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Cohort-based Income Status and Population Health

A dissertation submitted in partial satisfaction of the requirements
for the degree Doctor of Philosophy

in

Public Health (Health Behavior)

by

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2016

The Dissertation of Jongho Heo is approved, and it is acceptable in quality and form for publication on microfilm and electronically:

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Chair

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2016

DEDICATION

To my Lord,
to my blessed family,
and to my lovely wife, Jiyoun

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Chapter 2, in full, will be submitted for publication of the material as it may appear in the *American Journal of Public Health*: Jongho H, Suzanne L, Shih-Fan L, Audrey NB, and Brian KF: Cohort-Based Income Gradients in Obesity among U.S. Adults. Jongho Heo was the primary investigator and author of this paper.

Chapter 3, in full, will be submitted for publication of the material as it may appear in the *International Journal of Epidemiology*: Jongho H, Suzanne L, Shih-Fan L, Audrey NB, and Brian KF: Economic Conditions in Early Life and the Risk of Adult Mortality. Jongho Heo was the primary investigator and author of this paper.

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ABSTRACT OF THE DISSERTATION

Cohort-based Income Status and Population Health

by

Jongho Heo

Doctor of Philosophy in Public Health (Health Behavior)

University of California, San Diego, 2016
San Diego State University, 2016

Professor Suzanne Lindsay, Chair

Background: Research has shown that income is a fundamental determinant in population health; however, little research has examined the relationship between income and health in a birth-cohort dimension.

Objectives: Based on the fundamental cause theory and life-course theory, we examined income gradients in obesity among whites and blacks across cohorts (chapter 1) and assessed the effects of economic conditions in early life and income inequalities in early life on the risk of all-cause and cause-specific mortality (chapter 2 and 3).

Methods: In chapter 1, we fitted a series of logistic hierarchical Age-Period-Cohort models using the National Health and Nutrition Examination Survey (1971-2012). Predicted probabilities of obesity by poverty income ratio were estimated based

on the models and graphed for 5-year cohort groups from 1901-1990. We also stratified this relationship for four gender and race/ethnicity subgroups. For chapter 2 and 3, we employed parametric frailty survival models using linked General Social Survey and National Death Index data (1979-2008). Gender-stratified analyses were conducted on the all-cause and disease-specific mortality. To better understand the underlying mechanisms, we further examined interactions between the macro-level income status in early life and parental education on adult mortality.

Results: Analyses in the chapter 1 revealed that the weaker income gradients in obesity were found in post-world war generations, mid-1920s, mid-1940s, and 1950s cohorts (absolute index: 0.03-0.08; relative index: 1.14-1.35), than the other cohorts. Moreover, cohort-based income gradients in obesity vary markedly by gender and race/ethnicity. In the second study, we found that exposure to recession in the first year of life was associated with an increase all-cause mortality only among women (hazard ratio=1.54, 95% CI 1.03-2.31) and mortality from cancers (hazard ratio=2.24, 95% CI 1.18-4.28). In the third study, we found that a higher level of income inequalities in early life was associated with increased risk of all-cause mortality among males (hazard ratio=1.038-1.041, 95% CI 1.001-1.070) and mortality from various diseases. Paternal education moderates these relationships for the second and third study.

Conclusion: Cohort-specific strategies based on life-course approach are needed to improve population health status and tackle socioeconomic disparities in health.

CHAPTER 1: Introduction

Health disparities are defined by the Health Resources and Services Administrations as "population-specific differences in the presence of disease, health outcomes, or access to health care" (HRSA Working group for the Eliminiation of Health Disparities 2000)—populations are usually defined with respect to advantaged/disadvantaged status in the U.S. and include such populations as racial/ethnic minorities and women. Despite that the mortality rates in the United States have declined dramatically over the past century, disparities across racial and gender subgroups still resist change. For example, while the cause-specific mortality rate is declining for the entire U.S. population, blacks continue to experience a higher death rate compared to whites: black persons still live approximately six fewer years (Carvalho et al. 1989).

Genetics plays an important role in determining the health of individuals; however, as most genetic variation is within—rather than between—populations (Evans, Hodge, and Pless 1994). The determinants of population health have very little to do with genetics. The case of racial and gender disparities is no exception (Cooper 1984, Goodman 2000). One of the primary impediments for reaching the goal of health equity is the lack of understanding of which social processes generate health disparities and how these disparities persist amidst declining morbidity and mortality rates.

Critiques of socio-epidemiological studies on health and health disparity

Epidemiological evidence on mortality has been unsatisfactory in analyzing socioeconomic factors contributing to mortality disparities because such evidence over-emphasizes individual-level socioeconomic characteristics (Berkman, Kawachi, and Glymour 2014). These include lower income, job loss, and lower educational attainment (Gravlee 2009, Marmot 2005). One of the concerns for such an approach is that greater risk of mortality is thought to be caused by lower SES and lower SES is solely attributed to individual diligence or will (Berkman, Kawachi, and Glymour 2014). It is also expected that once the healthful information or skills are provided, individuals will alter their behaviors accordingly. However, public health efforts to change individuals' lifestyles based on such assumptions have generated very disappointing results (Lorenc et al. 2013, White, Adams, and Heywood 2009). Moreover, poorly designed individual-level interventions may actually widen health inequalities by benefiting those who have resources to participate in such interventions or to alter their behaviors (Lorenc et al. 2013).

Thus, it is increasingly recognized that this individual risk factor approach alone will not be effective in reducing health gaps across subgroups (Berkman, Kawachi, and Glymour 2014). Because health disparities are generated through social inequalities, we need to describe and explain disparities that lie far upstream from the individual-level and is firmly rooted in the social context (Phelan et al. 2004, Link and Phelan 1995). In other words, effective strategies to narrow the health gaps need to identify macro-level determinants of population health and to take actions at multiple levels: from individuals to society (Marmot 2005).

Another critique is that most health disparity research takes into account only individual-level characteristics when examining "static" health disparities (Beck et al. 2014). However, when considering that population composition and social forces are constantly changing, it is necessary to apply a demographic lens to look at health disparities by investigating changes over time and across generations (Yang and Land 2008).

Theoretical background: Fundamental Cause Theory and Life-Course Theory

In response to the above critiques, this dissertation is based on two theories: fundamental cause theory and life-course theory. Fundamental cause theory regards social conditions as the fundamental causes of health conditions in the population (Phelan et al. 2004). If social conditions are fundamental causes of health and disease, then social conditions are surely fundamental causes of health disparities because social and environmental factors differentially affect population subgroups (Link and Phelan 1995).

Fundamental cause theory regards socioeconomic status as the fundamental cause of health because the association between SES and health persists regardless of changing mediators, and the association is relevant for virtually all health outcomes (Phelan and Link 2005). Thus, the theory stresses the importance of broader social and environmental contexts related to SES rather than intervening on proximal individual-level risks to reduce socioeconomic inequalities in health (Link and Phelan 1995, Phelan et al. 2004). This theory pays attention to resources such as knowledge, money, power,

prestige, and beneficial social connections that can be used to improve health and avoid disadvantages to maintaining health. Thus, the critical question to understand disparities is who gets what in terms of risk and protective factors, and why they get it (Williams and Collins 2001).

Life-course theory refers to a multidisciplinary paradigm for the study of people's lives, structural contexts, and social change (Elder Jr, Johnson, and Crosnoe 2003, Elder 1998). The theory focuses on the joint effects between one's life pathways and historical and socioeconomic contexts. The theory also underscores the cumulative effects of life trajectories through a given period and birth years. Thus, this theory recognizes the role of time in shaping health outcomes and incorporates time into models explaining health outcomes (Wethington 2005). For example, men's adult mortality risk was associated with socioeconomic conditions during childhood (Hayward and Gorman 2004).

Cohort analysis

Based on these two theories, I focused on birth cohort effects in the dissertation. If individuals born during similar periods and entering into pre-existing social systems, they can be regarded as a birth cohort (Yang 2007b). Thus, cohort effects represent the effects of exposure early in life and act persistently over time to produce health and mortality risk differences in specific cohorts (Yang 2008a). While birth cohorts move through the life cycle together and experience similar historical and social events, every cohort differs in their exposure to socioeconomic, behavioral, and environmental risk

factors leading to health disparities across population subgroups (Yang 2008b, O'Brien 2000).

As we categorize generations such as baby boomer, X-generation, or Y-generation, each birth cohort may have its own health outcomes, which are shaped by unique experiences in its life trajectories (O'Brien 2000). As such, members of birth cohorts move through the life cycle together and experience similar historical and social events; however, gender and racial groups in each cohort may be different in their exposure to the events leading to gender/racial health disparities (Ogden 2009, Beck et al. 2014).

Methodological background: demographic framework considering age, period, and cohort dimensions

To study cohort effects on health and health disparities, we need specific methodological skills to consider three different time scales at the same time: age, period, and cohort (Beck et al. 2014, Yang 2007b, Yang and Land 2008). Until quite recently, it has been methodologically difficult to separate the three time scales because there is an exact linear dependence between age, period, and cohort, which is called the Model Identification Problem (Yang 2007a). To solve the problem, I adopted cutting-edge multilevel analytic methods and used multiple cross-sectional surveys to break this perfect linear dependence.

Dissertation goals and research questions

Based on these backgrounds, the dissertation represents a systematic attempt to understand and explain how cohort-based income status affects population health using multiple years of nationally representative U.S. data and combining theory and methods from demography, sociology, and public health. The dissertation also critically examines the contribution of the cohort effects on health disparities across gender and racial subgroups.

My first investigation, entitled "Cohort-Based Income Gradients in Obesity among U.S. Adults," aimed 1) to examine income gradients in obesity by five-year birth cohorts from the 1890s to the 1990s and 2) to explore income-obesity gradients by gender and race/ethnicity. My research questions were:

1. Do income gradients in obesity differ across cohorts in both magnitude and direction?
2. Do the cohort-based income gradients in obesity differ across groups stratified by race and gender?
3. Does Fundamental Cause Theory hold in the study?

In my second study, I assess the effects of economic conditions in early life, as well as their interaction with parental education, on the risk of adult mortality in the U.S. Research questions were as below:

1. What are the associations between economic conditions in early life and all-cause mortality?
2. Are there gender differences in the associations?
3. Are the associations related to specific-cause of death?

4. Are there any parental moderating influences in the associations?

My third study assesses the effects of income inequalities in early life, as well as their interaction with parental education, on the risk of adult mortality in the U.S. Research questions were as below:

1. What are the associations between income inequalities in early life and all-cause mortality?
2. Are there gender differences in the associations?
3. Are the associations related to specific-cause of death?
4. Are there any parental moderating influences in the associations?

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Chapter 2: Cohort-Based Income Gradients in Obesity among U.S. Adults

Abstract

Background: Research has documented income gradients in obesity at single points in time confounding period and cohort effects. Identifying distinct temporal patterns remains crucial to identifying the source of changes over time.

Methods: This study examines cohort-based income gradients in obesity among whites and blacks using the National Health and Nutrition Examination Survey (1971-2012). We fitted a series of logistic hierarchical Age-Period-Cohort models to control for the effects of age and period, simultaneously. Predicted probabilities of obesity by poverty income ratio were estimated and graphed for 5-year cohort groups from 1901-1990. We also stratified this relationship for four gender and race/ethnicity subgroups.

Results: Analyses revealed that the weaker income gradients in obesity were found in post-world war generations, mid-1920s, mid-1940s, and 1950s cohorts, than the other cohorts. Moreover, income gradients of obesity across cohorts vary markedly by gender and race/ethnicity. White women consistently showed negative relationships between income and likelihood of obesity across birth cohorts; whereas, black men showed positive relationships in most cohorts.

Conclusions: We found that inter-cohort differences in the income gradients in obesity and intra-cohort differences in the gradients across gender and race/ethnicity subgroups. Intra-cohort gender and race disparities in obesity contributed to inter-cohort variations. Our findings suggest that cohort-specific strategies based on life-course approach are needed to tackle socioeconomic disparities in obesity among targeted gender and race/ethnicity groups.

INTRODUCTION

Epidemiological studies have shown that the relationship between socioeconomic status (SES) and health is not simply a threshold effect, but a graded association (i.e., gradient) (Baltrus et al. 2005, Adler et al. 1994). It is generally thought that people of higher SES enjoy longer, healthier, and happier lives than those of lower SES (Adler et al. 1994, Deaton 2002, House et al. 1994, Link and Phelan 1995). This relationship is detailed in the fundamental cause theory (FCT) (Phelan, Link, and Tehranifar 2010, Link and Phelan 2010, 1995). The theory deems SES a "fundamental" cause of health inequalities as the association between SES and health persists regardless of changing mediators and is relevant for all virtually all health outcomes (Link and Phelan 2010, Phelan, Link, and Tehranifar 2010). SES facilitates or inhibits access to a wide range of flexible resources, such as money, knowledge, prestige, power, and social connections that protect health even when the profile of risk factors and diseases change over time (Link and Phelan 2010). Thus, the theory stresses the importance of broader social and environmental contexts related to SES rather than intervening proximal individual-level risks to reduce socioeconomic inequalities in health (Freese and Lutfey 2011).

Recent studies testing the FCT generally confirmed the premise yet also uncovered a peculiar challenge to its tenets (Miech et al. 2011, Polonijo and Carpiano 2013, Chang and Lauderdale 2009). Many studies found that SES evolves throughout the life course for individuals (Willson, Shuey, and Elder Jr 2007), and can result in divergent historical and contextual experiences for different birth cohorts (Masters,

Hummer, and Powers 2012, Masters, Link, and Phelan 2015). Previous studies have failed to examine how socio-economic health disparities emerge, diminish, or widen, over time (Miech et al. 2011). More research is needed to integrate FCT with other intersectional social dimensions of health inequality by adopting a temporal view of SES and health (Miech et al. 2011, Yang and Lee 2009).

The FCT is also relevant for the relationship between SES and obesity: in general, higher income groups have a lower risk of obesity (McLaren 2007). To date, a great deal of cross-sectional studies have documented socioeconomic gradients in obesity that have not focused on period and birth-cohort effects (Reither, Hauser, and Yang 2009); little attention has been devoted to examining the long-term trends of income gradients by birth cohort.

A birth cohort moves through life together and encounters similar historical changes such as social, economic, and technological events at the same ages (Reither, Hauser, and Yang 2009, Yang and Land 2013b). Birth-cohort effects lead to generational differences between individuals who are born in different years due to their differential exposures at various stages of the life course (Yang and Land 2008). Examining birth-cohort effects in obesity is critical as individuals gain weight cumulatively across the life span, a trajectory that is linked to formative environments and exposure to disparate social conditions (Yang and Land 2013b). Recently, studies have realized the importance of birth cohort effects in the analysis of obesity trends that are independent of age and period effects (Reither, Hauser, and Yang 2009, Sassi et al. 2009, Allman-Farinelli et al. 2008, Diouf et al. 2010). Analyzing birth cohort effects in obesity is

supported by past studies which showed that the influences of lifestyle factors and the effects of early advantage or disadvantage contributes to weight accumulation over the life course, thereby increasing heterogeneity within cohorts (Freedman et al. 2005, Guo et al. 2000, Dietz 1994, Parsons et al. 1999).

Very little research has examined socioeconomic disparities in obesity across cohorts. Among SES factors, income is essential to behaviors related to weight gain/loss given its relationship with caloric intake and physical activity; income also reflects access to resources and the ability to capitalize on new health technologies (Link and Phelan 1995). The types of resources and the timing of the access toward them may differ across cohorts as contexts change over time, for example, as technology and knowledge advances. In fact, more recent birth cohorts adapt more easily to social changes caused by cultural transitions and technological innovations (Ryder 1965); whereas, older cohorts are more resistant to social changes (Allman-Farinelli et al. 2009). Combined, associations between income and obesity may result in differential income gradients in obesity across cohorts in both magnitude and direction if we consider social changes and the vastly different historical and life experiences of birth cohorts during the 20th century. Thus, we hypothesize that relationship between income and obesity may produce different patterns across birth cohorts.

