, there exists some evidence from U.S. population that support this negative association among birth cohorts in the early twentieth century.

Records in 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). As we known, the social contexts of farmers in U.S. was different from China in early twentieth century, since most Chinese people in the countryside were peasants without ownership of land and worked for landlord.

Peasants were on the bottom of social ladders in rural China. It is worthy to be noted that people in countryside were peasants in China's social contexts during that period, though our sample cannot distinguish peasants from land lord based on the self-reported occupational status in our analysis.

Children with rural background are more likely to be peasants or living in countryside in their adulthood and less likely to live in urban areas where were with higher risks of pollution, infectious diseases and shorter life expectancy than rural areas in early twentieth century in China (Campbell 1997). This may explain that rural born and having father in low SES tend to have lower levels of frailty trajectories than their counterparts. Results from urban sample also support that being rural born has a decreased mean level of frailty trajectories than those who were born in urban areas in early twentieth century. Alternative explanation is that people who were born in rural areas or living on country side had harsher childhood. Rural born individuals can live to old ages partially because they are the "selected" hardest individuals with genetic traits which may enhance their health outcomes and survivals over their whole life compared to other counterparts (Preston et al 1998, Shen and Zeng 2014).

We also find differentials of frailty trajectories in education and nutrition converge in successive birth cohorts in the turn of the twentieth century. To interpret this trend, we consider three aspects that can contribute the convergence: (1) distribution of educational levels across cohorts in early twentieth century; (2) the history of public health to 1937 in China and (3) state level policy around 1950s. First, during the turn of the twentieth century, CLHLS data show that about 80 percent of

Chinese born before 1905 were illiterate, about 65 percent of 1910s birth cohorts were illiterate, and nearly 50 percent of people born in 1920s and 1930s were illiterate. The overall educational level of Chinese were very low during that period. Only social elites and higher class people were able to have education in early twentieth century of China. Education reflects social capital which indicates educated people were more likely to cumulate various resources such as social network, financial supports, medical service etc. over life course (Lynch 2003). Health disparities between schooling and no schooling were high in early birth cohorts. Among recent birth cohorts, percentage of people who had schooling increased from 20 in 1900s to 50 percent in 1930s. Effects of literate and illiterate educational levels on health narrowed in recent birth cohorts, and health gaps in higher educational levels may enlarge among them. Second, 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 which 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 had become 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 until 1937 (Bowers 1973). Third, 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 three 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 nineteenth 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 and 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 supply, 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 can benefit 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 cohort (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 households 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 income inequality decline slightly at the first decade of economic reform era but dramatically increase in subsequent years in major cities of China (Bian and 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 and 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 *et al.* 1999). The diverging gap of age trajectories of frailty in nutrition for younger birth cohorts can reflect 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 at aggregate level, 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 additive gains or losses on health outcomes for urban elderly in their late lives.

Our study has limitations. Less than 10 percent of respondents survived throughout the 10 year observation period (see Figure S1). The cumulative impacts of childhood conditions on health are influenced by premature mortality that depicts people in low SES and/or in poor health are more likely to die early than others (Dupre 2007). Cumulative inequality in SES leads to premature mortality as ages due to initial inequality and reproduction of inequality in social system through demographic and developmental processes (Ferraro et al. 2009). 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 childhood disadvantage on late life health trajectories. I argue the long term impacts of childhood disadvantage on age trajectories of frailty may even increase if adjusting mortality selection. 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; Hass and 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.

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

| Variable | Whole Sample (N = 14,617) | | Rural (N = 7,857) | | Urban (N= 6,760) | | Female (N= 8,239) | | Male (N= 6,378) | |
|----------------------------------|------------------------------|-------|----------------------|-------|---------------------|-------|----------------------|-------|--------------------|-------|
| | 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 2 Growth Curve Model Estimates of Childhood Disadvantage with Age Trajectories of Frailty in China

