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ORIGINAL RESEARCH

Divergent Trends in the Effects of Early Life Factors on Adult Health

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Abstract

Life course theories have shaped social and health scientists' understanding of the origins and pathways of health, aging, and mortality. However, few studies have examined how these origins might have changed across cohorts. This study investigates the impact of birth, childhood, and adolescence factors on adult health across birth cohorts born in the second half of the twentieth century in the United States. Data come from the Panel Study of Income Dynamics Family and Individual Files 1968–2013 and the Childbirth and Adoption History File 1985–2013. Multilevel growth models are used to capture the growth trajectories of two adult health outcomes: self-rated health and health summary index. We find the association between three pre-adulthood factors (birth weight, mother's education, childhood family income-to-needs ratio) and health outcomes weakens in more recent cohorts, while the association strengthens for the other two early life factors (early-life disease index and parental smoking status before age 17). These findings demonstrate the complexity of the social-to-biological embodiment across the life course, and suggest that the effects of early-life factors on adult health can increase or decrease across cohorts due to macro social, economic, policy, technological, and medical changes. They also illuminate the long-term debate on the period and cohort effects in shaping the health trend, and suggest that the cohort effect is multidimensional and is weaker or stronger depending on the dimension of early life examined.

Keywords Cohort analysis · Life course analysis · Early origins of health · Birth weight · Early-life diseases · Childhood family background

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Life course theories have shaped social and health scientists' understanding of the origins and pathways of health, aging, and mortality. Since the late 1980s' seminal work of David Barker et al. on the fetal origins of adult disease (Barker 1990), many researchers have elaborated on the life course model of health and illness by extending the fetal origins hypothesis to include other early-life factors (e.g., mother's education, parental income, early-life health status) (e.g., Ben-Shlomo and Kuh 2002; Blackwell et al. 2001; Case et al. 2005; Case and Paxson 2010; Elo and Preston 1992; Freedman et al. 2008; Hayward and Gorman 2004; Johnson and Schoeni 2011). However, few studies have examined how these origins might have changed across birth cohorts. It is not clear whether some early-life factors that were consequential for adult health in the past are still relevant today, or if other early-life factors are emerging that will be increasingly important for more recent cohorts.

Patterns of health and aging should be embedded in historical context (Ryder 1965; Riley 1973). Each cohort moves through the life course with its own unique story; therefore, the origins and pathways of health may shift across cohorts. Understanding how life course trajectories differ across individuals and birth cohorts provides important mechanistic and etiological clues about how health and aging unfold (Ben-Shlomo et al. 2016). In this study, we contend that in order to understand how the origins of health may shift across cohorts, we need to place them in the context of macro social, economic, technological, and medical changes. Using Panel Study of Income Dynamics data 1968–2013 (PSID 2013), we focus on four birth, childhood, and adolescence factors (i.e., birth weight, early-life health, parental smoking, and parental socioeconomic status). We first review the literature on the period and cohort effects in the trend of mortality decline that may shed light on the changing effect of early life factors on adult health, then review the historical context that may alter the effects of these factors. We develop competing predictions from literature with regard to how the effects of early life factors on adult health may change, and then use PSID data to conduct empirical tests.

Existent Approaches and Limitations

Numerous studies in demography, sociology, and epidemiology have explored the impact of early-life factors on health and aging in later life (e.g., Ben-Shlomo and Kuh 2002; Hayward and Gorman 2004; Johnson and Schoeni 2011; Luo and Waite 2005), but few studies have examined how these early-life catalysts change across birth cohorts. Some demography research has focused on disentangling age, period, and cohort effects (Masters et al. 2012; Yang 2008; Zheng et al. 2016). This line of work examines the relative contribution of period and cohort forces to the mortality decline in developed countries and provides some indirect evidence of the changing impact of early-life conditions on later-life health.