Previous cross-sectional research has found substantial heterogeneity in obesity gradient patterns and trends across subgroups stratified by gender and race/ethnicity (Sanchez-Vaznaugh et al. 2008, Sánchez-Vaznaugh et al. 2009). Although there are differences in methodology across the studies (e.g., obesity outcome definition,

operationalization of income status, and data source), growing evidence consistently showed a stronger inverse association between income and obesity especially among non-Hispanic white women, whereas, patterns among men were largely inconsistent (Sobal and Stunkard 1989, McLaren 2007, Chang and Lauderdale 2005, Singh et al. 2011). On the other hand, the ability to convert income into better health is significantly contextualized in American society by one's race/ethnicity and gender (Colen 2011, Crimmins, Hayward, and Seeman 2004, Williams et al. 2010). For example, consumption behaviors, which affect obesity, may differ according to social norms or preferences of body size or beauty within subgroups by gender and race/ethnicity (Sánchez-Vaznaugh et al. 2009). Thus, a certain subgroup of a specific birth cohort may tend to adopt an obesity-promoting lifestyle based on their income, whereas, another subgroup may tend to use income to minimize the risk of obesity, along with the different patterning between cohorts.

To test these hypotheses, we investigated how cohort–based income gradients in obesity varied among the U.S. adult population using nationally representative data from the National Health Nutrition and Examination Survey (NHANES). We aimed 1) to examine income gradients in obesity by five year birth-cohorts from the 1890s to the 1990s and 2) to explore income-obesity gradients by gender and race/ethnicity.

METHODS

Study population

We used pooled data from the NHANES I (1971-1975), NHANES II (1976-1980), NHANES III (1988-1994), and NHANES Continuous (1999-2012). The NHANES uses a complex, multistage, and clustered sampling design to provide cross-sectional and nationally representative data on the health and nutritional status of the civilian non-institutionalized U.S. population. We excluded children, adolescents, and pregnant women in this study. Given our primary interest in comparing the income gradient patterns between non-Hispanic white (hereafter white) and non-Hispanic black (hereafter black) populations across cohorts in our analyses, we restricted our sample to whites and blacks according to the racial/ethnic identification variable in the NHANES. We further excluded missing values in the dependent variables, respondents' body mass index (BMI; 4.1%) and primary independent variable, the poverty income ratio (PIR; 12.0%) from all of our analyses. The final sample of our study consists of 56,820 adults aged 18-80 years. The racial distribution of our sample is as followed: 35.4% (n= 20,144) white men; 37.9% (n= 21,545) white women; 12.7% (n= 7,196) non-Hispanic black men; and 14.0% (n= 7,935) black women. Table 2.1 presents descriptive statistics of person-level samples.

Measurement

Outcome: obesity

Obesity was dichotomized based on the respondents' BMI, calculated as weight in kilograms divided by height in meters squared (WHO 2000). We considered a respondent was obese if his or her BMI was at least 30 (WHO 1995, 2000). Weight and height measurements were collected by interviewers through physical examination in a

mobile examination center (CDC 2010).

Main determinant: economic status

To measure the economic status of respondents, we employed the poverty-to-income ratio (PIR), rather than income, which has different range categories across survey waves. The PIR is based on the ratio of household income to the poverty threshold of each year after, accounting for inflation and family size. The PIR was entered as a continuous variable in our models (range: 0-5). We quantitatively measured slopes (i.e., inequalities) using both the regression-based absolute and relative index (Mackenbach and Kunst 1997, Wagstaff, Paci, and Van Doorslaer 1991) as recommended in health disparity literature (Braveman 2006, Keppel et al. 2005). The absolute index provides a measure of the absolute size of inequalities calculated by the difference between the probabilities estimated for those at the lowest and those at the highest ends of the PIR. The relative index is the ratio of the probabilities estimated for those at the lowest and the highest ends of the PIR. The relative index, although it is dimensionless, may be preferable for understanding change over time as it is not sensitive to the overall prevalence of obesity within a country (Pamuk 1985, Mackenbach and Kunst 1997).

Covariates

Consistent with previous studies (Devaux and Sassi 2012, Mackenbach et al. 2008), we used age, education level, marital status, employment status, birthplace, and smoking status as covariates. Age was measured in single year increments. We centered

age based on the median age of the cohort to ensure age estimates which are unbiased with respect to cohort (Rasbash et al. 2000). A quadratic age term was used to reflect the concave relationship between age and obesity (Reither, Hauser, and Yang 2009, Bell and Jones 2014). Education level was measured with four categories: less than high school degree (reference), high school degree, college degree, and more than college degree. Marital status was measured with three categories: married (reference), never married, and other status (widowed, divorced, and separated). Employment status of respondents was categorized by employed (reference), unemployed, retired, and other. Birthplace was dichotomized as U.S. born and foreign born. We used smoking status as a control variable which has three categories: non-smoker (reference), current user, and past user. Periods were measured according to the survey year.

To examine the income gradients in obesity across cohorts, we collapsed respondents' birth years into 5-year cohort groups. This 5-year grouping is conventional in birth cohort studies in demography to ensure a sufficient sample size within each cohort (Yang 2008, Reither, Hauser, and Yang 2009). However, we excluded the earliest cohort (1895-1900) from our analyses due to its small sample size (0.6%) to avoid a distortion in the estimates for this cohort.

Statistical analysis

We used a series of logistic hierarchical Age-Period-Cohort (HAPC) regression models with cross-classified random-effects (CCREMs) to account for individuals nested within cohorts and periods (Yang and Land 2008, Kawachi and Berkman 2003). As detailed in Yang and Land (Yang and Land 2013b), we fit the model that estimates

age and its quadratic term as fixed, while estimating periods and birth cohorts as random effects. Our basic model was as follows:

Level 1 (within-cell) model:

$$\text{logit}\left\{Pr\left(Y_{ijk}=1\middle|A_{ijk,}A_{ijk}^{2}\right)\right\}=\ \beta_{0jk}+\beta_{1}A_{ijk}+\ \beta_{2}A_{ijk}^{2}$$

where Y_{ijk} represents whether a respondent is obese or not for the i^{th} participants for i=1,…, n_{jk} within cohort j and period k. A_{ijk} and A_{ijk}^{2} represent age and age-squared, respectively.

Level 2 (between-cell) model:

$$\beta_{ojk}=\gamma_{0}+\ u_{0j}+v_{0k},\ u_{0j}\sim N(0,\tau_{u}),\ v_{0k}\sim N(0,\tau_{v}),$$

β_{ojk} indicates that the overall mean varies for each period of observation j and each birth cohort k. γ_{0} and is the model intercept, which is the expected mean when all level-1 variables are averaged across all periods and cohorts. u_{0j} is the residual random effect of cohort j and is assumed to be normally distributed with mean 0 and a within-cell variance τ_{u}. v_{0k} is the residual random effect of period k and is assumed to be normally distributed with mean 0 and a within-cell variance τ_{v}.

We chose this model based on our preliminarily cross-validated model selection statistics (Table 2.2). According to the recommendations from Reither et al (Reither, Hauser, and Yang 2009) and Yang and Land (Yang and Land 2013a) for modeling decision, we presented log likelihood, Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), and degrees of freedom for various APC models. The

cross-classified random-effects models (CCFEMs) estimate effects of the cohorts and periods are assumed fixed and unique to each of the respective cohorts and periods, The fixed effects of the cohorts and periods are estimated post-hoc by the incorporation of two sets of indicator/dummy variables for J − 1 cohorts and K − 1 periods. Both the AIC and BIC suggest that the age-period-cohort model was preferred to all other models. To ensure that the CCREM is appropriate for our data, we conducted a Hausman specification test to compare results from fixed- and random-effects models. We failed to reject the null hypothesis of no difference in coefficient vectors (x^2= 1158.69; p <0.001), confirming that the CCREM is preferable.

First, we estimated and graphed the predicted probability of obesity for each level of PIR across the five-year cohort groups for the whole population. Subsequently, to examine the gender/racial-specific income gradient across the cohorts, we stratified the above analysis for each of the four gender and racial subgroups. To stratify the analysis by gender and race/ethnicity, we tested for an interaction between PIR and gender in the race/ethnicity-stratified analysis. We estimated a logit CCREM of obesity for the U.S. population using Stata version 14 *meqrlogit*.

Sensitivity analyses

Sensitivity analyses were conducted to assess the extent of the bias effects on regression results due to a negatively skewed PIR distribution (13.4%). Results from the sensitivity analysis suggested that there were few differences between models.

RESULTS

Based on our tests for a three-way interaction between PIR and gender in the race/ethnicity-stratified analysis (results are available by request), we calculated the predicted probabilities and prevalence of obesity across birth cohorts within race/gender subgroups.

Figure 2.1 presents predicted income gradients in obesity by 5-year birth cohorts among black and white adults. The lengths of the bars in figure represents the strength of the gradient or the magnitude of obesity inequality due to income. Figure 2.1 (a) shows the overall relationship between income and obesity since 1900s has been consistently negative, but the magnitude of income inequality in obesity varied across birth cohorts. Notably, the inequalities were narrowed in post-world war cohorts: mid-1920s (absolute index: 0.03; relative index: 1.14) and during mid-1940s and 1950s (absolute index: 0.06-0.08; relative index: 1.33-1.35) (Figure 2.1 (c)).

Figure 2.2 presents graphs showing the income gradients in predicted probabilities in obesity and obesity prevalence across 5-year birth cohorts within four race/ethnicity-gender subgroups. Figure 2.3 provides the absolute and relative index for the gradients in the Figure 2.2. It is evident that the strength and the direction of income gradients varied by gender and race/ethnicity within cohorts. White women showed robust and negative relationship between income and likelihood of obesity across birth cohorts (absolute index: 0.06-0.26; relative index: 1.33-3.22). Whereas, black men, except for several cohorts, showed positive relationships.

DISCUSSION

Our primary goal was to document cohort-based income gradients in obesity of U.S. adults by 5-year birth cohorts during the 20th century net of age and period effects, and then to explore income gradients in obesity after stratifying by gender and race/ethnicity. We found that there were weaker income inequalities in obesity for post-world war cohorts (born in the mid-1920s, mid-1940s, and 1950s). Notably, the relationship between income and obesity differed markedly by gender and race/ethnicity across birth cohorts in terms of strength and direction. White women consistently showed negative relationships between income and obesity across birth cohorts; whereas, black men showed positive relationships in most cohorts.

Overall trends of income gradients in obesity by 5-year birth cohorts

Our study found that the post-war generations had weaker income gradients in obesity than the other cohorts. In terms of overall obesity prevalence, previous studies from France, Australia, and U.S. have consistently shown that post-world war generations had lower obesity risks than previous and subsequent generations after taking into account age effects and period effects (Reither, Hauser, and Yang 2009, Robinson et al. 2013, Diouf et al. 2010, Allman-Farinelli et al. 2008). Our study, with a view of obesity inequality, found that post-world war generations had weaker income related obesity inequalities than the other birth cohorts. Identifying causes of these income gradient patterns is outside of the scope of our study; however, the growing prosperity after World War I and II may reduce psychosocial and socioeconomic stress that may predispose the cohorts to have a weaker socioeconomic inequality in obesity

(Ribble and Keddie 2001). The periods of economic boom and consumerist expansion in U.S. following the two world wars were frequently referred as "Roaring Twenties" and "Golden Age of Capitalism", respectively. Especially, after the World War II, the middle class swelled as GDP growth and productivity sharply increased, and unemployment was dropped (French 1997). Thus, socioeconomic inequality was generally decreased during the economic boom with unusual financial stability. Given that early-childhood SES have latent biological or behavioral consequences that increase within-cohort inequality throughout adulthood (Gluckman and Hanson 2008, Lawlor et al. 2008, Ravelli et al. 1999, Yang and Lee 2009), such small social inequality in early life during post-world wars may also result in small income inequality in obesity in adulthood. In contrast, the nutritional deprivation and stress experienced by those born during the Great Depression may predispose these cohorts to have stronger obesity inequality, whereas obesogenic social and economic trends in the mid-1960s and 1970s may cause in stronger income gradients in obesity.

Gender and race/ethnic differences in the gradients in obesity

Many recent studies have shown that the association between SES and obesity varies by gender and race/ethnicity; however, they failed to separate cohort effects and period effects resulting in inconsistent findings across studies (McLaren 2007). Our study which decomposed successfully the two effects may partly resolve the inconsistencies in previous findings. Our study found that relationship between income and obesity differed markedly by gender and race/ethnicity across birth cohorts in terms of strength and direction. Thus, intra-cohort sex and race disparities in obesity may

contribute to inter-cohort variations over the life course. This may provide an important evidence that the relationships between SES and obesity can be highly contextualized in American society not only by one's gender and race/ethnicity but also by birth year (Williams et al. 2010, Colen 2011, Williams and Sternthal 2010). Although the mechanisms responsible for these gender- and race/ethnic-based changes within cohorts are not clear, such an intra-cohort differences may be driven by variation in aesthetic norms or cultural expectations with respect to weight standards (Chang and Lauderdale 2009, Chang and Christakis 2003).

We found that white women consistently showed negative relationships between income and obesity risk throughout the birth cohorts. Our study finding is in a line with past cross-sectional studies that have revealed strong inversed relationship between income and obesity among white women. We added to the current knowledge by showing the strong inversed relationship has lasted from 1900s. Previous studies showed that white women from higher SES group had a higher susceptibility for thinner-attractive body image compared to other gender-racial/ethnic groups (Paeratakul et al. 2002, Demarest and Allen 2000). Our finding may imply that the thinness has already been valued to white women since the early 20th century causing a large inversed correlation between income and obesity across cohorts. Given the pressure of maintaining thinness, white women may have internalized thinner body depicted in media as their ideal body in conforming to the social preference. Besides the psychosocial factors, behavioral factors including in diet, physical activity, and sleep as well as neighborhood factors may also account for the patterns of income gradients in

obesity among white women (Buxton and Marcelli 2010). For example, higher income groups among white women might be able to afford to live in safer and more physical activity-friendly neighborhoods and purchase more fruits and vegetables, weight control products, or gym memberships; whereas lower income group may have to live in an unsafe and obesogenic community and purchase more unhealthy foods (Lopez 2007, Hill and Peters 1998). Thus, income may have been a stronger preventer on obesity for decades among white women than other gender-racial/ethnic groups because they may be more willing to use income to stay thinner and effectively transform income into physically fit (Myers and Biocca 1992, Sypeck, Gray, and Ahrens 2004). Compared to white women, black women showed weaker relationships in most of the cohorts yet a higher risk of obesity than other racial/ethnic groups. This may be because of their more flexible cultural standard of attractiveness suggesting the minor role of income in the obesity disparities in black women (Celio, Zabinski, and Wilfley 2002). Our study also found consistent positive relationships between income and obesity risks among black men in most cohorts although inconsistent associations has been found among black male in previous studies. Those studies showed that it is more socially regarded for men to have larger body size as a sign of physical dominance and ability (Thompson, Sargent, and Kemper 1996, McVey, Tweed, and Blackmore 2005). To appear as rich and competitive in U.S. society that has a long history of racial discrimination, black men may have pursued physical dominance using income as a main resource to gain weight for last decades (McLaren 2007).