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

For Quadratic Growth Rate

| | | | | | | | |
|---|----------------------|----------------------|----------------------|----------------------|----------------------|----------------------|----------------------|
| Intercept | -0.720*** (0.051) | -0.646*** (0.050) | -0.361*** (0.047) | 0.076 (0.043) | 0.073 (0.043) | 0.073 (0.053) | 0.099 (0.053) |
| Covariates | | | | | | | |
| Female | | | 0.027*** (0.004) | 0.036*** (0.004) | 0.035*** (0.004) | 0.036*** (0.004) | 0.036*** (0.004) |
| Rural | | | -0.030*** (0.003) | -0.022*** (0.003) | -0.022*** (0.003) | -0.022*** (0.003) | -0.022*** (0.003) |
| Non-married (Married = 0) | | | 0.078*** (0.004) | -0.000 (0.003) | 0.001 (0.003) | 0.000 (0.004) | 0.001 (0.004) |
| Low occupations | | | -0.002 (0.006) | -0.010 (0.005) | -0.007 (0.005) | -0.008 (0.006) | -0.008 (0.006) |
| Attrition | | | | | | | |
| Died | | | 0.133*** (0.004) | 0.032*** (0.004) | 0.033*** (0.004) | 0.035*** (0.004) | 0.035*** (0.004) |
| Nonresponse | | | 0.050*** (0.005) | -0.003 (0.004) | -0.002 (0.004) | -0.001 (0.005) | -0.000 (0.005) |
| Smoking | | | -0.007 (0.004) | 0.005 (0.003) | 0.004 (0.003) | 0.005 (0.004) | 0.005 (0.004) |
| Alcohol | | | -0.003 (0.004) | -0.008** (0.003) | -0.009** (0.003) | -0.010** (0.003) | -0.010** (0.003) |
| Exercising | | | -0.028*** (0.003) | -0.030*** (0.003) | -0.029*** (0.003) | -0.031*** (0.003) | -0.031*** (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

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

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

| | | | | |
|---|----------------------|----------------------|----------------------|----------------------|
| Cohort × Poor nutrition | -0.027** (0.011) | 0.047*** (0.014) | - - | - - |
| For Quadratic Growth Rate | | | | |
| Intercept | 0.021 (0.057) | 0.219** (0.068) | 0.173** (0.062) | 0.044 (0.059) |
| Covariates | | | | |
| Female | 0.037*** (0.005) | 0.032*** (0.006) | - - | - - |
| Rural | - - | - - | -0.023*** (0.004) | -0.020*** (0.004) |
| Non-married (Married = 0) | 0.006 (0.004) | -0.005 (0.005) | 0.003 (0.005) | 0.000 (0.004) |
| Low occupations | -0.003 (0.010) | -0.009 (0.006) | -0.001 (0.011) | -0.007 (0.006) |
| Attrition | | | | |
| Died | 0.034*** (0.005) | 0.036*** (0.006) | 0.037*** (0.006) | 0.031*** (0.005) |
| Nonresponse | 0.000 (0.006) | -0.000 (0.006) | 0.001 (0.006) | -0.001 (0.006) |
| Smoking | 0.009* (0.004) | 0.000 (0.005) | 0.010 (0.006) | -0.001 (0.004) |
| Alcohol | -0.008* (0.004) | -0.009 (0.005) | -0.013* (0.005) | -0.007 (0.004) |
| Exercising | -0.030*** (0.004) | -0.026*** (0.004) | -0.032*** (0.004) | -0.022*** (0.004) |
| <hr/> | | | | |
| 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