Epidemiologic transition theory has long been utilized in demography and epidemiology to explain historical mortality declines in developed countries' populations, while a growing body of work has advanced cohort-based observations and explanations such as "cohort morbidity phenotype" (Finch and Crimmins 2004) and "technophysio evolution" (Fogel and Costa 1997). These contemporary theories

emphasize the enduring consequences of early-life inflammation and nutrition. When later cohorts have an advantage over earlier cohorts due to lower exposure to infection and inflammation or improved nutrition, they will maintain this advantage over the life course. This then links old-age mortality decline in later periods to young-age mortality decline in earlier periods. The cohort effect may have been especially strong since the 1960s when the major causes of death shifted from infectious diseases to chronic illnesses in the recent stage of the epidemiologic transition known as the age of delayed degenerative diseases (Yang 2008). This is because chronic diseases are influenced by early life socioeconomic (SES), behavioral and medical conditions, but span longer time intervals since their effects on mortality take longer to emerge. If cohort effect on mortality decline is stronger in the second half of the twentieth century, the effects of early-life conditions on later health should have increased across birth cohorts.

Finch and Crimmins (2004), however, argue that cohort effects originate from reductions in exposure to inflammation in early life. Therefore, when childhood infection has been low, the effect of early-life conditions on later life mortality should be weak. Similarly, Zheng (2014) find that the effect of early life factors on rate of biological aging might have decreased since the early twentieth century birth cohort. Barbi and Vaupel (2005) argue that the cohort contribution is overstated, as the differences in infant death rates between the two populations do not accurately predict the differences in death rates in old age between the corresponding birth cohorts. Instead, period effects trump cohort effects, especially for older age groups in developed countries after the 1950s (Vaupel et al. 1997). Heart diseases, cerebrovascular diseases, smoking-related cancers, and all other cancers related to mortality declined across all adult age groups in the United States from 1940 to 2002 (Crimmins 1981; Jemal et al. 2005; Kannisto et al. 1994; Ouellette et al. 2014). These results imply that period factors (e.g., cardiovascular revolution) may have been the dominant forces driving the mortality decline since the 1950s. If this is true, the effects of early-life conditions on later health should have decreased across birth cohorts.

Therefore, this period-cohort debate leads to competing predictions on the effect of early-life factors on health across cohorts. Even though all these aforementioned studies are informative, they neglect the heterogeneity in early-life factors and how social, economic, and technological changes may interact with these factors and shape divergent trends. In order to better understand this complexity and shed light on the relative contribution of period or cohort effects, research needs to take a more mechanical approach to disaggregate early-life factors and put each of them in the context of macro social, economic, technological, and medical changes.

This approach, however, was not feasible until recently when we started having the data to do so. Montgomery et al. compare how the impact of family conflict on children's height growth before age 7 may have changed from the 1958 birth cohort to millennium birth cohort in the UK (Montgomery et al. 1997; Kelly et al. 2010). They find that family conflict was associated with height growth in boys up to the pre-pubertal growth spurt at age 7 in the 1958 birth cohort, but this association was not replicated in the year 2000 cohort. They hypothesize that family conflict is a marker for economic adversity. Growing up in the obesogenic environment, poorer

children in the later cohorts would be more likely to have higher weight, which is associated with faster pre-pubertal height growth.[1] This work demonstrates the complexity of the social-to-biological embodiment over the early life and the importance of the environment (Kelly-Irving et al. 2013; Krieger 2005). Built on these prior studies, we focus on four early-life factors: birth weight, early-life health, parental smoking, and parental socioeconomic status. We dig into the macro socioeconomic and environmental changes in the United States in the second half of the twentieth century and examine how they may moderate the impact of each early-life factor.