Our study has some limitations. Rather than an exact measure of income, we

used PIR, which is not an absolute indicator of real income. Although income data were available in the NHANES, inconsistent income categories across the survey waves precluded us from normalizing income data reported by respondents. Instead, the PIR was preferable because it was provided with consistent survey waves after controlling for household size and for inflation. Second, we were not able to control for specific period effects because the NHANES has survey gaps during 1981-1987, 1990, and 1994-1999 before the survey conducted annually. Thus, there could be residual confouding by period effects. Third, there may be a bidirectional causal relation between income and obesity because obesity may reversely affect income status by influencing access to education, occupation, and marriage (Gortmaker et al. 1993, Cawley 2000, Roehling, Roehling, and Pichler 2007). Lastly, this study is limited somewhat in estimating the income gradients in obesity by birth cohorts due to the small sample size of the earliest and last cohorts. Early cohorts were composed mainly by older samples, whereas recent cohorts by younger adult samples. Thus, based on the recommendations from previous studies (Yang and Land 2008, Snijders 2011), we did not estimate the income gradients in obesity in the oldest 1895-1899 cohorts. Especially, interpretation on the income gradients in obesity in the latest cohorts should be made carefully because the estimate is subject to revision as more data become available.

In summary, we found inter-cohort variation in the negative relationship between income and obesity, which is supportive of the FCT; however, we simultaneously found that there are inter-cohort variations with respect to race/ethnicity, which is potentially challenging to the FCT. Our findings imply that policies and

interventions should take into account cohort effects stratified by gender and race/ethnicity based on a temporally informed approach in order to attenuate SES inequalities in the obesity epidemic.

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Chapter 2, in full, will be submitted for publication of the material as it may appear in the *American Journal of Public Health*: Jongho H, Suzanne L, Shih-Fan L, Audrey NB, and Brian KF: Cohort-Based Income Gradients in Obesity among U.S. Adults. Jongho Heo was the primary investigator and author of this paper.

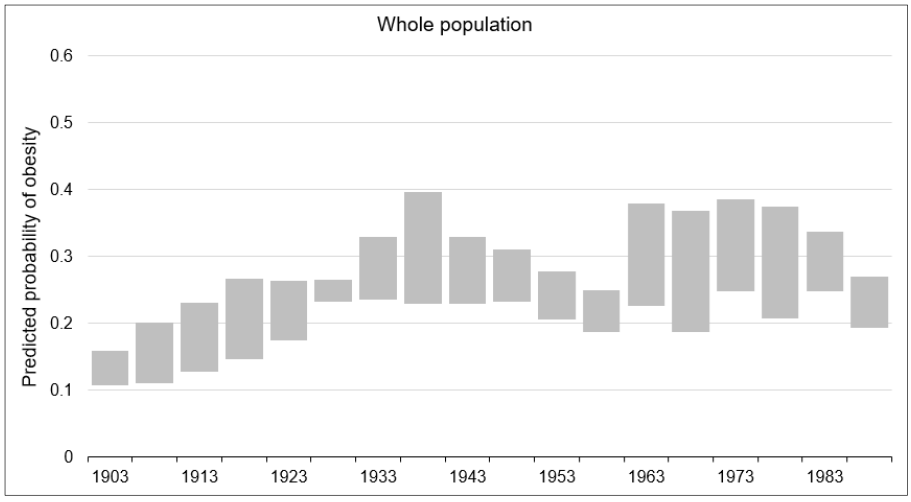
Table 2.1. Characteristics of respondents (N=56,820 adults) from all the wave of NHANES (1971-2012)

		Men				Women			
		White		Black		White		Black	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Obesity rate		22.4	0.4	26.8	0.4	26.8	0.4	43.7	0.5
Age		50.3	18.3	47.9	17.8	50.7	18.2	47.8	17.5
Poverty income ratio (PIR)		2.7	1.5	2.3	1.5	2.5	1.5	2.0	1.4
		N	%	N	%	N	%	N	%
Education	< High school	7,312	36.3	2,914	40.5	7,347	34.1	3,023	38.1
	=High school	5,378	26.7	2,044	28.4	6,722	31.2	2,317	29.2
	=College degree	3,787	18.8	1,504	20.9	4,374	20.3	1,746	22.0
	>College degree	3,666	18.2	734	10.2	3,102	14.4	849	10.7
Marital status	Married	20,144	68.5	3,353	46.6	12,367	57.4	2,539	32.0
	Never married	13,799	19.0	2,360	32.8	3,102	14.4	2,412	30.4
	Other	3,827	12.5	1,482	20.6	6,076	28.2	2,984	37.6
Employment	Employed	12,590	62.5	4,238	58.9	9,264	43.0	4,110	51.8
	Unemployed	2,639	13.1	1,749	24.3	7,067	32.8	2,419	30.5
	Retired	4,150	20.6	1,187	16.5	5,214	24.2	1,389	17.5
	Other	765	3.8	22	0.3	862	4.0	17	0.21
Smoking status	Current smoker	6,708	33.3	2,871	39.9	5,946	27.6	2,420	30.5
	Previous smoker	6,889	34.2	1,648	22.9	4,029	18.7	1,095	13.8
	Non-smoker	6,547	32.5	2,670	37.1	11,548	53.6	4,420	55.7
Birthplace	U.S born	16,438	81.6	6,563	91.2	18,141	84.2	7,348	92.6
	Foreign born	3,706	18.4	626	8.7	3,404	15.8	579	7.3
	N	20,144		7,196		21,545		7,935	

Table 2.2. Goodness-of-fit statistics for model selection statistics for NHANES data

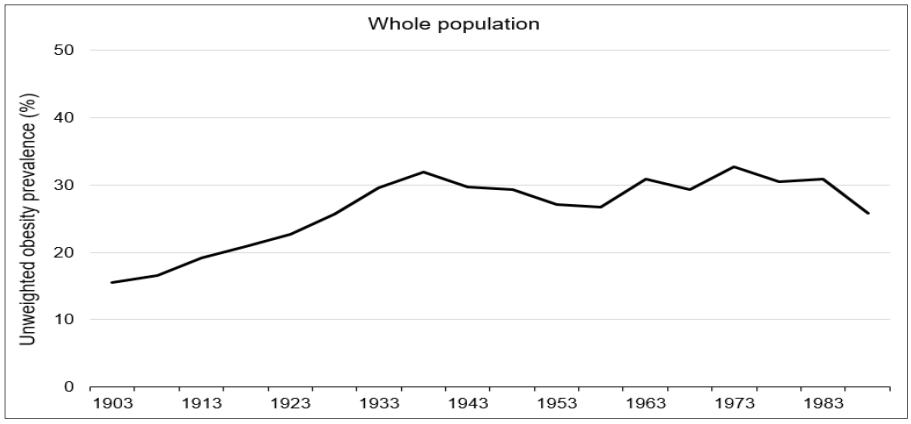
	Age	Age+period	Age+cohort	Age+period+cohort
Log likelihood	-34686.2	-33404.5	-33343.4	-33222.2
AIC	69378.4	66869	66728.8	66540.4
BIC	69386.7	66952.1	66787.0	66673.4
Df	3	30	21	48
N	58,937	58,937	58,937	58,937

Notes: AIC is the Akaike Information Criterion and is estimated to be -2*log(L) + df*2. BIC is the Bayesian information Criterion estimated to be -2*log(L) + df*log(N). The model fit statistics were estimated for unweighted cross-classified fixed-effects models (CCFEMs). Results using CCREMs shows consistent patterns with those above.



(a) Predicted income gradients in obesity (BMI≥30) across 5-year birth cohorts among U.S. adults

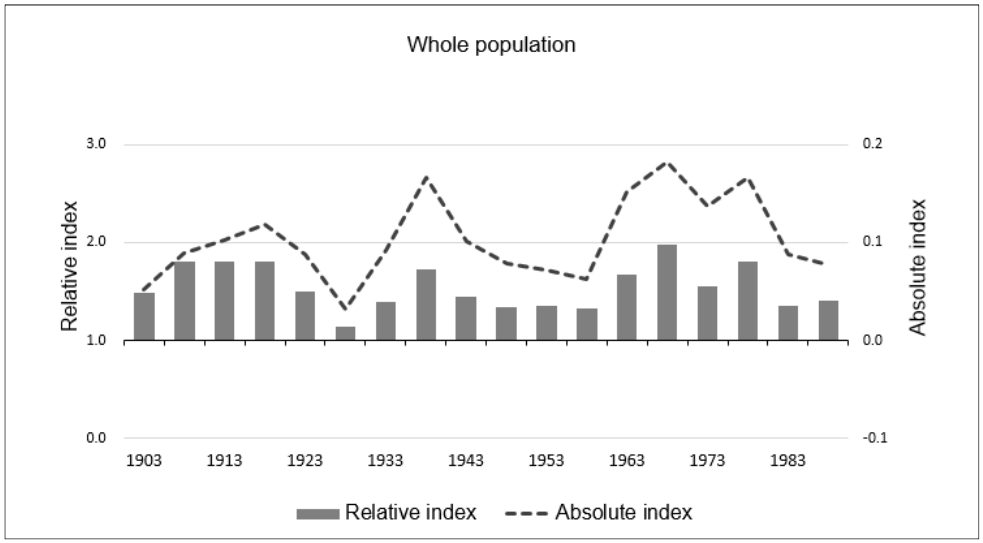
Note: The upper (lower) bound of each bar represents the predicted probability in obesity among the poorest (richest) group (poverty income ratio=0 to 5). Thus, the length of the bar represents the strength of the income gradient. The grey bar represents negative relationship between income and obesity.



(b) Unweighted obesity prevalence across 5-year birth cohorts among U.S. adults

Note: The solid line represents unweighted obesity prevalence across 5-year birth cohort.

Figure 2.1. Predicted income gradients in obesity, unweighted obesity prevalence, and absolute and relative index of income gradients in obesity across 5-year birth cohorts among U.S. adults (N=56,820) from the NHANES (1971-2012)



(c) Absolute and relative index of income gradients in obesity (BMI≥30) across 5-year birth cohorts among U.S. adults

Note: A bar or a dotted line above (below) the line of 1.0 in relative index and 0.0 in absolute index showed negative (positive) relationship between income and obesity risk.

Figure 2.1. Predicted income gradients in obesity, unweighted obesity prevalence, and absolute and relative index of income gradients in obesity across 5-year birth cohorts among U.S. adults (N=56,820) from the NHANES (1971-2012), Continued

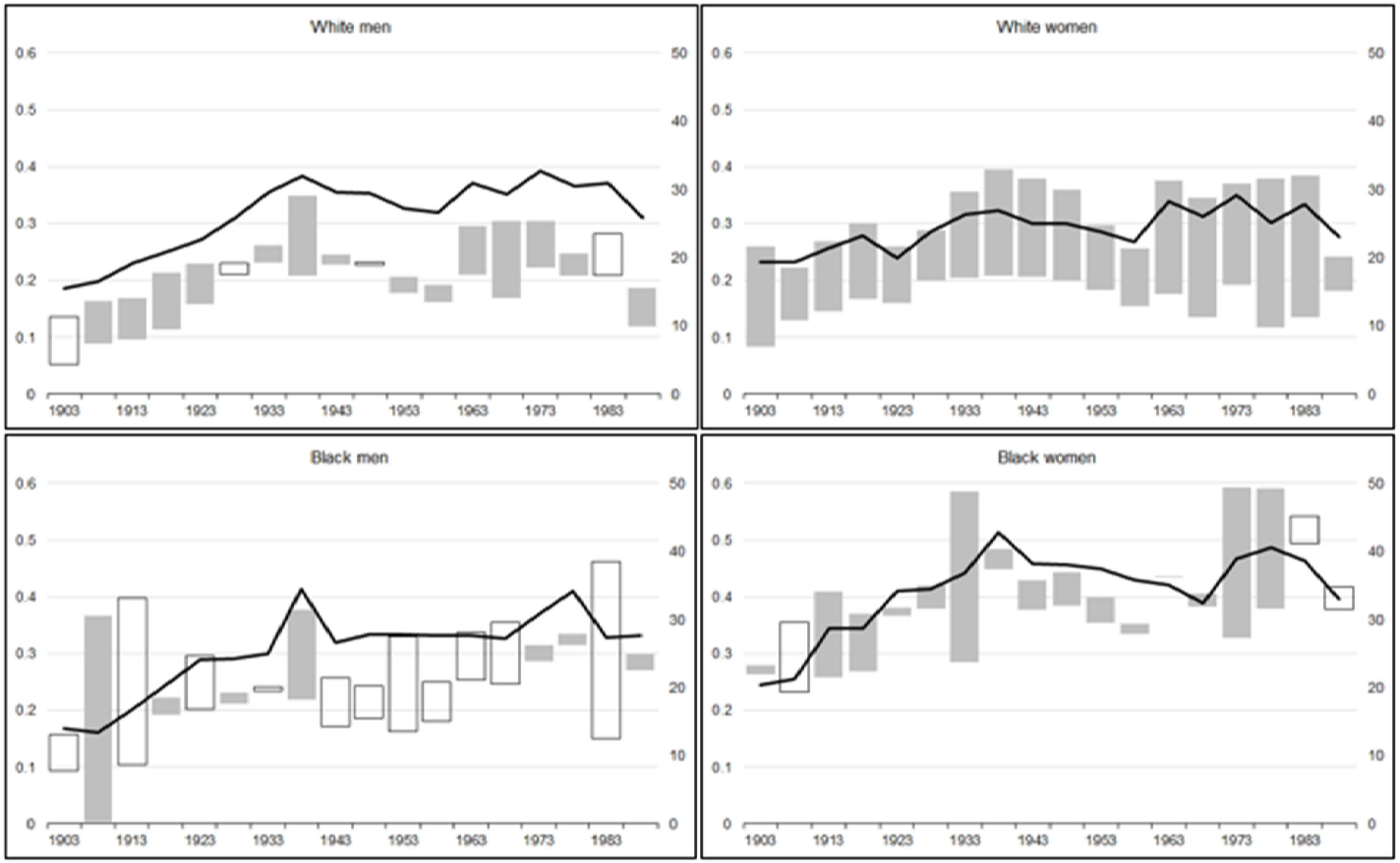


Figure 2.2. Predicted income gradients in obesity (BMI≥30) and the unweighted obesity prevalence across 5-year birth cohorts within four race/ethnicity-gender subgroups among U.S. adults (N=56,820) from the NHANES (1971-2012)

Note: The upper (lower) bound of each bar represents the predicted probability in obesity among the poorest (richest) group (poverty income ratio=0 to 5). Thus, the length of the bar represents the strength of the income gradient. The grey (hollow) bar represents negative (positive) relationship between income and obesity. The solid line represents unweighted obesity prevalence across 5-year birth cohort.