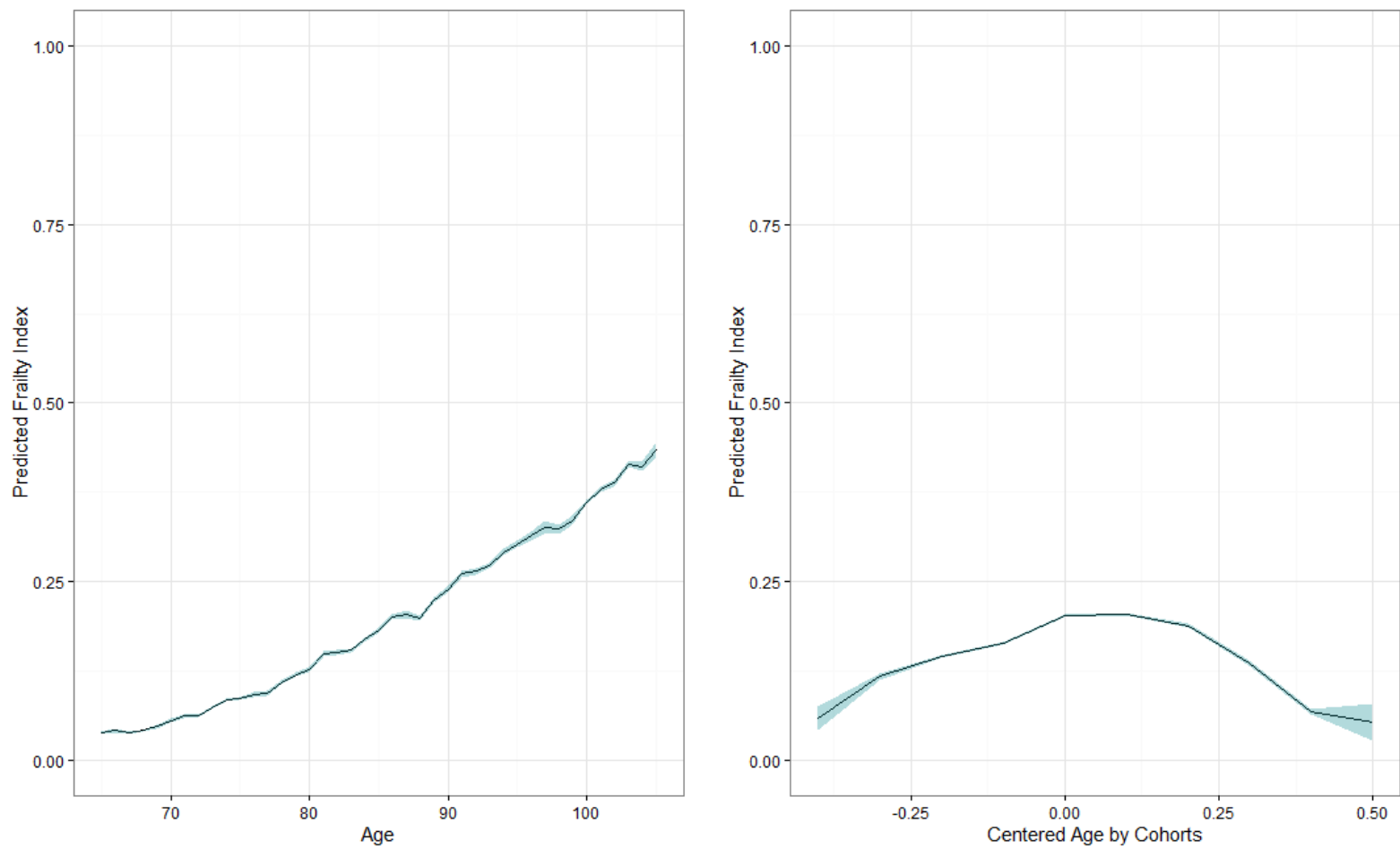


Figure 1. Predicted age trajectories of frailty (N=14,617)