Early Life Origins in Historical Context

Birth Weight

Low birth weight is a major cause of perinatal, infant, and childhood morbidity. However, the United States and other developed countries have experienced an increase in the survival rates of low birth weight infants since 1975 (Goldenberg and Culhane 2007). The development of Neonatal Intensive Care Units (NICUs) in the 1970s and improvement of health treatments for premature infants may be the cause behind the decline. As physicians gained a better understanding of the needs of premature infants and mechanical incubators reduced respiratory distress in babies with underdeveloped lungs, the survival rate improved (Penn Nursing 2018). Before the 1970s, most infants born 3 months premature died due to their inability to breathe on their own. In the 1980s and 1990s, new and improved treatments were introduced in NICUs that further contributed to the survival of low birth weight babies (Phibbs et al. 2007). For example, surfactant therapy, which reduces the tension of fluid in the lungs, was developed during the 1980s and widely implemented in the 1990s (Penn Nursing 2018). This led to a decrease in bronchopulmonary dysplasia, which is an abnormal development in lung tissue that results from long-term ventilation. Improved medical treatments for premature and low birth weight infants not only increase their survival, but also have long-term benefits for cognitive development and health. A recent study conducted in the UK finds that the association between low birth weight and lower cognitive ability has declined over time (Goisis et al. 2017). That study attributes this to advancements in obstetric and neonatal intensive care since the 1970s.

But at the same time, due to medical advancements to treat premature and low birth weight babies, more individuals who would have otherwise died earlier in life are capable of reaching adulthood. These individuals nonetheless may suffer from the long-term negative consequence of low birth weight, including school difficulties during youth, hypertension, non-insulin dependent diabetes mellitus, dyslipidemia, increased body fat, and chronic diseases during adulthood (Curhan et al. 1996; Saigal et al. 2000; Valdez et al. 1994). Hack et al. (1995) finds that there have been improvements in the survival rates of low birth weight babies but there have

[1] We thank an anonymous reviewer for suggesting this research and the implications for our study.

not been changes in the rates of cerebral palsy and neurodevelopmental handicap among low birth weight infants since the introduction of neonatal intensive care.

Therefore, the cohort trend in the association between low birth weight and adult health may go either way. As more advancements improve care of these infants, the preterm and low birth weight babies may continue to experience better health and lower risk of mortality in childhood as well as adulthood. Thus, the association between low birth weight and adult health may have declined across birth cohorts in the second half of the past century. But increasing survival of weaker individuals may strengthen the association between lower birth weight and adult health.

Childhood Health

Even though intensive medical care has increased the survival of low birth weight infants, children have experienced a growing number of diseases during the same time period. For instance, childhood obesity, cancer, and allergic disease have been on the rise (Beggs and Bambrick 2005; Charpin and Gouitaa 2001; Schmidt 1998; Wang and Lim 2012). A report from the World Health Organization (WHO) in 2016 indicates that the number of overweight or obese school-aged children has increased since 1975 in the United States, reaching levels of around 30% in recent years. Schmidt (1998) explicates that cancer is the leading cause of disease-related mortality among children under the age of 15 in the United States. The rate of children diagnosed with acute lymphoblastic leukemia has increased during the 1970s and 1980s. Other forms of childhood cancer (e.g., Wilm's tumor and non-Hodgkin's lymphoma) have also increased. Tobacco, food additives, certain medications, viral infections, and industrial and agricultural chemicals are possibly the causes for the rise in cancer incidence rates among children (Schmidt 1998).

Moreover, the prevalence of atopic disorders and asthma has risen dramatically since the 1960s (Beggs and Bambrick 2005). Atmospheric carbon dioxide concentration may have contributed to the growth of respiratory diseases (Beggs and Bambrick 2005). Since 1950, sulfur dioxide emissions in the United States have increased, reaching their highest peak in 1970. Due to concerns over the impact of these emissions, the United States began producing low-sulfur coal, which was the main driver of emission reductions after 1970 (Smith et al. 2011). Nevertheless, the impact of emissions on previous generations is still a matter of concern. Fuel combustion has detrimental effects on children's health. In fact, fetuses and young children are more vulnerable to the adverse effects of air pollutants than adults. Thus, exposure to these pollutants could have long-term consequences for child health. Lower lung function, exacerbated asthma, airway inflammation, and airway oxidative stress are some of the most common health outcomes associated with exposure to air pollutants (Andersen et al. 2008; Perera 2017; Tzivian 2011). Neurodevelopmental disorders have also been linked to air pollutants. Perera et al. (2012) find that prenatal exposure to polycyclic aromatic hydrocarbons is associated with symptoms of anxiety, depression, and attention deficit among children.