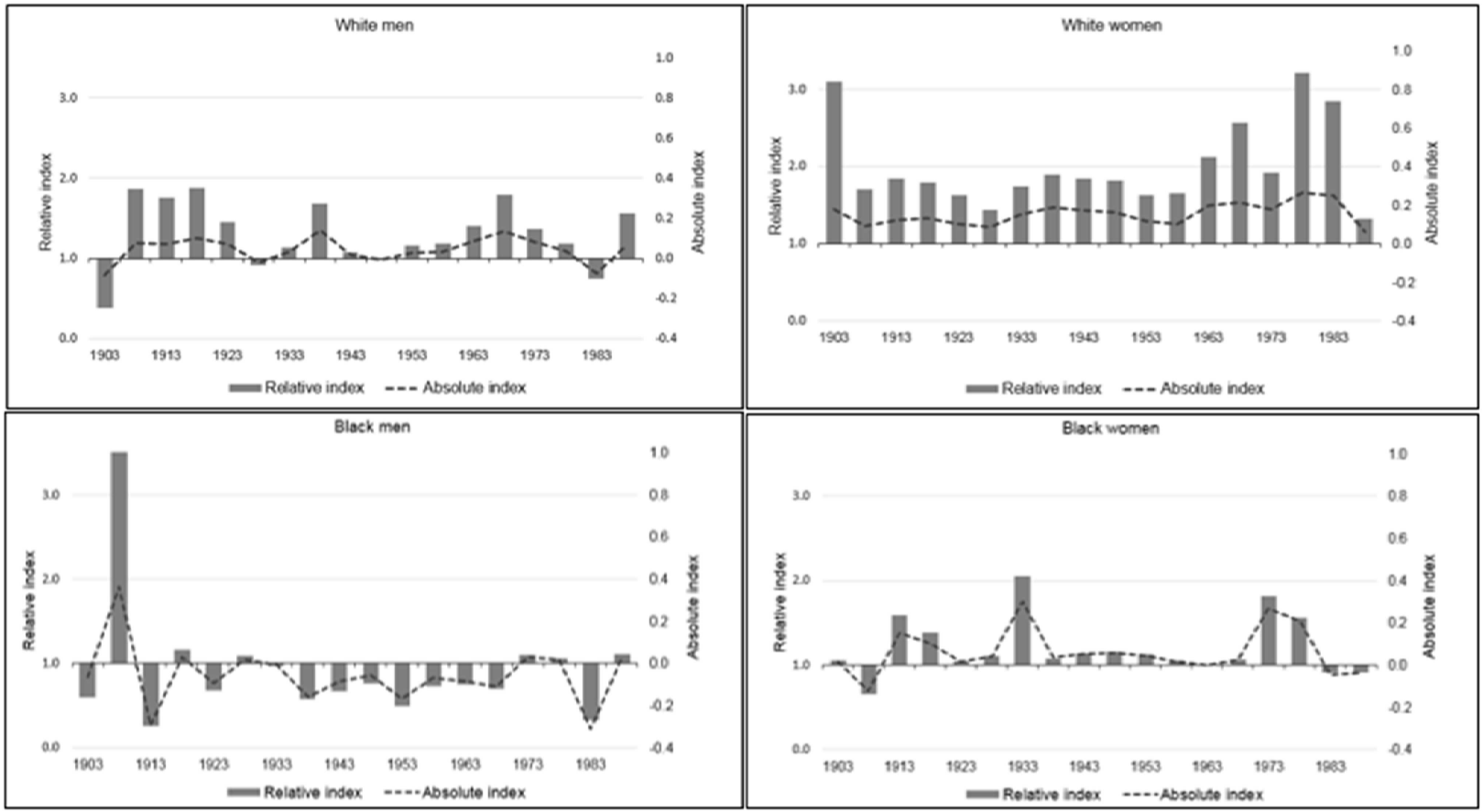


Figure 2.3. Absolute and relative index of income gradients in obesity (BMI≥30) across 5-year birth cohorts within four race/ethnicity-gender subgroups among U.S. adults (N=56,820) from the NHANES (1971-2012)

Note: A bar or a dotted line above (below) the line of 1.0 in relative index and 0.0 in absolute index showed negative (positive) relationship between income and obesity risk.

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Chapter 3: Economic Conditions in Early Life and the Risk of Adult Mortality

Abstract

Background: Empirical evidence from European countries has shown that economic conditions in early life are associated with risk of death. The aim of this study is to assess the effects of economic conditions in early life, as well as their interaction with parental education, on the risk of adult mortality in the U.S.

Methods: To capture exogenous variation of economic conditions early in life, we use the Gross Domestic Product cyclical deviation of the birth year. Using the linked General Social Survey and National Death Index data (1979-2008), we employed parametric frailty survival models to examine the effects of economic conditions in early life on all-cause and cause-specific mortality.

Results: We found that exposure to recession in the first year of life, but not in the birth year, or the year prior to birth, was associated with an increase all-cause mortality only among women (hazard ratio=1.54, 95% CI 1.03-2.31). This adverse effect was only found, specifically, for women's mortality from cancers (hazard ratio=2.24, 95% CI 1.18-4.28). We also found a significant interaction between economic conditions in infancy and paternal education on women's mortality risk—

higher paternal education was protective against mortality under good economic conditions in infancy; however, higher paternal education was associated with greater mortality risk under poor economic conditions in infancy. We discuss how aspiration theory may explain these results.

Conclusion: Our study concludes that women's risks of dying in later life are associated with worse macroeconomic conditions in early life, and paternal education moderates this relationship.

INTRODUCTION

Demographic studies have demonstrated that shared contexts within birth cohorts may affect mortality later in life (Masters et al. 2013, Yang 2008, Bray and Weiderpass 2010). The critical period model from the field of life-course epidemiology also recognizes the importance of early life for understanding the relationships between exposures to specific contexts and health at both the individual and population levels (Lynch and Smith 2005). Based on this model, researchers demonstrated that exposure to adverse nutritional or psychological conditions during or around pregnancy may be detrimental to children's health in the long run by triggering diseases in adulthood (Kuh and Shlomo 2004, Hayward and Gorman 2004, Galobardes, Lynch, and Smith 2004). However, most studies suffer from possible simultaneity bias, as individual variation in early-life conditions and later health may be jointly affected by unobserved heterogeneity.

To address this methodological issue, investigators have used GDP cycle near birth as an economic condition at early life, which is exogenuous at the individual level. Using this indicator, studies from European countries have consistently found that experiencing a recession in early life had significant negative effects on adult mortality. A study using Dutch data which followed cohorts from 1812 to 1912 found that individuals who were born during recessions lived a few years less than those who were born during boom periods (Van Den Berg, Lindeboom, and Portrait 2006). Another study using the Dutch data identified gender differences in adverse effects of recessions on cause-specific mortality (Yeung et al. 2014). A study using Danish data from post-

1870 birth cohorts also showed negative effects of poor economic conditions at birth on cardiovascular mortality but not on cancer mortality (Van den Berg, Doblhammer-Reiter, and Christensen 2011). These studies have consistently shown that economic conditions during early life have long-term impacts on mortality during adulthood. However few U.S. studies have explored the relationship between early-life economic conditions and adult mortality. Cutler, Miller, and Norton (2007) did not show a relationship between economic conditions in early life (measured by crop yield, income, and employment) and disability in later life among the U.S population born during the Great Depression. However, such studies focused on extreme events, such as the Great Depression, epidemics, or famine, which may lead to selection bias due to high infant mortality or fertility selection within cohorts, which reduces the generalizability of the findings (Catalano et al. 2011, Suhrcke and Stuckler 2012).

It is well established that lower parental education at early-life presents a significant risk to mortality (Hayward and Gorman 2004, Pensola and Valkonen 2002). However, it is unclear how parental education and economic conditions interact to affect parent health and how this effect extends to offspring mortality in the United States. During a recession, access to nutritious food, clothing, and housing for pregnant women and infants may be diminished, and household stress may be elevated. These effects may be moderated by household socioeconomic status. Being born during a recession may be less detrimental for families with richer socioeconomic resources that can act as a buffer.

To help fill the gap in this literature, this paper examines the effects of economic

conditions in early life on U.S. adult mortality in adulthood. We use mortality data that have been linked with birth-year GDP as an exogenous measure of economic conditions at birth. We fit parametric survival models with shared frailty to analyze the birth cohort-specific effects of all-cause mortality while controlling for individual socio-demographic variables. Additional analyses focusing on disease-specific mortality were also conducted. To better understand the underlying mechanisms, we further examined interactions between economic conditions in early life and parental education on mortality in later life. Previous studies have largely ignored the possibility that the complex interplay of biological processes, parental care behavior, and family-environments that shape adult mortality may operate differently for men and women. Given that there are gender differences not only in U.S. infant mortality but also gendered differences in familial relationships, work, and family role (Vance et al. 1995, Hill and Upchurch 1995, Cinamon and Rich 2002), we stratified our analyses by gender.

METHODS

Study population

We used two primary individual-level data sources: the General Social Survey (GSS) linked to the National Death Index (NDI). The GSS utilized repeated cross-sectional samples during 1972-2010 with annual surveys between 1972 and 1993 except 1979, 1981, and 1992; and biannual surveys between 1994 and 2010. We were able to link the GSS with the NDI covering the years between 1979 and 2008, yielding a sample

of 9,271 deceased individuals from a total sample of 32,830 respondents. The birth cohorts spanned 1889 to 1984. The process of data linkage is described in more detail elsewhere (Muennig et al. 2011). For each observation, we have a set of survey data for a given year as well as an indicator of whether subjects were alive as of 2008 and the number of days until death or censoring in 2008.

Measurement

Outcomes: all-cause and cause-specific mortality

The dependent variables for our analyses are all-cause and cause-specific mortality. Deaths were identified from the NDI and linked with the GSS dataset. The NDI dataset contains the Clinical Classifications Software (CCS) code for International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM). The CCS code is a diagnosis and procedure categorization scheme developed by collapsing the ICD-9-CM into clinically definable and mutually exclusive categories (Cost 2007). Based on the CCS Diagnosis Categories, we collapsed several disease groups to prevent results from being driven by only a few observations, we categorized a total of 231 codes to 11 disease categories: infectious and parasitic diseases (CCS 1-8); neoplasms (CCS 9-47); endocrine, nutritional, and metabolic diseases, immunity disorders, and diseases of the blood and blood-forming organs (CCS 48-64); mental illnesses (CCS 65-73); diseases of the nervous system and sense organs (CCS 74-95); diseases of the circulatory system (CCS 96-121); diseases of the respiratory system (CCS 122-134); diseases of the digestive system (CCS 135-155); diseases of the genitourinary system (CCS 156-161); suicide or self-inflicted injuries (CCS 662); and others including injury-

related deaths. Further, we focused on deaths from subgroups of neoplasms and diseases of the circulatory system, which are two leading cause of death categories.

Main determinant: economic conditions in early life

Economic conditions in early life were calculated by applying the Hodrick-Prescott filter (Hodrick and Prescott 1997) performing a trend/cycle decomposition of log annual real Gross Domestic Product (GDP) per capita with a smoothing parameter of 500 (Yeung et al. 2014, Van Den Berg, Lindeboom, and Portrait 2006). The filter has been used widely in macroeconomics and demography to capture clear transitory macroeconomic conditions of boom or recession by decomposing the GDP series into a long-term trend component and a stationary cycle (Hodrick and Prescott 1997). A positive (negative) deviation is interpreted as an economic upsurge (recession). The U.S. GDP deviation in our study ranged from -0.25 to 0.27. In order to capture the effects of recession in early life in our models, we switched positive GDP deviations to negative so that a unit increase in "worsening" economic conditions would be expected to increase the risk of mortality. As this study aims to examine birth cohort-specific effects on mortality in later life, we assumed that the GDP cycle of a year represents the economic conditions that affect those born in a given year regardless of birth month. We operationalized economic conditions in early life with GDP cycles at three time points: (1) the birth year-1, (2) the birth year, and (3) the birth year+1. Accordingly, we linked the GDP cycles with those year of life of respondents.

Covariates

Covariates include: age, age squared, race, marital status, employment status, education status of respondents and their parents, family income, and region. Age and family income were treated as continuous variables, and other independent variables were entered as dummies in our model. Age was a continuous variable as it was drawn directly from the GSS dataset. Total family income was coded per 1,000 dollars (e.g. if a respondent chose an $8,000-$9,999 category for the family income, we coded it as 9). Race/ethnic group had three categories: white (reference), black, and others including Hispanic, Asian, and Pacific Islanders. Marital status was measured with four categories: married (reference), never married, widowed, and divorced or separated. Employment status of respondents was categorized as working full time (reference), working part time, retired, unemployed, and other. The unemployed category includes those who were unemployed, laid off, looking for work, and have a job but not at work because of temporary illness, vacation, and strike. The final category includes being in school, keeping house, and other responses. Parental as well as respondents' education levels were measured by total formal education years completed. Region was categorized into West (reference), Northeast, South, and Midwest. We excluded individuals born outside the country (n=2,494). Missing information on age (0.1%), respondent's education (0.2%), paternal education (28.0%), maternal education (15.4%), and family income (9.4%) was imputed by replacing the mean age (45.3 years), the mean years of education (12.8 years), the mean years of education completed by mothers (10.8 years) and fathers (10.6 years) and the mean family income (18,988 dollars), respectively. Missing data for marital and employment status (0.02%) was replaced with the modal category. All

independent variables except for ECB were obtained from the GSS. The study sample for our analysis was 30,331 adults.

Statistical analysis

The present analyses used a multivariable parametric survival model with shared frailty. A shared frailty model is a random effects model that examines unobserved influences common to all members of a cluster (Gutierrez 2002, Guo and Rodriguez 1992). We used the model to know the extent to which individual's survival times are correlated within birth cohort, after considering the effects of observed covariates. We fit a Gompertz distribution to reflect survival times to individual mortality, and assumed a gamma distribution for the shared frailty. The Gompertz distribution (proportional hazard metric) has been widely used in mortality studies (Bender, Augustin, and Blettner 2005, Cleves et al. 2008) because it is adequate for modeling data with hazard rates that increase exponentially with time. The shared frailty model is essentially a random effects model for survival data in that it allows individuals to share the same latent frailty value among groups. The hazard rate for the i^{th} individual from the j^{th} group is,

$$h_{ij}(t \mid \beta_j, v_j) = h_0(t) w_{ij} \exp(\beta_j x_{ij}), \text{ where } w_{ij} = \exp(v_j z_{ij}) \quad (1)$$

$$h_{ij}(t \mid \beta_j, v_j) = \mathrm{h_0(t)exp}(\beta_j \mathrm{x_{ij}} + v_j z_{ij}) \quad (2)$$

$h_{ij}(t \mid .)$ is the conditional hazard function for the ith individual from the j^{th} group at time t, $h_0(t)$ is the baseline hazard function at time t, β_j is a vector of cohort-specific fixed-effects corresponding to a vector of covariates x_{ij}, v_i is a vector of

random effects associated with a vector of covariates z_{ij} and is also known as the subgroup frailty shared among the ith individual from the j^{th} group. v_j is assumed to be randomly distributed with mean 0 and variance-covariance matrix θ. Frailty is shared among individuals occupying the same birth cohort. The frailty follows a gamma distribution (Cleves et al. 2008, Gutierrez 2002); the variance of the frailty (θ, theta) was estimated by iterative maximum profile log-likelihood. The likelihood-ratio was tested to examine the significance of the shared frailty effect. We fit the models sequentially to examine the effects of GDP cycle during the birth year-1; during the first year of life; and during the birth year+1. To obtain further insights into the underlying mechanisms, we additionally analyzed the interaction effects between economic conditions in early life and paternal education as a proxy variable to represent early family environments. All our shared frailty models were unweighted as the GSS-NDI data do not provide conditional weights to take account the hierarchical data structure. Data was analyzed using Stata (version 14, Stata Corp.).

RESULTS

Table 3.1 shows unweighted descriptive statistics for all-cause mortality and covariates used in our gender stratified analyses. Men had a higher mortality rate (30.4%) during the study period than women (27.3%). Men had more years of education than women. Men were more likely to be married than women. Women were more likely to be widowed, divorced, or separated. Men had higher rates of full time employment and

were less likely to be unemployed or laid off than women.

Table 3.2 represents the results of gender-stratified multivariate parametric survival models with shared frailty for economic conditions during infancy (birth+1); results for economic conditions during the birth year and birth year-1 are presented in Table 3.3. Compared to not being expose to a recession, exposure to a recession during infancy was associated with a 54 % increase in all-cause mortality in later life only among women (hazard ratio=1.54, 95% CI 1.03-2.31). This result means that a 0.1 unit decrease in the GDP cycle deviation is associated with a 0.5% increase in all-cause mortality implying that women born during the worst recessions had about a 28.1% higher risk of dying than those born during the best boom periods. As a sensitivity analysis to test robustness of the result among women, we dichotomized the GDP deviation into economic boom or recession according to the positive or negative sign of the value. The results also consistently showed that recession at the first year of life was associated with an increased risk of women's dying (hazard ratio=1.07, 95% CI 1.00-1.16; results not shown in a table). However, neither exposure to a recession during the birth year nor the birth year-1 affected the risk of death for either gender. Across models, several significant demographic predictors of higher mortality were identified among men and women: older age, black race, lower family income, retirement and other employment status (keeping house, school, temporarily not working, and other). Women who were widowed had a significantly higher hazard of death.