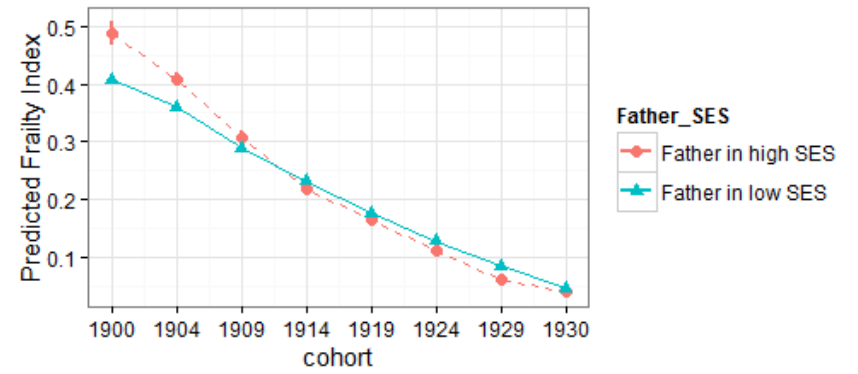
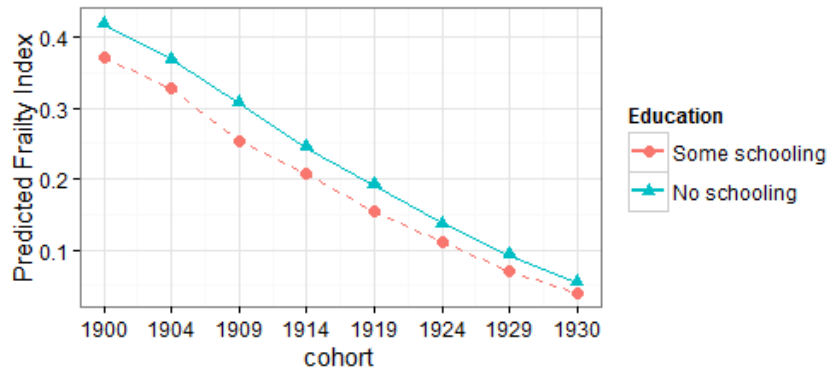
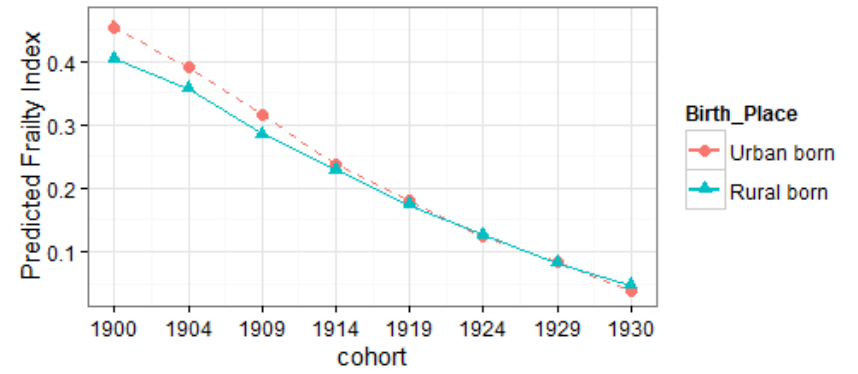
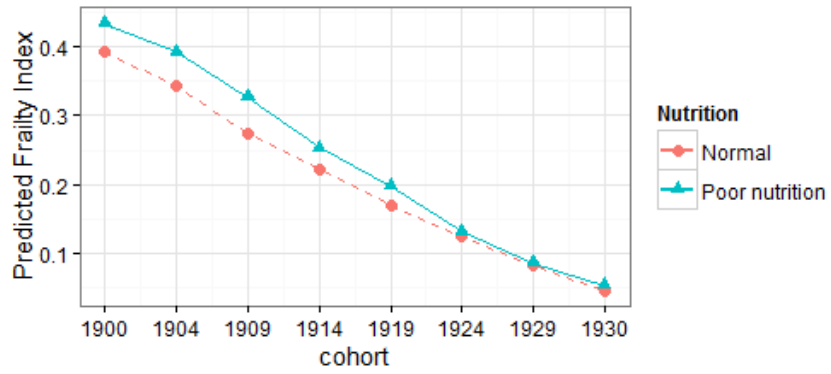


Figure 2. Cohort variations in predicted mean levels of frailty index among Chinese elderly