Childhood diseases have a long lasting effect on adult health (Adler et al. 1994; Case et al. 2005). For example, asthma has a long-term effect as it is associated

with airway obstructions, chest tightness, shortness of breath, and other pulmonary/breathing problems (Toelle et al. 1992). Using the National Health Interview Survey and the National Longitudinal Study of Adolescent Health data, Fletcher et al. (2010) find that ever having asthma is associated with worse self-rated health status, increases in obesity, and more days of school/work missed. Due to the increasing number of diseases experienced in childhood, we would expect the association between early childhood health status and adulthood health status strengthens across cohorts. Survival of these kids into adulthood due to effective medical treatments may further strengthen this association.

Family Socioeconomic Background

Family socioeconomic background (e.g., parental education and income) has long been regarded as important determinants of children's health (Gage et al. 2012; Glewwe 1999; Schultz 1984; Thomas et al. 1991). But how the importance of family background changes across cohorts is still uncertain. Many studies find health disparities by adult SES have substantially widened over time and across cohorts in the U.S. (e.g., Case and Deaton 2015; Lauderdale 2001; Masters et al. 2012; Olshansky et al. 2012; Pappas et al. 1993), and rising income inequality partially contributes to the increase (Zheng and George 2012). Therefore, whether family socioeconomic background becomes a more important determinant for adult children's health today than in the past may partially depend on whether it becomes more influential for their socioeconomic status. Chetty et al. (2014) find that even though the correlation between parent and child income percentile ranks has remained stable for the cohorts born between the 1970s and 1980s, a child's income depends more heavily on his parents' position in the income distribution over time because of the increase in income inequality. If this is the case, we would expect the association between family socioeconomic background and offspring health to strengthen across birth cohorts.

But at least two countervailing forces may offset or dilute the importance of family socioeconomic background. First, as many prior studies have concluded (e.g., Meara et al. 2008; Olshansky et al. 2012; Pappas et al. 1993), because attained status (e.g., adulthood education and income) is an increasingly important determinant of an individual's health, ascribed status (e.g., family background) may become viewed as less important over time. Second, many social and policy changes (i.e., development of childcare assistance policies and financial aid for low-income families) may also reduce the impact of family background on adult children's health. Crosby et al. (2005) explain that childcare policies in the late 1980s to the late 1990s made childcare centers more affordable for low-income families. Childcare quality gives low-income parents the ability to provide for an environment that enhances the cognitive, behavioral, and social development of children. Childcare assistance available through government programs, including welfare and employment programs, also has a positive effect on employment, income, and children's health outcomes (Gennetian et al. 2004; Bloom and Michalopoulos 2001). The primary objective of welfare and employment programs for low-income families has been to increase

parents' self-sufficiency by requiring and supporting employment. Since the 1970s, some of these programs have expanded benefits to include childcare assistance and health insurance in order to reward work outside the welfare system. These programs increased parental employment and the use of childcare centers (Gennetian et al. 2004), led to higher school achievement, reduced problem behaviors, and improved children's overall health (Morris et al. 2001).

Therefore, prior literature provides competing directions on the changing impact of family background on adult children's health across cohorts. Rising income inequality and increasing importance of parental income on child income across generations may imply a strengthening association across cohorts. Meanwhile, increasing importance of adulthood conditions on health may dilute the impact of family background. The large implementation of government programs and the positive outcomes associated with the use of these may further reduce the impact of disadvantaged family socioeconomic background on adult children's health for recent cohorts.