We provide results for specific causes for women in Table 3.4. Significant effects were only found for mortality due to cancers (hazard ratio=2.24, 95% CI 1.18-

4.28). The other collapsed causes of death did not attain statistical significance at the 5% level. We further examined the interaction between GDP cycle and parental education as a proxy for family socioeconomic status in early life among women. The models controlled for the same covariates discussed earlier. Table 3.5 shows the interaction effects of economic conditions during infancy and paternal education as well as and maternal education on women's mortality. The results show a significant interaction effect between recession during the first year and paternal education. To facilitate interpretation of these interaction effects on all-cause mortality, we present figure 3.1. Regardless of paternal education, there is an inverse relationship between economic conditions during infancy and mortality, meaning the better the economic conditions in the first year of life, the lower the mortality. However, when we examined the relationship between economic conditions in the first year and mortality at three levels of paternal education (primary school or lower; high school graduation; and college or higher), we found an education gradient in the effect of mortality that reversed based on economic conditions. The results showed that higher paternal education showed a reduced risk of female adult mortality if women were born in an economic boom. However, higher paternal education was associated with an increase in female mortality risk if they were born in economic recession.

DISCUSSION

Empirical evidence has shown that economic conditions in early life are

associated with risk of death (Suhrcke and Stuckler 2012). These studies have been conducted with Western European samples. We extend the literature by examining these association among a national sample of adults in the United States. Our results showed that (1) recession in the first year of life is associated with an increased risk of all-cause mortality among women; (2) such effects were only found for cancer-related mortality; and (3) there was a significant interaction between economic conditions in the first year and paternal education on female adult mortality. Specifically, higher paternal education was protective against mortality when women were born in better economic conditions; whereas, higher paternal education was a risk for mortality when women were born in poor economic conditions.

Effects of economic conditions in early life on women's mortality risk

Our study demonstrated that recession during the first year of life was significantly associated with increased risk of all-cause adult mortality among women. However, exposure to a recession one year before birth and during the birth year were not associated with an increase in all-cause mortality in later life. For men, economic conditions one year before, one year after, or on the year of birth did not significantly predict adult mortality risk. In terms of the critical period model, our finding among women aligns with previous studies which indicate that recession at critical periods (one year before, one year after, or on birth year) was associated with an increase in adult mortality risk (Yeung et al. 2014, Van den Berg, Doblhammer-Reiter, and Christensen 2011, Lindeboom, Portrait, and Van Den Berg 2004, Doblhammer 2004, Almond and Currie 2011). It is well known that the first year of life as well as pregnancy is a critical

period for human development, health, and mortality in adulthood. The first year of life is an important period for infants, as they are initially exposed to nutrition (e.g., breastfeeding) and disease environments (e.g., infectious diseases) (Doblhammer 2004, Almond and Currie 2011). In contrast to findings from other studies, our results did not show that macro-economic conditions during pregnancy were not associated with an increase in adult mortality risk. We suspect that the inconsistency is attributed to different cohort ranges and data collected in different countries (U.S. vs. European countries). Previous studies that examined cohort-based effects of economic conditions on adult mortality used European birth cohort data from the 19th to the early 20th century (Yeung et al. 2014, Van den Berg, Doblhammer-Reiter, and Christensen 2011, Van Den Berg, Lindeboom, and Portrait 2006); whereas, our birth cohorts range from 1889 to 1984. In the 19th century, risks such as starvation induced by job/income loss, lack of social welfare systems and safety nets, and prevalent infectious and parasitic diseases were often associated with a recession, which can eventually lead to mortality. However, for the 20th century cohorts, the mortality risks associated with recession might be substantially reduced due to higher levels of average population wealth, epidemiologic transition from infectious diseases to chronic diseases, advanced knowledge of medicine and medical technologies for family planning, and better welfare policies (Cutler and Meara 2004, Stuckler et al. 2009). Given the body of studies that have demonstrated the importance of maternal nutritional intake, especially during later pregnancy months (Yeung et al. 2014), it is plausible that overall improvement in maternal nutritional intake in the 20th century has mitigated the negative effects of

recession during pregnancy. The nutritional support of better food assistance policies to support pregnant women may cancel the negative effects of recession during pregnancy. This difference also may be explained by birth selection dependent on economic conditions based on modern medical technologies for family planning. Previous studies demonstrated that individuals who had uncertainty in employment prospects, lower income, and anxiety about the future during recession were more likely to postpone or retract childbearing plans (Sobotka, Skirbekk, and Philipov 2011, Adsera 2011). Notably, individuals with higher education were more likely to adopt risk-adverse behaviors (Sobotka, Skirbekk, and Philipov 2011). This pro-cyclical fertility, especially among highly educated parents, may fundamentally change cohort compositions in terms of parental socioeconomic status, in turn leading to an increase in mortality in later life among those born during a recession.

In terms of gender differences, previous studies have shown that significant associations between early-life conditions and adult health were differentiated by gender. For example, women's health is more likely to be influenced by childhood conditions than men's health due to biological differences (Luo and Waite 2005, Hamil-Luker and Angela 2007, Bruckner and Catalano 2007, Byrne et al. 1987). However, it is yet unclear why women's health would be more strongly affected by early-life environments than men's. Biological differences between men and women may be explained by the frailty of male fetuses to exogenous insults in utero. A few studies suggests that natural selection allows the bodies of pregnant women to detect and terminate the gestation of weak males when environmental stressors increased in frequency or virulence

(Bruckner and Catalano 2007, Kaitz et al. 2015). Thus, survived male infants may be stronger to the early-life exposures to stressors than female infants. Parental infant care including nutrition may be another mechanism underlying gender differences in the association. Further work is needed to explore the extent to which gender disparities in early life nutrition and infant care contribute to inequalities in mortality. If male infants are more likely than female counterparts to receive better care and nutrition even within disadvantaged families, female infants may experience more deleterious health effects of growing up in impoverished circumstances.

Effects of economic conditions in early life on women's disease-specific mortality risk

Our analyses of cause-specific mortality outcomes showed that recession during the first year of life was associated with increased risk of women's dying from cancers. It is widely recognized in the etiology of certain cancers (Gluckman et al. 2008). There are studies shows that adverse early life exposures, for example secondhand smoking and lack of breastfeed may lead to a higher risk of cancers (Potischman and Troisi 1999, Sandler et al. 1985). Previous studies that examined the relationship between recession in early life and cause-specific mortality have reported mixed results. Several studies were unable to detect a long-run effects of economic conditions in early life on cancer mortality, yet a recent study identified the effects of economic recession in early life on cancer mortality in adulthood, specifically colon cancers for men and smoking-related cancers among women (Yeung et al. 2014). Another study reported that the long-run effects were only significant with mortality due to infectious diseases and

coronary heart diseases (Van den Berg, Doblhammer-Reiter, and Christensen 2011). The discrepancy may result from differences in the cohort range as well as the nationality of respondents. Susceptibility to disease is embedded in individuals' biological makeup, but diseases are expressed through interactions between individuals and particular social contexts (Halfon and Hochstein 2002, Power and Hertzman 1997).

Interaction effects of economic conditions in early life and paternal education

To obtain further insights into the underlying mechanisms of how economic condition in early life relate to mortality, we further explored the interaction effects between economic conditions in the first year of life and paternal education as a proxy of family environments in early life. Parental socioeconomic status is a fundamental determinant in adult health operating through multiple mechanisms over considerable periods of time (Pudrovska 2014, Paeratakul et al. 2002). It is well-established that lower parental education at early-life presents a significant risk to mortality (Hayward and Gorman 2004, Pensola and Valkonen 2002). Given that paternal education interacted with economic conditions in the first year of life, it is likely that inferior housing conditions or parental infant-caring behaviors were related to mortality. For instance, maltreatment in early life stages is more prevalent among socioeconomically-disadvantaged parents who are frequently exposed to stress, lack of parenting time, overcrowding in housing, poor nutrition, and poor household sanitation (Morton, Schafer, and Ferraro 2012). The results showed that higher paternal education exacerbates the mortality risks during a recession but attenuated the mortality risk during a boom. Note that our model controlled for the effects of individual

socioeconomic risk factors (education, income, and job status) in adulthood. One possible explanation, drawing on aspirational theory, suggests that during recessions, highly educated fathers may experience a greater discrepancy between aspirations and reality than to fathers with lower education levels, ultimately differentially affecting paternal stress levels and the health of their children. A level of aspiration is a subjectively established goal and "a reference point of feelings of success or failure" based on comparisons of one's position to that of other individuals (Kim 1970). According to the aspiration theory, an individual's well-being is determined by the gap between aspiration and achievement (Michalos 2012, Inglehart 1990). Thus, the deleterious effect of higher paternal education during the recession may be explained by the incongruence between aspirations and reality (e.g., less prestigious occupation or less income) during recession among more highly educated fathers. This explanation is also supported by the negative experiences of employees who retained a job during recessions. Such experiences may be caused by employer pursuit of higher efficiency: for less cost (reducing working hours, salaries, and benefits) and for more productivity (shrinking decision latitude but demanding higher workloads due to staff lay-off). Given that employees with higher education were more likely to hold their jobs during recessions, they may be more distressed or dissatisfied due to their company's pursuit of higher efficiency to survive during recessions (Wilson, Tienda, and Wu 1995). Past empirical studies demonstrated that job restructuring during recessions caused greater stress among job holders than unemployed individuals or those who has an unstable job (Fenwick and Tausig 1994, Tausig and Fenwick 1999). This psychological stress for

highly educated fathers may exacerbate the negative effects of recession in the first year of life on offspring mortality. This may result from elevated householder stress, lower level of household living comfort, more conflicts among householders, and reduced time and quality for infant care (Slopen, Koenen, and Kubzansky 2012, Garssen 2004, Everson-Rose and Lewis 2005). During better economic conditions, employees of higher educational attainment may be more likely to enjoy opportunities of promotion and be less distressed by reduced workload with support from supplemental workers hired or better work environments during the boom times (Keane, Prasad, and Minneapolis 1993, Kydland 1984, Gertler and Trigari 2006).

Although our findings significantly contribute to further understanding adult mortality, the present study has some limitations that require caution in interpreting our results. First, the indicator of economic conditions might not accurately represent the population's actual economic status. The GDP, which was used to calculate the economic condition in early life, has been criticized for not accurately reflecting individual economic status if the distribution of economic benefits is concentrated among a small percentage of the population (Sen 1976, 1979, Van den Bergh 2007). Second, a causal relationship between economic conditions in the first year and adult mortality cannot be inferred. As we mentioned above, based on modern medical technologies for family planning may hamper our interpretation of a causal relationship. Third, there could be a selection bias since disadvantaged individuals may have already died before data was collected in the GSS. Thus, our results will be tempered by the conclusion that they apply only to current surviving members of each cohort. Fourth,

although the data contains information on birth month, we cannot align exact pregnancy periods with economic conditions as we used the shared frailty at the birth cohort level, and GDP information was available only on an annual basis. Future studies stratified by race/ethnicity is needed to examine heterogeneity in the relationships between economic conditions in early life and adult mortality. It is certain that the benefits or adversities from economic conditions in early life vary by race/ethnicity. Moreover, increased racial discrimination in labor markets during economic booms may disproportionately distribute boom benefits across racial groups (Bradbury 2000).

Our study points to the importance and long-term effects of economic conditions in early life on women's cancer mortality. We also have shown that importance of parental socioeconomic status that interacted with economic conditions in early life. By using exogenous indicators of economic conditions, we are at a methodological advantage over studies using family income or social status as a predict variable for adult mortality. Policies during economic recessions to improve infants' conditions, for example by way of enhanced food, job, housing, and health care provisions may yield positive impacts on the mortality risk in adulthood.

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Table 3.1. Characteristics of respondents (N=30,331 adults) from the General Social Survey linked to the National Death Index

	Men (n=13,100)		Women (n=17,231)	
	N	%	N	%
Vital status				
Dead	3,986	30.4	4,706	27.3
Alive	9,114	69.6	12,525	72.7
Race				
White	11,203	85.6	14,054	81.6
Black	1,587	12.1	2,784	16.2
Other	310	2.3	393	2.3
Marital status				
Married	7,509	57.3	8,281	48.1
Never married	3,168	24.2	3,117	18.1
Widowed	558	4.3	2,649	15.4
Divorced or separated	1,865	14.2	3,184	18.4
Job status				
Full time job	8,420	64.3	6,942	40.3
Part-time	1,001	7.6	2,267	13.2
Retired	2,083	15.9	1,978	11.5
Unemployed or laid off	856	6.5	626	3.6
Other	740	5.7	5,418	31.4
Region				
West	2,421	18.5	3,034	17.6
Northeast	2,427	18.5	3,210	18.6
South	4,658	35.6	6,315	36.7
Midwest	3,594	27.4	4,672	27.1
	Mean	SD	Mean	SD
Age	44.4	16.9	46.3	18.1
Education	13.0	3.2	12.6	2.9
Years of paternal education	10.7	3.6	10.5	3.5
Years of maternal education	11.0	3.2	10.7	3.3
Family income (1K dollars)	20.1	6.7	18.1	7.6

Gender differences for the all variables were significant at $p<0.001$

Table 3.2. Gender-stratified regression estimates for all-cause mortality from multivariable parametric survival model with shared frailty using the General Social Survey linked to the National Death Index

	Men (n=13,100)			Women (n=17,231)		
	Hazard Ratio	95% CI	p	Hazard Ratio	95% CI	p
Recession in the first year	1.32	0.87-2.03	0.191	1.54	0.75-1.77	0.036
Age	1.05	1.03-1.06	<0.001	1.06	1.05-1.08	<0.001
Age squared	0.99	0.99-1.00	0.564	0.99	0.99-0.99	0.036
Race (reference: white)						
Black	1.38	1.25-1.51	<0.001	1.32	1.22-1.43	<0.001
Other	1.23	0.98-1.56	0.080	1.07	0.84-1.36	0.603
Education	1.00	0.99-1.01	0.728	0.99	0.98-1.00	0.110
Paternal education	1.00	0.99-1.01	0.709	1.00	0.99-1.01	0.763
Maternal education	0.99	0.98-1.01	0.300	1.00	0.99-1.01	0.878
Family income	0.99	0.98-0.99	<0.001	0.99	0.98-0.99	0.001
Marital status (reference: married)						
Never married	0.98	0.88-1.09	0.679	1.10	0.98-1.24	0.093
Widowed	0.90	1.04-1.23	0.088	1.14	1.05-1.24	0.003
Divorced or separated	1.01	0.92-1.11	0.868	0.96	0.87-1.06	0.397
Job status (reference: full time job)						
Retired	1.27	1.13-1.41	<0.001	1.34	1.20-1.50	<0.001
Unemployed	1.14	0.99-1.31	0.070	1.08	0.89-1.31	0.444
Part-time	1.13	0.99-1.30	0.068	1.11	0.99-1.24	0.075
Other	1.35	1.16-1.58	<0.001	1.27	1.16-1.38	<0.001
Regions (reference: West)						
Northeast	0.94	0.85-1.05	0.296	0.92	0.83-1.01	0.090
South	1.03	0.93-1.13	0.598	0.97	0.88-1.06	0.474
Midwest	0.99	0.89-1.10	0.786	1.01	0.92-1.11	0.846

Table 3.3. Estimation results of multivariable parametric survival model with shared frailty for three periods of economic conditions in early life using the General Social Survey linked to the National Death Index