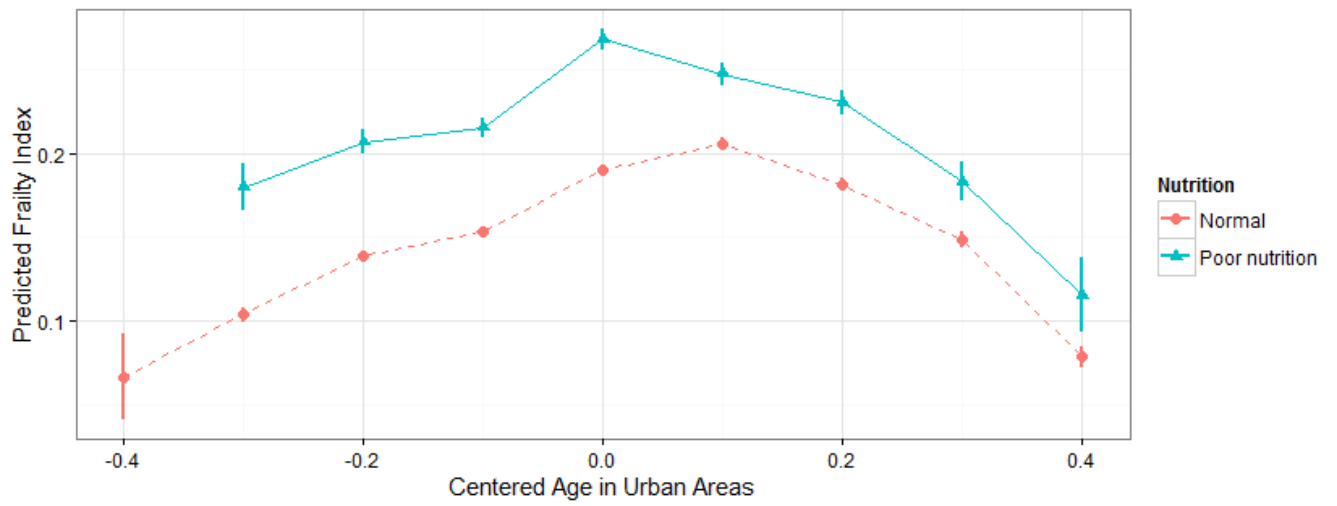
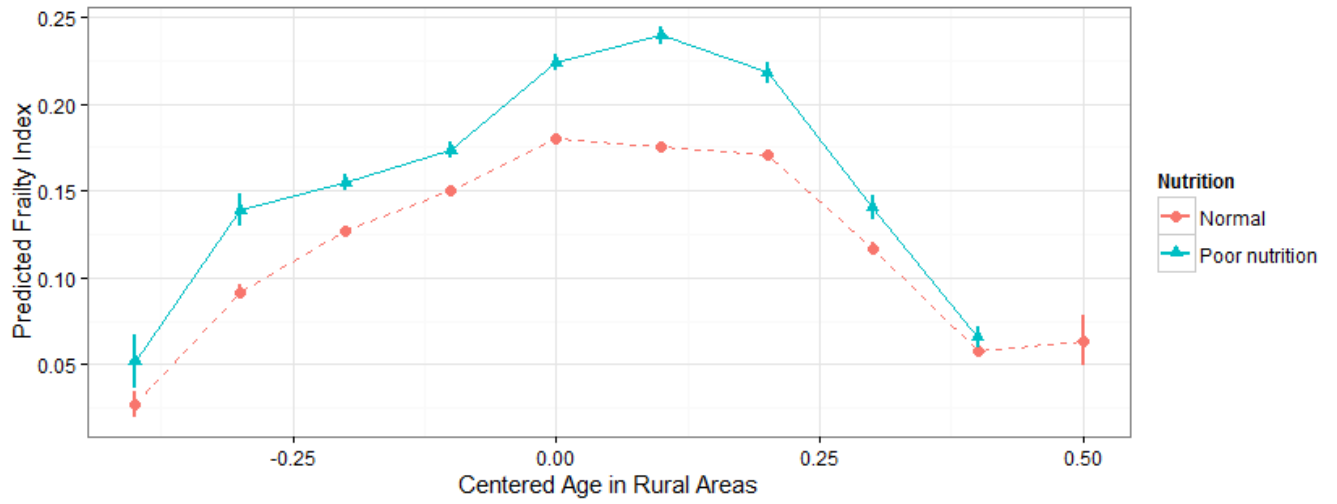


Figure 3. Predicted age trajectories of frailty within cohorts by nutrition in rural and urban subsamples