Parental Smoking

Smoking is a major cause of respiratory and cardiovascular disease as well as cancer. Since 1965, the United States has experienced a steady decline in the number of adults that smoke cigarettes. In 1965, 42.4% of American adults smoked cigarettes. More than 50 years later, only 14% of U.S. adults smoke (CDC 2018). The decrease in smoking is also accompanied by a decrease in exposure to secondhand smoke (Pirkle et al. 2006). Secondhand smoke causes ear infections, asthma, respiratory symptoms, respiratory infections, and an increased risk of sudden infant death syndrome (CDC 2019; Difranza et al. 2004). Due to the steady decline in smoking prevalence among adults, we might expect the association between parental smoking and offspring health has weakened across cohorts.

Even though cigarette smoking has declined, the negative health outcomes associated with smoking continue to be a matter of public concern. Thun et al. (2013) find that the risks of death from smoking substantially increase from the 1960s, 1980s, to contemporary cohorts for female smokers. Among men, the risks associated with smoking have plateaued at the high levels in the 1980s, but mortality from chronic obstructive pulmonary disease (COPD) continues to increase in contemporary cohorts in nearly all the age groups. Edwards et al. (2017) report an increase in rates of lung cancer among smokers in the United States in recent decades.

These rising mortality risks associated with smoking may be related to the chemical components used in cigarette additives and their influence on tobacco addiction (Edwards et al. 2017). Cigarette additives contribute to a greater rate of addiction and worse health outcomes because these components hide the odor of tobacco smoke and increase the amount of nicotine delivery (Rabinoff et al. 2007). Levulinic acid, which is an additive used to improve the flavor of cigarettes, may enhance the binding of nicotine to neurons that are not normally responsive to nicotine (Keithly et al. 2005). Furthermore, levulinic acid is associated with a decrease in olfactory responses and may increase mainstream smoke (i.e., the smoke exhaled

by smokers). In addition to levulinic acid, there are other additives used in cigarettes that are believed to entice people to smoke (Rabinoff et al. 2007) and chemicals that have carcinogenic potency, such as tobacco-specific nitrosamines (TSNA) (Stepanov et al. 2011).

The presence of levulinic acid or levulinated nicotine, TSNA, and other components in cigarettes not only contribute to worse health outcomes among smokers but also put their children at a higher risk of diseases due to exposure to tobacco constituents during fetal development and through environmental tobacco smoke. In addition, as the negative health outcomes associated with smoking become more realized by the society, smoking increasingly becomes a strong marker of both material and cultural circumstances. Individuals who continue to smoke may become a more selected group who may be more concentrated at the lower and lowest socioeconomic status, have worse health behaviors and face greater risks of developing serious health problems, that could impact the health status of their children, either during birth or after birth. For the foregoing two reasons, it is likely that even though the percent of individuals who smoke in the U.S. has declined, the health consequence of smoking continues to increase, which may have caused a strengthening association between parental smoking and offspring health across cohorts.

Data and Methods

We use data from the Panel Study of Income Dynamics (PSID 2013). This survey began in 1968 with a nationally representative sample of families. The survey was administered annually until 1997, then biennially thereafter. We use the Family and Individual Files 1968–2013 and the Childbirth and Adoption History File 1985–2013. Children from the original 1968 families are interviewed in the Family Files after they become the head or spouse in a household. Parental information for respondents was linked using the Family Identification Mapping System (FIMS) that identified each individual's father and mother. This allows for detailed parental information about respondents who have parents in the PSID. Parental linkages are used to obtain a respondent's birthweight and childhood family income-to-needs ratio.