	Birth year-1			Birth year			Birth year+1		
	Hazard Ratio	95% CI	p	Hazard Ratio	95% CI	p	Hazard Ratio	95% CI	p
Men	1.12	0.73-1.72	0.601	1.15	0.74-1.77	0.520	1.33	0.87-2.04	0.191
Women	1.26	0.83-1.93	0.278	1.32	0.87-2.02	0.189	1.54	1.02-2.31	0.036

Note: Estimates were based on the main model (controlled for respondent's age, age^2, race, education, marital status, job status, parental education, family income, and region)

Table 3.4. Causes of death with hazard ratios and 95% confidence intervals (CIs) of multivariable parametric survival models with shared frailty for recession in the first year among women using the General Social Survey linked to the National Death Index

Cause of death (deceased/total observed)	Hazard Ratio	95% CI	p
Infectious and parasitic diseases (180/ 9,281)	1.03	0.18- 5.85	0.972
Cancers (1,205/13,703)	2.24	1.18-4.28	0.014
Endocrine; nutritional; and metabolic diseases and immunity disorders; diseases of the blood and blood-forming organs (247/ 12,747)	2.41	0.64-9.07	0.194
Mental illness (146/ 12,644)	0.88	0.03-26.64	0.941
Diseases of the nervous system and sense organs (72/12,570)	1.88	0.15-22.90	0.621
Diseases of the circulatory system (1,682/14,180)	0.79	0.34-1.83	0.580
Diseases of the respiratory system (267/12,767)	0.98	0.67-1.44	0.928
Diseases of the digestive system (137/12,635)	1.20	0.72-2.02	0.469
Diseases of the genitourinary system (18/ 12,516)	36.6	0.05-2897.6	0.279
Suicide or self-inflicted injuries (24/12,522)	0.05	0.01-4.67	0.205
Others including injury-related deaths (494/12,992)	1.35	0.28-6.63	0.710

Note: Estimates were based on the main model (controlled for respondent's age, age^2, race, education, marital status, job status, parental education, family income, and region)

Table 3.5. Hazard ratios and 95% confidence intervals (CIs) of all-cause mortality predicted by recession, recession and paternal education interaction, and recession and maternal education interaction among women using the General Social Survey linked to the National Death Index

		Hazard Ratios	95% CI	p
Model 1[1)]	Recession in the first year	1.54*	0.75-1.77	0.520
Model 2[2)]	Recession in the first year	1.92	0.48-2.80	0.742
	Recession × paternal education	1.09*	1.01-1.18	0.034
	Recession × maternal education	0.95	0.87-1.03	0.179

Note: 1) Main effect from the main model (controlled for respondent's age, age^2, race, education, marital status, job status, parental education, family income, and region); 2) Adding interaction terms between economic conditions in the first year and parental education in Model 1

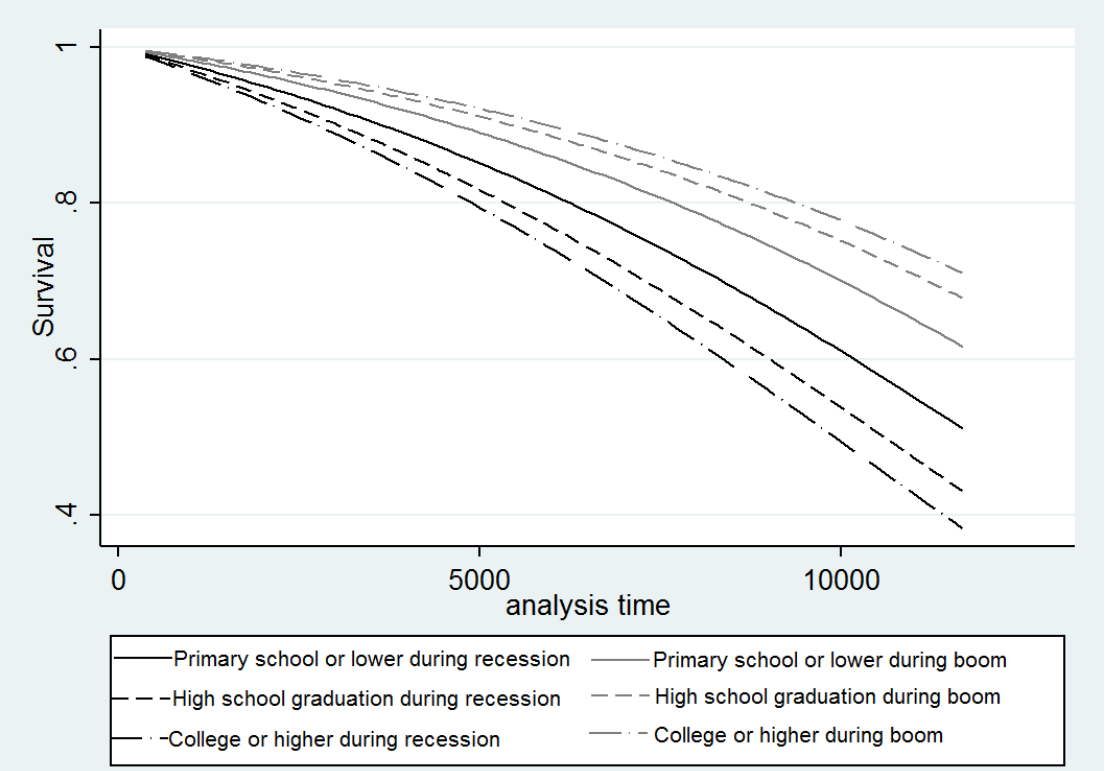

Figure 3.1. Interaction effects of paternal education and the economic conditions in early life on all-cause mortality risk among women (n=17,231) from the General Social Survey linked to the National Death Index

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Chapter 4: Income Inequalities in Early Life and the Risk of Adult Mortality in the United States

Abstract

Background: Despite a growing literature suggesting links between income inequality and health, no previous studies have been conducted to examine the effects of income inequality in early life on the risk of all-cause and cause-specific mortality.

Methods: To examine these effects, we fit a multivariable parametric survival model with shared frailty using the General Social Survey-National Death Index (GSS-NDI) linked data. To capture exogenous variations in income inequality early in life, we used the top wage income share series per birth year.

Results: Based on a sample of 13,100 males and 17,231 females, we found that a higher level of income inequalities in early life was associated with increased risk of all-cause mortality among males (hazard ratio=1.038-1.041, 95% CI 1.001-1.070). This increased risk of mortality among men was due to cancers; endocrine, nutritional, and metabolic diseases; immunity disorders; circulatory and respiratory system diseases; and suicide. We also found that there was a significant interaction effect between income inequality in the birth year and paternal education in risk of mortality among males.

Specifically, higher paternal education was protective against the risk of male mortality when the respondents were born during period of lower income inequality. On the other hand, higher paternal education was a risk factor for the risk of male mortality when respondents were born during period of higher income inequality.

Conclusions: Our study based on a life-course approach, concludes that income inequalities in early life have a long-term impact on risk of male mortality, and that paternal education moderates this relationship. Further research is required to elucidate the biological mechanisms of income inequality.

INTRODUCTION

Coinciding with increasing concerns over the extent of income inequality between the rich and poor within countries, there is a growing literature that large variation in the income inequality is an important risk of population health. The research is based on the relative income hypothesis (RIH), which assumes that unequal income distribution within societies in developed countries influences average levels of health (Lynch et al. 2004). After Rodgers' seminal 1979 study that showed an association between the infant mortality and income distribution (Rodgers 1979), the RIH has been tested at various geographic levels. Most of these analyses performed using U.S. data showed mixed results due to heterogeneity in methodological approaches and geographical levels, and the use of disparate health and income inequality (Fuchs 2004). Although most extant studies rely on geographic variation within a given country, our study uniquely capitalizes on temporal variation in income inequality within the United States.

Three main hypotheses used to interpret the relationship between income inequality and health have focused on the material, social, and psychosocial dimensions. First, a high level of income inequality may reflect a set of economic, political, social, and institutional processes that result in a systematic underinvestment in human, physical, health, and social infrastructure that supports a healthy lifestyle for those with the least individual resources (Lynch et al. 2004, Subramanian and Kawachi 2004). For example, disparities in educational support, social welfare, affordable housing, good roads, and environmental protection may directly and indirectly result in health

inequalities (Lynch et al. 2000). Second, income inequality may affect health via the disruption of social fabric, termed social capital. Social capital has been defined as those features that reflect norms of reciprocity, civic engagement, and mutual trust among community members (Putnam, Leonardi, and Nanetti 1994, Kawachi et al. 1997). It has been hypothesized that the widening of income inequality has led to latent social conflict and the erosion of social capital. This, in turn, affects health via inegalitarian patterns of political participation and the passage of social policies that are detrimental to the poor (Kawachi et al. 1997, Kawachi et al. 1994). Third, inequitable income distribution may directly affect people's psychosocial perceptions of a disparity in social position based on individual resources. This negative psychosocial status effect may in turn influence health partly through the direct physiological effects of chronic stress (Brunner 2007), and partly through its influence on health related behavior. De Vogli (2004) has argued that low social status is stressful because it reduces people's control over their lives and work [4, 9, 10, 11]. Others have argued that low social status is stressful because people feel devalued and inferior (Charlesworth, Gilfillan, and Wilkinson 2004, Wilkinson 2005). Both suggestions are borne out by a recent review of the most salient stressors affecting cortisol responses (Dickerson and Kemeny 2004). People with lower social status were more likely to adopt unhealthy behaviors including tobacco smoking, alcohol consumption, substance use, poor diet, and low exercise levels (Berkman, Kawachi, and Glymour 2014, Adler and Ostrove 1999).

Income inequality studies on mortality in U.S. contexts showed mixed results. However, studies on lagged effects of income inequality consistently showed that the

strongest associations in mortality were at later ages (Subramanian and Kawachi 2004, Blakely et al. 2000, Mellor and Milyo 2001, Huynh et al. 2005, Zheng 2012). These studies suggest that exposure to income inequality may affect health years or decades later.

To our knowledge, no study has been conducted to examine the effects of early-life income inequality on adult mortality, even though it is well known that the early years of life are critical for human development, health, and mortality in adulthood. This is an important period for infants, as they are initially exposed to nutrition and disease environments (Doblhammer 2004, Almond and Currie 2011). Moreover, conceptualizations of cause-specific etiology have received little attention in the previous studies that discussed associations between income inequality and health.

Income inequality in early life may affect adult mortality via poor birth outcomes and detrimental conditions in early life. Psychosocial stress due to early life income inequality may increase poor birth outcomes such as preterm birth or low birth weight, which can result in negative health consequences in adulthood (Huynh et al. 2005, Macinko et al. 2003, Wadhwa et al. 2001). Detrimental conditions in early life have long-lasting effects that are independent of conditions during later life. The mechanism is based on the neo-material and social capital interpretation of income inequality that suggests a direct influence on infants in the post-neonatal period (Lynch et al. 2000).

In our study, based on the critical period model in life-course epidemiology and previous study findings, we aimed to examine that income inequality in early life may

exert their detrimental influence on adult mortality. The critical period model recognizes the importance of the early life stage for understanding the causal relationships between exposures to specific contexts in early life and health at both individual and population levels (Lynch and Smith 2005, Smith and Egger 1996, Kermack, McKendrick, and McKinlay 1934).

We used mortality data linked birth year with the top wage income concentration as an exogenous measure of income inequality in early life to increase the generalizability of study findings. We fit parametric survival models with shared frailty for survival data to analyze the birth cohort-specific effects on all-cause mortality while controlling for individual socio-demographic variables. Additional analyses of disease-specific mortality were also conducted. To explore plausible mechanisms, we further tested interaction effects between income inequalities in early life and parental education as a proxy of early-life socioeconomic status. It is well established that lower parental education at early-life presents a significant mortality risk (Hayward and Gorman 2004, Pensola and Valkonen 2002); however, it is unclear how parental education and early-life economic conditions interact and how this effect extends to offspring mortality in US contexts.

Given that there are gender differences not only in U.S. infant mortality, work, and family role (Vance et al. 1995, Hill and Upchurch 1995, Cinamon and Rich 2002), we stratified our analyses by gender. It is plausible that gender differences may be presented in the associations between adult mortality and early-life income inequalities.

METHODS

Study population

The General Social Survey-National Death Index (GSS-NDI) was used as an individual level dataset. The GSS-NDI linked data prospectively from 18 waves of the GSS (1978 through 2002) to death certificate data from 1979 through 2008. The original GSS utilized repeated cross-sectional samples during 1972-2010 with annual surveys between 1972 and 1993 except 1979, 1981, and 1992; and biannual surveys during 1994-2010. The NDI utilizes a probabilistic matching algorithm to maximize the linkage between individual identifiers and death certificate information. This yielded a sample of 9,242 deceased individuals from the total sample of 32,781 respondents. For each observation, we have a set of survey questions for a given year as well as an indicator of whether subjects were alive as of 2008, and the number of days until death or censoring in 2008.The birth cohorts spanned from 1889 to 1991. The process of data linkage is described in more detail elsewhere (Muennig et al. 2011).

Measurement

Outcomes: all-cause and cause-specific mortality

The dependent variables for our analysis were all-cause and cause-specific mortality. Deaths were identified from the NDI and linked with our master dataset. Of the 9,242 GSS records determined to have a vital status of deceased, 99.8% were linked to underlying cause of death information. Until the year 1999, the NDI provided users

with cause of death using codes from the Ninth Revision of the International Classification of Diseases (ICD-9), and Tenth Revision (ICD-10) codes thereafter. To have consistency across the survey years, we chose 285 mutually-exclusive categories from the Clinical Classification Software (CCS) system. The CCS code is a diagnosis and procedure categorization scheme developed by collapsing the ICD-9-CM into clinically definable and mutually exclusive categories (Cost 2007). Based on the CCS Diagnosis Categories, while collapsing several disease groups to prevent results from being driven by only a few observations, we categorized the total codes to 11 categories: infectious and parasitic diseases (CCS 1-8); neoplasms (CCS 9-47); endocrine, nutritional, and metabolic diseases, immunity disorders, and diseases of the blood and blood-forming organs (CCS 48-64); mental illness (CCS 65-73); diseases of the nervous system and sense organs (CCS 74-95); diseases of the circulatory system (CCS 96-121); diseases of the respiratory system (CCS 122-134); diseases of the digestive system (CCS 135-155); diseases of the genitourinary system (CCS 156-161); suicide or self-inflicted injuries (CCS 662); and others including injury-related deaths.

Main determinant: income inequality in early life

There are several indices that represent income inequality at the national level, including the Gini coefficient, Atkinson index, and the Robin Hood index (De Maio 2007). Among them, we used the top wage income share series from the Piketty-Saez data (Piketty and Saez 2001). The Gini coefficient is by far the most popular measure of income inequality, although that index is available only beginning in 1965. In contrast, the top wage income share series is available from 1913. Given that our sample of birth

cohorts begins in1890, we chose the latter series as our main determinant to prevent loss of data. The series is based on wage income reported on individual tax returns. Tax filers are ranked by size of wage income and top groups are defined relative to the total number of U.S. families earning positive wage income. Income is defined as annual gross income reported on individual tax returns including salaries and wages, small business and farm income, partnership and fiduciary income, dividends, interest, rents, royalties, and other small items reported as other income. Income is defined as amount prior to deduction of individual income taxes and employees' payroll taxes and excludes all government transfers (such as Social Security, Unemployment Benefits, Welfare Payments, etc.) (Piketty and Saez 2001). Annual series of shares of total income was constructed by accruing various higher fractiles within the top decile: the top 5 percent (P95–100), the top 1 percent (P99–100), the top 0.5 percent (P99.5–100), the top 0.1 percent (P99.9–100), and the top 0.01 percent (P99.99 –100). All series and complete technical details about our methodology are available elsewhere (Piketty and Saez 2001). To choose the best indicator, we compared model fits across top percentage of income concentration using the Akaike Information Criterion (see Supplement table 4.1). Based on our preliminary analysis using our full model, we chose income concentration within 0.5% at birth as our main independent variable. For example, if income concentration within 0.5% in 2014 is 13.7%, we can interpret that the richest 0.5% disproportionately shares 13.7% of the national income. The GSS contains information about both birth year and month, and we assigned our income inequality measures based on birth year in addition to specifying shared frailty for similar birth years. Given potential overlap

between time periods, we fit models sequentially to examine the effects of income inequality during the years prior to birth (birth year-2 and -1); the actual birth year (birth year); and the years after birth (birth year+1 and +2).