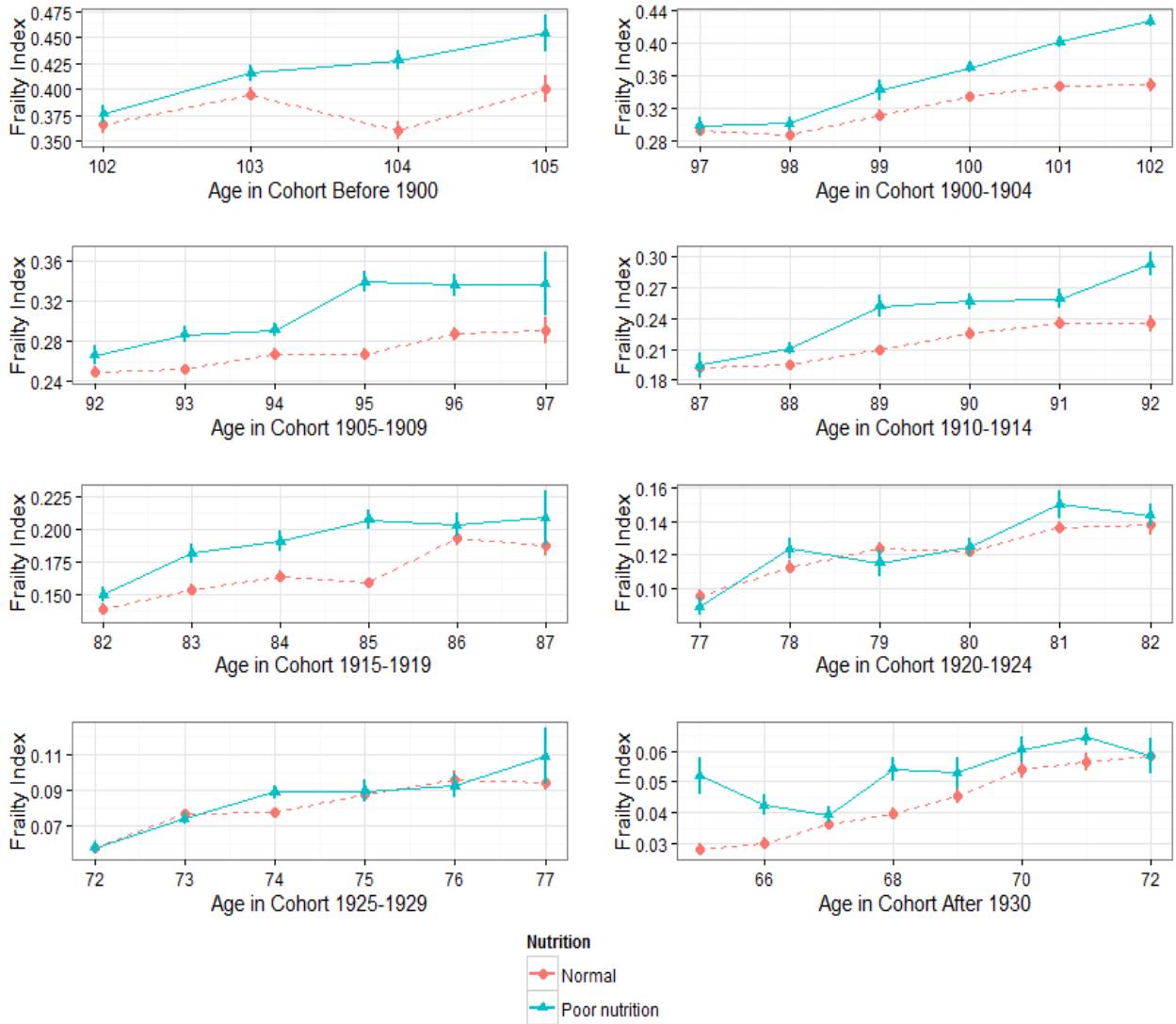


Figure 4. Predicted age trajectories of frailty for each birth cohort by nutrition in rural areas.