Health outcomes are obtained from questions asked of the head of household and spouse in the Family Files. Sample sizes vary for the two health outcomes analyzed in this study because of differences in the number of waves in which each health outcome is observed. Self-rated health is observed in the Family Files from 1984 to 2013. The total number of head of household and spouse observations during these waves is 281,113. We restrict our sample to heads of households and spouses born in 1950–1989, reducing our potential sample to 159,848. Missing data for self-rated health and control variables (e.g., age, race, gender, and adulthood SES characteristics) further reduces the sample to 150,445. Health summary index is obtained from questions asked in the Family Files from 1999 to 2013. During these waves, there are 99,459 head of household and spouse observations. Of those observations, 70,326 were gathered for respondents born in 1950–1989. Missing data for health summary index and control variables reduces the final sample size to 63,574.

We conduct analyses by each early-life factor separately. We use mother's education and childhood income-to-needs ratio as two indicators for family socioeconomic background. Sample sizes vary depending on the main explanatory variable analyzed. The smallest samples occur for birthweight and childhood income-to-needs ratio because these variables are obtained from parental linkages. Birthweight is obtained from the Childhood and Adoption History File. Childhood family income-to-needs ratio is measured from parental information obtained from the Family Files. Both variables require respondents' parents to be in the PSID.[2] The largest sample sizes are for variables obtained from Family File questions; respondents were asked about mother's education in 1974–2013, and early-life diseases and parental smoking in 2007–2013. Tables 1, 2 display the descriptive statistics of these explanatory variables.

Measures

Dependent Variables

We analyze two health outcomes, self-rated health and a health summary index. Self-rated health is obtained from a single question asking respondents to rate their overall health on a scale of 1 to 5, with 1 being excellent and 5 being poor, so higher number means poorer health. The health summary index consists of 10 health problems respondents might have. Respondents were asked if a doctor ever told them that they had one of the following conditions: stroke, diabetes, chronic lung disease, high blood pressure, cancer, heart attack, heart disease, emotional problems, arthritis, or asthma. A respondent's health summary index score consists of the sum of all conditions he or she reported, and scores for this index ranged from 0 to 10. Both outcome variables are time varying, and treated as continuous variables.

Main Explanatory Variables

Our main explanatory variables of interest consist of five early-life factors. Birthweight is measured as a dummy variable with 0 indicating that a respondent's birthweight was 5.5 lb or less and 1 indicating more than 5.5 lb. Early-life disease index consists of the sum of any of the 12 health problems a respondent reported before age 17, and scores for this index range from 0 to 12. These health problems are asthma, diabetes, respiratory disease, allergies, heart trouble, epilepsy, severe headaches/migraines, stomach problems, high blood pressure, depression, drug/alcohol problem, and emotional/psychiatric problem. Parental smoking before age 17 is a dummy variable with 1 indicating that at least one parent/guardian smoked when the respondent was 0 to 17 years old. Mother's education consists of five categories: 1 = did not graduate from high school, 2 = high school graduate, 3 = some college, 4 = college graduate, and 5 = graduate degree. Childhood family income-to-needs

[2] Mother's identification and information were used for these links.