Covariates

Covariates include gender, age, race, marital status, employment status, education status, and family income. Further, educational status of respondents' parents (highest year completed) was reported by the respondents. Age and family income were treated as continuous variables, and other independent variables were entered as dummies in our model. Age was a continuous variable, and total family income was coded per 1,000 dollars of the average value of each income range categorized in the GSS (e.g. if a respondent chose an $8,000-$9,999 category for the family income, we coded it as 9). We categorized race into white (reference), black, and others. Marital status was measured with four categories: married (reference), never married, widowed, and divorced or separated. Employment status of respondents was categorized by working full time (reference), working part time, retired, unemployed, and other. The unemployed category includes those who were unemployed, laid off, looking for work, and have a job but not at work because of temporary illness, vacation, and strike. The other category includes being in school, keeping house, and other responses. Parental as well as respondents' education levels were measured by highest years completed. Region was categorized into West (reference), Northeast, South, and Midwest. Missing information on age (0.1%), respondent's education (0.2%), paternal education (28.0%), maternal education (15.4%), and family income (9.4%) was imputed by replacing the

mean age (45.3 years), the mean education (12.8 years), the mean years of education completed by mothers (10.8 years) and fathers (10.6 years) and mean family income (18,988 dollars), respectively. Missing data in variables for marital or employment status (0.02%) was replaced with the modal category. All independent variables except for the income inequality indicator were obtained from the GSS. After additional exclusion of individuals born outside of the U.S., and for which income inequality data would be neither comparable nor available (n=2,494), the study sample was 30,331 adults. Table 4.1 shows unweighted descriptive statistics of respondents' demographic information by gender.

Statistical analyses

We specified a multivariate parametric survival model with shared frailty. We fit a Gompertz distribution to reflect survival times to individual mortality, and assumed a gamma distribution for shared frailty. The Gompertz distribution (proportional hazard metric) has been widely used in mortality studies (Bender, Augustin, and Blettner 2005, Cleves et al. 2008) for hazard rates that increase exponentially with time. The shared frailty model is essentially a random effects model for survival data in that it allows individuals to share the same latent frailty value among groups. The hazard rate for the i^{th} individual from the j^{th} group is,

$$h_{ij}(t \mid \beta_j, v_j) = h_0(t) w_{ij} \exp(\beta_j x_{ij}), \text{ where } w_{ij} = \exp(v_j z_{ij}) \quad (1)$$

$$h_{ij}(t \mid \beta_j, v_j) = \mathrm{h_0(t)exp}(\beta_j \mathrm{x_{ij}} + v_j z_{ij}) \quad (2)$$

$h_{ij}(t \mid .)$ is the conditional hazard function for the ith individual from the j^{th}

group at time *t*, $h_0(t)$ is the baseline hazard function at time *t*, β_j is a vector of cohort-specific fixed-effects corresponding to a vector of covariates x_{ij}, v_i is a vector of random effects associated with a vector of covariates z_{ij} and is also known as the subgroup frailty shared among the ith individual from the jth group. v_j is assumed to be randomly distributed with mean 0 and variance-covariance matrix θ, which depends on parameter θ. We used the shared frailty at birth cohort level (i.e. birth year). The frailty follows a gamma distribution (Cleves et al. 2008, Gutierrez 2002); the variance of the frailty (θ, theta) was estimated by iterative maximum profile log-likelihood. The likelihood-ratio was tested to examine the significance of the shared frailty parameter estimate.

We assumed that the income concentration within 0.5% of a year represents the income inequalities that evenly affect those born in the year regardless of birth month. We cannot define exact pregnancy periods in our models even though the data has information about birth month, as we used the shared frailty at birth cohort level, and income concentration information was given on an annual basis. Being aware of overlap between the periods, we fit the models sequentially to examine the effects of income inequality during the birth year-1; during the birth year; and during the birth year+1. To obtain further insights into the underlying mechanisms, we additionally analyzed the interaction effects between income inequality in early life and paternal education as a proxy variable to represent early family environments. All our shared frailty models were unweighted as the GSS-NDI data do not provide conditional weights. Data were analyzed using Stata software packages (version 14, Stata Corp.).

Sensitivity analysis

Piketty and Saez provide three different data series, each of which treats capital gains slightly differently and therefore yields somewhat different estimates of the share of income going to each group. In order to assess the sensitivity of our results, we compared estimates of the original model to a model that included capital gains in income share computations. Realized capital gains are not an annual flow of income based on the income definition. Instead, they are a component of income with large aggregate variations from year to year depending on stock price variations.

RESULTS

Table 4.2 represents the results of multivariate parametric survival models with shared frailty for income concentration within the top 0.5% in the birth year. Our sensitivity analysis confirmed the robustness of the model (Supplement table 4.2). For males, exposure to an income inequality in the birth year was associated with increased risk of all-cause mortality in later life (hazard ratio=1.041, 95% CI 1.013-1.070). This result means that a 1 unit increase in income inequality was associated with a 4.1% increase in risk of all-cause mortality, implying that males born during the worst periods of inequality (1920s-1940s) had about a 41.4% higher risk of dying than those born during the most equal periods (1940s-1970s). Again, our survival models are age-adjusted and controlled for shared frailty by birth cohort, so these cannot be attributed solely to age effects. We also explored the effects of income inequality during birth year-

2, birth year-1, birth year, birth year+1, and birth year+2. Table 4.3 shows that there were significant associations between income concentration within the top 0.5% during the four years and risk of adult mortality among males (hazard ratio=1.038-1.070, 95% CI 1.001-1.070). There were no significant associations between income concentration and female mortality.

We analyzed which causes of death among males were associated with the income inequality at birth (Table 4.4). We found that income concentration at birth was associated with increased risk of mortality due to cancers (hazard ratio=1.15, 95% CI 1.08-1.23); endocrine, nutritional, and metabolic diseases, and immunity disorders (hazard ratio=1.16, 95% CI 1.04-1.30); diseases of the circulatory system (hazard ratio=1.11, 95% CI 1.05-1.18); diseases of the respiratory system (hazard ratio=1.17, 95% CI 1.04-1.32); and suicide (hazard ratio=1.47, 95% CI 1.15-1.87).

Then we tested the interaction between our measure of income inequality and parental education among males as a proxy of socioeconomic family environments in early life. Table 4.5 shows the results of the main effect of income concentration in the birth year and the interaction effects of income concentration and paternal education on risk of mortality.

The result shows that the effects of higher income concentration in the birth year varied with the degree of paternal education among males. Figure 4.1 graphically depicts the inequality at birth on male of male mortality at three levels of paternal education (primary school or lower; high school graduation; and college or higher).

Regardless of paternal education, there is a positive relationship between income concentration and risk of mortality, meaning the higher the income concentration in the birth year, the higher risk of the mortality among males. We also examined the relationship between income inequality (dichotomized income concentration in birth year by categorizing below or above mean) and male mortality risk at three levels of paternal education (primary school or lower; high school graduation; and college or higher). From this, we found that higher paternal education worsened the negative effects of income inequality if there was more income inequality, but buffered the effects if there was less income inequality.

DISCUSSION

To our knowledge, this is the first study of its kind to explore income inequality at birth and adult mortality, including information on parental education. We used a multivariate parametric survival model with shared frailty to find whether the effects of income inequality in early life were associated with risk of adult mortality. We found that (1) a higher level of income inequality in early life was associated with increased risk of all-cause mortality among males only; (2) such associations were found for male mortality due to cancers; endocrine, nutritional, and metabolic diseases, and immunity disorders; and diseases of the circulatory and respiratory systems; and (3) there was a significant interaction effect between income inequality in the first year and paternal education on later-life mortality among males. Specifically, higher paternal education

was protective against male mortality when the respondents were born in periods with less income inequality; whereas, higher paternal education was a risk factor for male mortality when respondents were born periods with more income inequality.

Effects of income inequality in early life on population level mortality among males

Our study demonstrates that greater income inequalities at the time of birth were significantly associated with increased all-cause adult mortality, yet this relationship holds only for males. This finding aligns with previous studies on income inequality that have shown its significant negative effects on infant mortality (Waldmann 1992) and low birth-weight (Huynh et al. 2005), though there is little gender-stratified research. Greater income inequality may reflect a systematic underinvestment in immunization or tuberculosis control programs; limited access to maternal and infant health services; lower standards for breastfeeding, smoking, or environmental pollution; and greater tolerance of racial and gender discrimination (Macinko et al. 2003). Income inequality may disrupt social capital and weaken family stability, resulting in ambiguity or cynicism regarding conventional parental roles. Previous studies note that a high degree of inequality promotes loss of social capital by widespread delegitimization of conventional norms at the family and community level (Messner 1988, Shihadeh and Steffensmeier 1994). Thus, parents in visible inequities undermine the willingness to protect and promote infant health before, during, and after birth. Income inequality may also create a psychosocial climate of bitterness, resentment, and a sense of exclusion for pregnant women and their families that directly influences infant health (Shihadeh and Steffensmeier 1994). Previous studies showed that maternal stress can result in infection

and risky health behaviors such as poor maternal nutrition and smoking, which are associated with poorer birth outcomes (Kramer et al. 2001, Pickett and Wilkinson 2015).

There has been little gender-stratified research on the association between income inequality in early life and mortality. The reason for gender differences is unknown. However, we can partially attribute gender differences in our study to historical changes in male infant mortality. Though overall mortality declined from the 18th century, male infant mortality has progressively risen in developed countries (Trovato and Heyen 2006). However, the trend in excess male infant mortality in most countries has dropped to lower levels since 1970 (Drevenstedt et al. 2008), which coincides with the increase in income inequality. The additional male infants that survived were more likely to be premature or have low birth weight, which could increase long-term health risks and adult mortality in men.

Effects of income inequalities in early life on male mortality due to specific cause of death

Our replicated analyses with cause-specific mortality showed that income inequality at birth significantly increased the risk of dying from multiple causes. There are little previous research on the association between income inequality in early life and cause-specific mortality. Given this relationship between income inequality and health, it is likely that limited access to healthcare resources, inferior housing conditions, chronic stress, parental infant-caring behaviors, adverse health behaviors including smoking, alcoholic, and poor diet were related to the specific causes of death (Lynch et

al. 2004, Huynh et al. 2005). However, it is out of our research scope to identify how these risk factors trigger offspring's specific causes of death by embodiment during pregnancy due to diverse pathways and complicated risk factor interactions. Further biological study into the cause-specific etiology may be needed.

Interaction effects of income inequalities in early life and paternal education

We further explored the interaction effects between income inequality and parental education. To our knowledge this is the first time that an interaction between paternal education, income inequality, and offspring mortality has been examined. The result shows that higher paternal education worsened the negative mortality effects of income inequality during periods of high inequality, but attenuated the effects in times of less inequality. It is well established that lower parental education at early-life presents a significant risk to offspring's mortality regardless of sex (Hayward and Gorman 2004, Pensola and Valkonen 2002). This may suggest that paternal aspiration-levels may vary according to education and affect paternal psychological status. This in turn may affect parental caring behaviors or household environments, and further offspring mortality in adulthood. According to the aspiration theory, individual well-being is determined by the gap between aspiration and achievement (Michalos 2012, Inglehart 1990). Wilkinson argues that, in developed nations, levels of depression, isolation, insecurity, and anxiety are associated with relative position (Wilkinson 1992, Wilkinson and Pickett 2006). During times of greater income inequality, highly educated fathers may be more frustrated as their social positions are viewed in comparison to the wider income stratum—they may experience more stress if they

realize their subjectively established goals are relatively low compared with others. Alternatively, highly educated fathers may be more directly affected by greater income inequality as they are more concerned about societal equity than those of lower education (Kawachi et al. 1997, Kawachi et al. 1994). The effects of these perceptions and expectations may then be passed on thru either: 1) birth outcomes which have long-term health effects among offspring, or 2) there is something about parental expectations during birth that have long-term implications for childhood (and ultimately adult) health that are independent of later experiences of income inequality. Certainly, conditions during birth are exceptionally important for the life-long health of individuals, but it may be the case that conditions during birth pre-dispose parents to mechanisms which have long-lasting health implications for their children. Further research is needed to both replicate and understand these findings.

Our results have some limitations that require interpretative caution. First, our measure of income inequality may have a unique effect. Since the top wage income share series was calculated using tax return data, it does not include data for individual non-filers. It also does not account for cases of underreporting. However, based on relationships between six different income inequality indicators and total mortality rates in US states, Kawachi and Kennedy (1997) concluded that the choice of income distribution measure does not appear to alter the conclusion that income inequality is linked to higher mortality. Given that all the indicators were highly correlated with each other, we chose the top wage income share series collected from the 1910s to increase our sample size and increase the amount of variation in inequality over a much longer

historical time-period. Second, a causal relationship between income inequalities in early life and adult mortality risk cannot be inferred because there may be unmeasured mediators between two variables of interest. Third, there could be a selection bias since disadvantaged individuals may have already died. Thus, our results will be tempered by the conclusion that they apply only to current surviving members of each cohort. Fourth, income concentration information was given on an annual basis and we therefore cannot specify the exact level of income inequality that occurred precisely during gestation or the time period prior to gestation, which introduces some measurement error. Thus, we assumed birth year-1, birth year, and the following year as pregnancy and the first year of life, being aware of overlap between the periods. It is presumable that the benefits or adversities from income inequalities in early life may vary by race/ethnicity because of racial differences in social processes and interactions in labor markets and family environments, consequently in cause-specific mortality (Davey et al. 1998, Howard et al. 2000, Wong et al. 2002). Thus, we will conduct further studies stratified by race to examine heterogeneity in the relationships between income inequalities in early life and adult mortality risk across gender/racial subgroups.

Our study based on a life-course approach provides evidence that income inequality in early life has a lasting impact on mortality among males. Thus, it may be an evidence for advocating redistributive fiscal and tax policies to improve U.S. population health.

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Chapter 4, in full, will be submitted for publication of the material as it may appear in the *Social Science and Medicine*: Jongho H, Suzanne L, Shih-Fan L, Audrey NB, and Brian KF: Income Inequalities in Early Life and the Risk of Adult Mortality. Jongho Heo was the primary investigator and author of this paper.