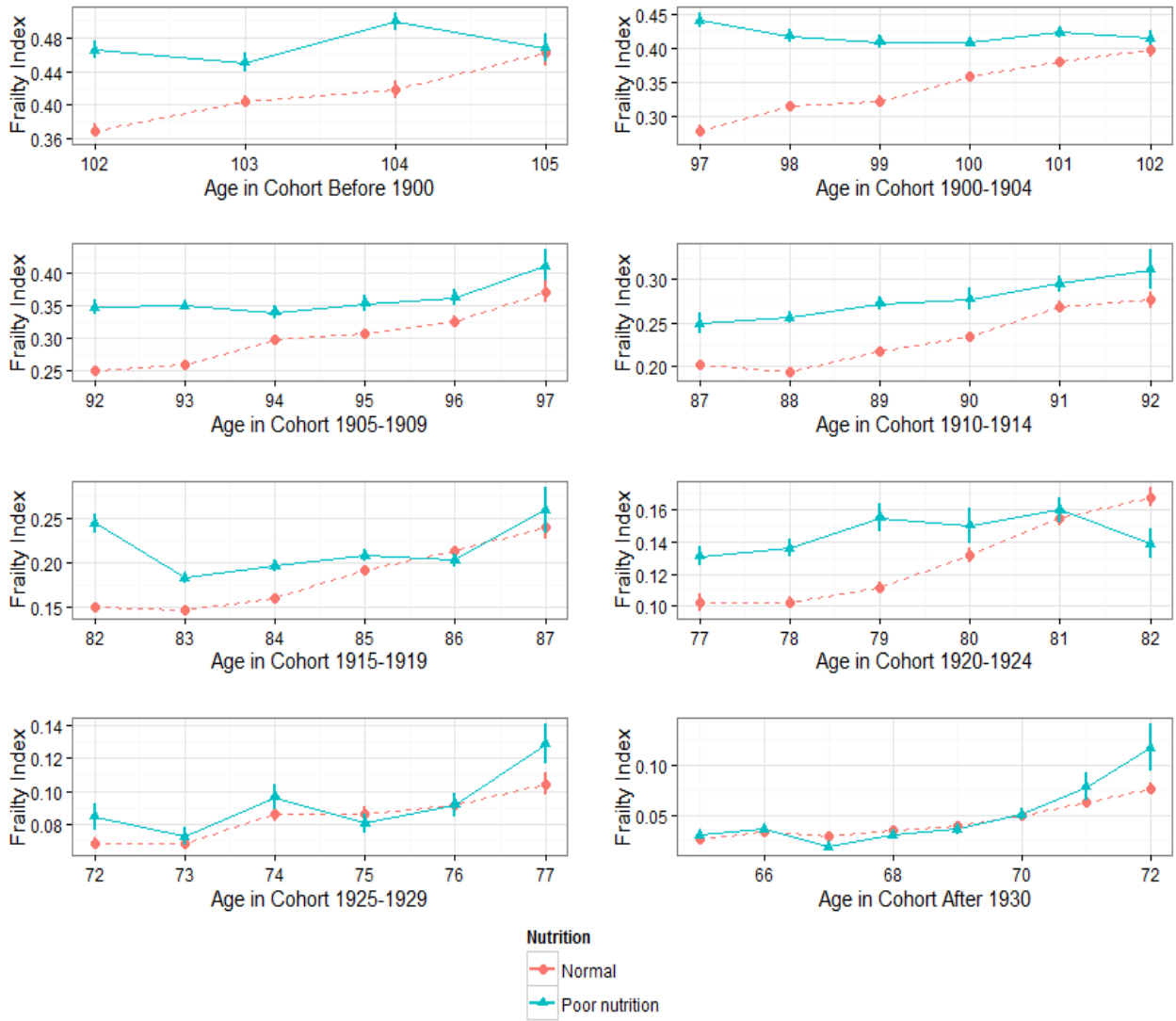


Figure 5. Predicted age trajectories of frailty for each birth cohort by nutrition in urban areas.