Table 1 Descriptive statistics by birth cohort for self-rated health sample

	Self-rated health									
	Full sample		1950 cohort		1960 cohort		1970 cohort		1980 cohort	
	Mean (stan. dev.)	*N*	Mean (stan. dev.)	*N*	Mean (stan. dev.)	*N*	Mean (stan. dev.)	*N*	Mean (stan. dev.)	*N*
Independent variables										
Age	35.44 (8.94)	150,445	39.94 (8.47)	69,274	33.16 (7.99)	49,163	30.43 (5.94)	22,228	26.41 (3.29)	9780
Female	0.53 (0.50)	150,445	0.52 (0.50)	69,274	0.55 (0.50)	49,163	0.53 (0.50)	22,228	0.53 (0.50)	9780
Black	0.35 (0.48)	150,445	0.35 (0.48)	69,274	0.35 (0.48)	49,163	0.31 (0.46)	22,228	0.36 (0.48)	9780
Hispanic	0.05 (0.21)	150,445	0.04 (0.20)	69,274	0.05 (0.21)	49,163	0.05 (0.21)	22,228	0.06 (0.23)	9780
Birth weight	0.93 (0.26)	80,018	0.92 (0.27)	34,220	0.93 (0.25)	27,618	0.93 (0.26)	12,556	0.93 (0.26)	5624
Early life disease index	0.44 (0.88)	105,981	0.34 (0.78)	43,470	0.39 (0.83)	33,720	0.62 (1.05)	19,536	0.65 (1.04)	9255
Mother's education	2.10 (1.03)	141,142	1.92 (0.97)	64,828	2.08 (0.98)	46,215	2.43 (1.08)	20,869	2.64 (1.13)	9230
Childhood income-to-needs	2.43 (1.82)	87,565	2.30 (1.78)	36,726	2.41 (1.63)	30,079	2.66 (1.83)	13,880	2.77 (2.58)	6880
Parental smoking before 17	0.61 (0.49)	106,311	0.65 (0.48)	43,477	0.62 (0.49)	33,990	0.57 (0.50)	19,547	0.47 (0.50)	9297
Highest grade completed	13.20 (2.07)	150,445	13.20 (2.12)	69,274	13.05 (1.99)	49,163	13.39 (2.08)	22,228	13.52 (2.04)	9780
Adult income-to-needs	3.88 (4.08)	150,445	4.28 (4.48)	69,274	3.66 (3.78)	49,163	3.56 (3.76)	22,228	2.92 (2.58)	9780
Working	0.78 (0.42)	150,445	0.78 (0.41)	69,274	0.77 (0.42)	49,163	0.77 (0.42)	22,228	0.74 (0.44)	9780
Disabled	0.02 (0.15)	150,445	0.03 (0.17)	69,274	0.02 (0.13)	49,163	0.02 (0.13)	22,228	0.01 (0.10)	9780
Dependent variables										
Self-rated health	2.23 (0.98)	150,445	2.31 (1.01)	69,274	2.15 (0.94)	49,163	2.17 (0.95)	22,228	2.14 (0.93)	9780

Table 2 Descriptive statistics by birth cohort for health summary index sample

	Health summary index									
	Full sample		1950 cohort		1960 cohort		1970 cohort		1980 cohort	
	Mean (stan. dev.)	*N*	Mean (stan. dev.)	*N*	Mean (stan. dev.)	*N*	Mean (stan. dev.)	*N*	Mean (stan. dev.)	*N*
Independent variables										
Age	39.71 (10.26)	63,574	51.17 (5.35)	18,736	41.94 (5.27)	17,713	32.55 (4.79)	17,359	26.41 (3.29)	9766
Female	0.53 (0.50)	63,574	0.53 (0.50)	18,736	0.54 (0.50)	17,713	0.51 (0.50)	17,359	0.53 (0.50)	9766
Black	0.33 (0.47)	63,574	0.33 (0.33)	18,736	0.35 (0.48)	17,713	0.31 (0.46)	17,359	0.36 (0.48)	9766
Hispanic	0.04 (0.20)	63,574	0.03 (0.18)	18,736	0.04 (0.19)	17,713	0.04 (0.20)	17,359	0.06 (0.23)	9766
Birth weight	0.93 (0.26)	33,054	0.92 (0.27)	8924	0.94 (0.24)	9120	0.93 (0.26)	9393	0.93 (0.26)	5617
Early life disease index	0.48 (0.93)	59,072	0.34 (0.77)	17,067	0.40 (0.85)	16,331	0.61 (0.85)	16,428	0.66 (1.04)	9246
Mother's education	2.29 (1.07)	58,203	2.03 (0.98)	16,748	2.18 (1.00)	16,034	2.47 (1.09)	16,201	2.64 (1.13)	9220
Childhood income-to-needs	2.63 (1.99)	36,762	2.51 (1.87)	9481	2.55 (1.71)	9958	2.74 (1.86)	10,453	2.77 (2.58)	6870
Parental smoking before 17	0.59 (0.49)	59,276	0.65 (0.48)	17,070	0.62 (0.49)	16,487	0.57 (0.50)	16,433	0.47 (0.50)	9286
Highest grade completed	13.54 (2.08)	63,574	13.52 (2.12)	18,736	13.45 (2.05)	17,713	13.65 (2.08)	17,359	13.52 (2.04)	9766
Adult income-to-needs	4.41 (4.96)	63,574	5.54 (6.19)	18,736	4.54 (5.09)	17,713	3.90 (3.99)	17,359	2.93 (2.58)	9766
Working	0.78 (0.41)	63,574	0.77 (0.42)	18,736	0.81 (0.40)	17,713	0.79 (0.41)	17,359	0.74 (0.44)	9766
Disabled	0.03 (0.18)	63,574	0.06 (0.23)	18,736	0.04 (0.19)	17,713	0.02 (0.14)	17,359	0.01 (0.10)	9766
Dependent variables										
Health summary index	0.65 (1.02)	63,574	0.98 (1.23)	18,736	0.60 (0.98)	17,713	0.50 (0.85)	17,359	0.40 (0.71)	9766