Table 4.1. Characteristics of respondents (N=30,331 adults) from the General Social Survey linked to the National Death Index

	Male (n= 13,100)		Female (n=17,231)	
	Mean	SD	Mean	SD
Vital status				
Dead	3,986	30.4	4,706	27.3
Alive	9,114	69.6	12,525	72.7
Age	44.4	16.9	46.3	18.1
Education	13.0	3.2	12.6	2.9
Years of paternal education	10.7	3.6	10.6	3.5
Years of maternal education	11.0	3.1	10.8	3.3
Family income (1K dollars)	20.2	6.7	18.2	7.6
	N	%	N	%
Race				
White	11,203	85.5	14,054	81.6
Black	1,587	12.1	2,784	16.2
Other	310	2.4	393	2.3
Marital status				
Married	7,509	57.3	8,281	48.1
Never married	3,168	24.2	3,117	18.1
Widowed	558	4.3	2,649	15.4
Divorced or separated	1,865	14.2	3,184	18.5
Job status				
Full time job	8,420	64.3	6,942	40.3
Part-time	1,001	7.6	2,267	13.2
Retired	2,083	15.9	1,978	11.5
Unemployed or laid off	856	6.5	626	3.6
Other	740	5.6	5,418	31.4
Region				
West	2,421	18.5	3,034	17.6
Northeast	2,427	18.5	3,210	18.6
South	4,658	35.6	6,315	36.7
Midwest	3,594	27.4	4,672	27.1

Gender differences for the all variables were significant at $p<0.001$

Table 4.2. Gender-stratified regression estimates for all-cause mortality from multivariable parametric survival model with shared frailty using the General Social Survey linked to the National Death Index

	Male (n=13,100)			Female (n=17,231)		
	Hazard Ratio	95% CI	P	Hazard Ratio	95% CI	p
Income inequality at birth	1.04	1.01-1.07	0.004	1.01	1.01-1.07	0.597
Age	1.03	1.01-1.05	0.001	1.06	1.04-1.08	<0.001
Age squared	1.00	1.00-1.00	0.560	1.00	1.00-1.00	0.562
Race (reference: white)						
Black	1.44	1.30-1.59	<0.001	1.46	1.33-1.61	<0.001
Other	1.32	1.04-1.68	0.025	1.03	1.04-1.68	0.836
Education	0.99	0.97-1.00	0.020	0.98	0.79-1.00	0.026
Paternal education	1.01	0.99-1.02	0.380	1.01	1.00-1.02	0.230
Maternal education	0.98	0.97-1.00	0.014	0.99	0.98-1.00	0.146
Family income	0.99	0.98-0.99	<0.001	0.99	0.98-0.99	<0.001
Marital status (reference: married)						
Never married	1.01	0.91-1.14	0.784	1.05	0.93-1.22	0.345
Widowed	0.94	0.78-1.12	0.461	1.12*	1.01-1.25	0.033
Divorced or separated	1.05	0.95-1.16	0.358	0.95	0.86-1.06	0.393
Job status (reference: full time job)						
Retired	1.29	1.14-1.46	<0.001	1.32	1.15-1.51	<0.001
Unemployed	1.11	0.96-1.27	0.162	1.15	0.95-1.21	0.157
Part-time	1.06	0.92-1.23	0.427	1.08	0.95-1.21	0.238
Other	1.28	1.08-1.49	0.004	1.23	1.12-1.35	0.000
Region (reference: West)						
Northeast	0.92	0.82-1.04	0.193	0.88	0.78-0.99	0.031
South	1.02	0.91-1.13	0.779	0.93	0.84-1.03	0.181
Midwest	0.95	0.85-1.06	0.343	0.97	0.87-1.08	0.574

Table 4.3. Estimation results of multivariable parametric survival model with shared frailty for five periods of income concentration within top 0.5% around using the General Social Survey linked to the National Death Index

	Men			Women		
	Hazard Ratio	95% CI	p	Hazard Ratio	95% CI	p
Birth year-2	1.039	1.013-1.066	0.003	1.000	0.978-1.023	0.966
Birth year-1	1.040	1.013-1.068	0.004	0.999	0.976-1.022	0.900
Birth year	1.041	1.013-1.070	0.004	0.995	0.971-1.018	0.664
Birth year+1	1.038	1.001-1.068	0.010	0.999	0.975-1.023	0.911
Birth year+2	1.030	0.999-1.061	0.052	1.000	0.977-1.025	0.953

Note: Estimates were based on a main model which controlled for respondent's age, age^2, race, education, parental education, family income, marital status, job status, and region.

Table 4.4. Causes of death with hazard ratios and 95% confidence intervals (CIs) of multivariable parametric survival models with shared frailty for recession income concentration at birth among male using the General Social Survey linked to the National Death Index

Cause of death (deceased/total observed)	Hazard Ratio	95% CI	p
Infectious and parasitic diseases (170/9,119)	1.08	0.96-1.21	0.197
Cancers (791/9,740)	1.15	1.08-1.23	<0.001
Endocrine; nutritional; and metabolic diseases and immunity disorders (133/9,082)	1.16	1.04-1.30	0.008
Mental illness (57/9,006)	1.12	0.26-4.90	0.882
Diseases of the nervous system and sense organs (72/12,570)	1.16	0.96-1.39	0.115
Diseases of the circulatory system (1,045/9,994)	1.11	1.05-1.18	<0.001
Diseases of the respiratory system (178/9,127)	1.17	1.04-0.32	0.008
Diseases of the digestive system (137/12,635)	1.00	0.89-1.13	0.994
Diseases of the genitourinary system (18/ 12,516)	1.00	0.82-1.19	0.939
Suicide (41/8,990)	1.47	1.15-1.87	0.002
Others including injury-related deaths (494/12,992)	1.05	0.97-1.13	0.206

Note: Estimates were based on a main model which controlled for respondent's age, age^2, race, education, parental education, family income, marital status, job status, and region.

Table 4.5. Hazard ratios and 95% confidence intervals (CIs) of multivariable parametric survival models with shared frailty and interactions with family socioeconomic variables among males using the General Social Survey linked to the National Death Index

		Hazard Ratio	95% CI	p
Model 1[1)]	Income inequality at birth	1.04	1.01-1.07	0.004
Model 2[2)]	Income inequality at birth	0.99	0.94-1.04	0.730
	Income inequality*paternal education	1.004	1.00-1.01	0.027
	Income inequality*maternal education	1.0004	0.995-1.005	0.867

Note: 1) Main effect from a main model controlled for respondent's age, race, education, parental education, family income, marital status, and job status; 2) Adding interaction terms between Income concentration within top 0.5% at birth and family socioeconomic variables in Model 1

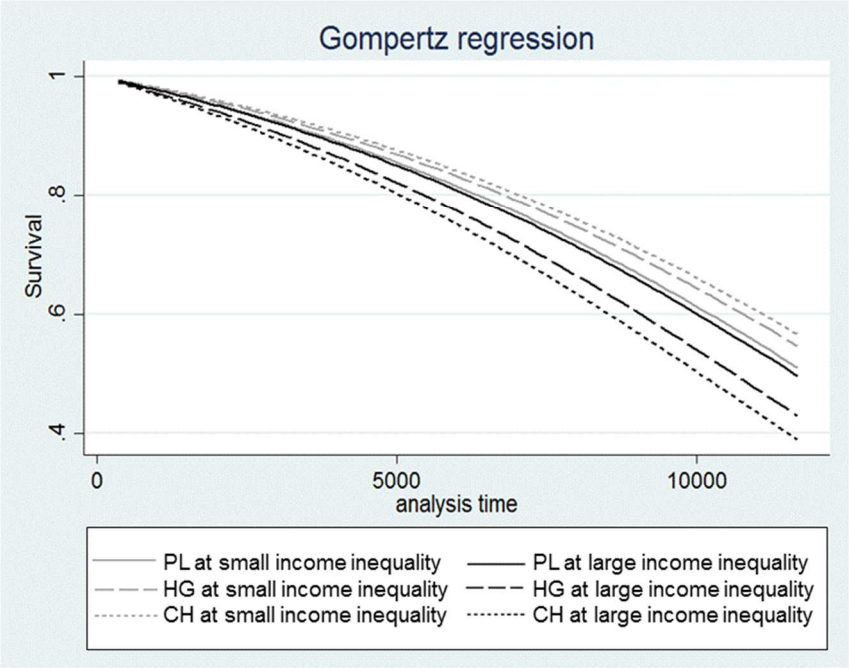

Figure 4.1. Interaction effects of paternal education and the income inequality at birth on mortality among males (n=13,100) from the General Social Survey linked to the National Death Index

Note: PL: primary school or lower, HG: high school graduation, CH: college or higher

Supplement table 4.1. Estimates of model fit across percentage of income concentration among males

Income concentration within top %	10%	5%	1%	0.5%	0.1%	0.01%
AIC	16323.87	16322.05	16319.37	16319.33	16320.82	16324.42

Note: AIC is the Akaike Information Criterion and is estimated to be -2*log(L) + df*2.

Supplement table 4.2. Results of sensitivity analysis among whole study population

	Original model		Compared model	
	Hazard Ratio	SE	Hazard Ratio	SE
Income concentration within top 0.5% at birth (excluded realized capital gains)	1.05***	0.02		
Income concentration within top 0.5% at birth (included realized capital gains)			1.02*	0.01
Age	1.04***	0.01	1.04***	0.01
Race (reference: white)				
Black	1.42	0.05	1.42	0.05
Other	1.15	0.11	1.14	0.10
Education	0.98***	0.001	0.98***	0.001
Paternal education	1.00	0.004	1.00	0.004
Maternal education	0.99*	0.01	0.99*	0.001
Family income	0.99***	0.002	0.99***	0.002
Marital status (reference: married)				
Never married	1.05	0.05	1.04	0.05
Widowed	0.95	0.04	0.95	0.04
Divorced or Separated	0.93	0.03	0.92	0.03
Job status (reference: full time job)				
Retired	1.28***	0.06	1.27***	0.06
Unemployed	1.16**	0.07	1.16**	0.07
Part-time	0.95	0.04	0.95	0.04
Other	1.00	0.04	1.00	0.04

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CHAPTER 5: Discussion

This dissertation examined how cohort-based income status affects population health (obesity and mortality) using multiple years of nationally representative U.S. data and combining theory and methods from demography, sociology, and public health. The dissertation also critically examined the contribution of the cohort effects on health disparities across gender and racial subgroups.

Analyses in chapter 1 revealed weaker income gradients in obesity among post-world war generations, the mid-1920s, mid-1940s, and 1950s cohorts, than the other cohorts. Moreover, cohort-based income gradients in obesity varied markedly by gender and race/ethnicity. In the second study, I found that exposure to recession in the first year of life was associated with an increase in all-cause mortality only among women and mortality from cancers. In the third study, I found that higher income inequality in early life was associated with increased risk of all-cause mortality among males and mortality from various diseases. Paternal education was estimated to have moderated these relationships in the second and third study.

The majority of previous studies on obesity and mortality have relied on two approaches focusing on effects of either age or period. My dissertation demonstrated cohort effects, which are external to individuals and operate at a macro level, played significant roles in driving the dynamic patterns in obesity and mortality over time. The cohort represents a unique socio-demographic concept suggesting that while dynamic

change is possible, social forces play out on each successive birth cohort. Specifically, cohort effects are salient to understanding gender and racial health disparities given that gender and race represent differential effects access to social resources and different risk-exposure. Thus, my dissertation suggests that persistent health disparities may reflect persistent unequal social forces across gender and racial subgroups. It may also present a unique opportunity to study how cohorts change over time and ultimately affect the health of the subgroups.

These findings, especially from chapter 2, also suggest that FCT is needed to integrate with other intersectional social dimensions of health inequality by adopting a temporal view of SES and health. The theory highlights the persistence of associations between SES and health outcomes regardless of changing mediators. Consistently with recent studies testing the FCT, my dissertation showed that the premise generally held yet also uncovered a peculiar challenge to its tenets (Miech et al. 2011, Polonijo and Carpiano 2013). As shown in chapter 2, the relationship between SES and health has evolved across cohorts and further showed divergent patterns within the cohorts across gender and racial subgroups. Thus, my dissertation suggests that more research is needed to consider synthesizing FCT, life-course theory, and racial and gendered contexts.

Extant literature suggests that arrangement in one's early life negatively impact health and survival at older ages (Case and Paxson 2009, van Den Berg, Lindeboom, and Portrait 2006, van den Berg, Doblhammer, and Christensen 2009, Doblhammer, van den Berg, and Fritze 2011). My dissertation, especially chapter 2 and 3, contributes to

existing life-course studies by demonstrating the long-lasting effects of macroeconomic status in early life on mortality. It was hypothesized that poor macroeconomic conditions (higher income inequalities) at the time of birth can act as a household stressor, reducing available resources, medical care and nutrition, and increasing exposure to diseases. Evidence suggests that these pathways may operate through epigenetic changes during critical periods of development. Barker (1997) argues that fetal under-nutrition can lead to cell division deficiencies that increase the risk of cardiovascular disease and mortality. Related to household stressors, Miller and Chen (2010) found that being raised in harsh family environments was associated with a greater pro-inflammatory phenotype over time. Doblhammer and colleagues (2011) argued that the pro-inflammatory phenotype can create a long-term allostatic toll, resulting in a higher risk of chronic diseases. My dissertation showed an obvious discrepancy of findings. In the second study, I found that exposure to recession in the first year of life was associated with an increase in all-cause mortality only among women and mortality from cancers. In the third study, I found that higher income inequality in early life was associated with increased risk of all-cause mortality only among males. It is still unclear why the macroeconomic index in early life had gender-based differential effects as there is little gender-stratified research. The discrepancy may be caused by the gender differences in behavioral, psychological, and social mechanisms to the household stressors caused by macroeconomic index. Moreover, genetic and epigenetic differences between genders may intertwine with the mechanisms complicating the task to elucidate the mechanisms. However, the

moderating effects of paternal education found in my dissertation may hint that parents play a crucial role in the mechanism.

Strengths and limitations

Several major limitations are worth noting. First, causal relationships cannot be inferred due to possible reverse causality, unmeasured mediators, and possible selection bias. Second, due to small sample size of the earliest and last cohorts, I did not estimate the oldest cohorts. Interpretation of findings in the latest cohorts should be made carefully because the estimate is subject to revision as more data become available. Third, there could be a selection bias because disparities may be understated since members of disadvantaged population groups have already died. There are various approaches for dealing with this problem (Beckett 2000), but none have been shown to accomplish the impossible. Thus, the results may be tempered by the conclusion that these results apply only to current surviving members of each cohort. Fourth, in chapter 2, controlling for specific period effects was not allowed because the NHANES has survey gaps during 1981-1987, 1990, and 1994-1999, that may result in residual confounding by period effects. Fifth, the GDP, which was used to calculate the economic condition in early life in chapter 2, has been criticized for not accurately reflecting individual economic status if the distribution of economic benefits is concentrated among a small percentage of the population (Sen 1976, 1979, Van den Bergh 2007). For that reason, several studies favored unemployment rates to GDP. However, we used GDP cycle which was generated by applying the Hodrick-Prescott

filter (Hodrick and Prescott 1997), as the index has been used widely in macroeconomics and demography to capture clear transitory macroeconomic conditions.

Despite these limitations, my findings highlight the importance of cohort effects in health and health inequality studies using cutting-edge statistical methods for cohort studies. I also used long-range data span, which is favored for generalizability of study findings. Moreover, racial- and gender-stratified analyses were conducted, which have been rarely conducted even it is clear that the effects of social forces are not uniform by sex or race/ethnicity.

The findings in my dissertation suggest cohort-specific strategies to reduce the racial and gender gaps in obesity and mortality. For example, the findings from chapter 1 imply that policies or interventions to reduce obesity disparities need be prioritized to lower income groups born during the Great Depression, the mid-1960s, and 1970s. Based on the findings from chapter 2 that bad economic conditions independently deteriorate male's mortality in later life, it may be advisable to develop health interventions targeted to male infants born during the recession and their families.

Conclusions

A cohort perspective is largely absent from recent epidemiological analyses of the drivers of health disparities. This dissertation focused on birth cohort effects and suggests unrealized potential contributions of cohort analyses in analyzing health outcomes and health disparities across gender and racial subgroups.

Results intimate that a shift from over-emphasized individualism to perspectives

of macro socioeconomic and generational determinants in gender and racial disparities in health are needed. They also suggest cohort-specific strategies are needed to develop health interventions or health policies targeted to specific generations or racial sub-groups.

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