Supplement

Table S1. Frailty Index Descriptive Statistics in CLHLS 2002 – 2011

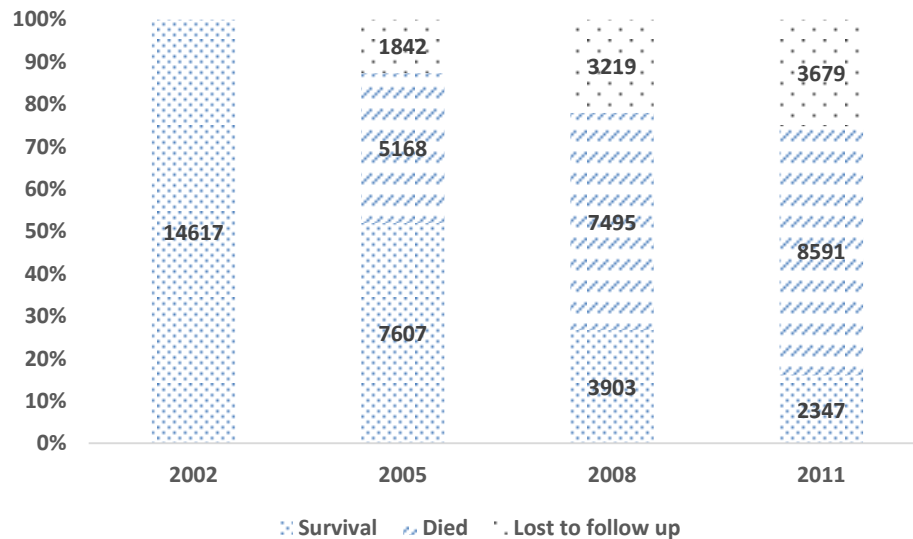
| Frailty Index Items | Coding | 2002 | 2005 | 2008 | 2011 |
|--|--|------------|------------|-------------|-------------|
| | | (N=14,617) | (N= 7,525) | (N= 3,838) | (N= 2,149) |
| | | % | % | % | % |
| <i>IADLs</i> | | | | | |
| Visit neighbors by oneself | | 15.76 | 15.92 | 12.45 | 14.33 |
| Shop 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 | 0 = Yes; 0 = al little difficult; 1 = not able to do so. | 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 |
| <i>Functional Limitations</i> | | | | | |
| Put hand behind neck | | 5.13 | 5.57 | 5.37 | 4.28 |
| Put hand behind lower back | 0 = right; 0 = left; 0 = both; 1 = neither | 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 | | 8.33 | 8.6 | 6.93 | 5.72 |
| Pick up a book from the floor | 0 = Yes; 1 = No | 10.7 | 11.89 | 9.3 | 9.21 |
| <i>ADLs</i> | | | | | |
| Bathing | | 25.71 | 20.82 | 13.11 | 19.59 |
| Dressing | | 11.66 | 10.7 | 7.42 | 10.28 |
| Toileting | 0 = without assistance; 1 = need assistance | 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 |

Notes:

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 which includes more than 10 percent of respondents didn't answer this question in 2002.

Figure S1. Attrition Descriptions in CLHLS 2002 – 2011



Note: Sample size of respondents survival in 2005, 2008 and 2011 (shown in Figure S1) is larger than analytical sample (in Table S1), because those with missing values in frailty index are excluded in model estimation.

Table S2 Description of Variables by Birth Cohorts in CLHLS 2002 - 2011

| | Before 1900 | 1900-1904 | 1905-1909 | 1910-1914 | 1915-1919 | 1920-1924 | 1925-1929 | After 1930 |
|-----------------------------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|
| | Cohort 1 | Cohort 2 | Cohort 3 | Cohort 4 | Cohort 5 | Cohort 6 | Cohort 7 | Cohort 8 |
| | (N=894) | (N=2,278) | (N=1,855) | (N=2,035) | (N=2,145) | (N=1,606) | (N=1,518) | (N=2,286) |
| Variables | Mean (s.d.) | Mean (s.d.) | Mean (s.d.) | Mean (s.d.) | Mean (s.d.) | Mean (s.d.) | Mean (s.d.) | Mean (s.d.) |
| Age | 103(.99) | 100(1.36) | 94(1.45) | 89(1.53) | 84(1.45) | 80(1.55) | 74(1.49) | 68(2.14) |
| <i>Frailty Index</i> | | | | | | | | |
| 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) |
| <i>Childhood Adversity</i> | | | | | | | | |
| 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) |
| <i>Covariates</i> | | | | | | | | |
| 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) |