ratio is a continuous variable that measures the respondent's average family income-to-needs ratio from age 0 to 17. Our focus is on testing the associations between these five early-life factors and adult health and how these associations change across birth cohorts. Ten-year birth cohort dummies are constructed: 1960–1969, 1970–1979, and 1980–1989, with 1950–1959 as the reference group.

Control Variables

All the analyses are adjusted for respondent's age, sex, race, highest grade completed, adult income-to-needs ratio, employment status, and survey year. Age and a square term of age are included, and both are grand-mean centered. Sex is a dummy variable where 0 = male and 1 = female. Race consists of three groups: Black and Hispanic with White as the reference. Respondents' highest grades completed is a continuous variable, which ranges from 1 to 17. Adult income-to-needs ratio is also a continuous variable and measured as a respondent's adulthood family income divided by the U.S. Census Bureau's defined needs of the family (PSID 2013). Employment status is measured by two dummy variables: working (0 = not working, 1 = working) and disabled (0 = not permanently disabled, 1 = permanently disabled). Survey year includes a set of year dummies. All information for these control variables was obtained from the Family and Individual Files.

Methods

We estimate multilevel growth models using Stata's xtmixed command. All models are random-intercept and random-slope models with an unstructured variance–covariance and are estimated using maximum likelihood. The basic model setup is specified as:

$$Y_{it} = \beta_0 + \beta_1 Age_{it} + \beta_2 AgeSq_{it} + \sum_j \beta_j C_{ji} + \sum_k \beta_k X_{ki} + \sum_p \beta_p Z_{pit} + \vartheta_{0i} + \vartheta_{1i} Age_{it} + \varepsilon_{it}$$

where Y_{it} is individual i's health outcome (self-rated health or health summary index) at age t. Age_{it} is the time metric. A quadratic function of age is included to model the possible nonlinear pattern of health over the life course. $\sum_j C_{ji}$ are three cohort dummies. $\sum_k X_{ki}$ are a k set of time-constant variables including gender, race/ethnicity, and early-life factors. $\sum_p Z_{pit}$ are a p set of time-varying variables including educational attainment in adulthood, income-to-needs ratio, employment, disability statuses, and survey year. ϑ_{0i} and ϑ_{1i} are a random intercept and random slope, and ε_{it} is the error term. Each main early-life explanatory variable is estimated and presented separately in Tables 3, 4, 5, 6 and 7. The interaction terms between early-life factors and cohort dummies are added to this basic model setup to test the changing association between early-life factors and health outcomes across birth cohorts (models 2 and 5). The interaction terms between each early-life factor and age and period dummies are further included in models 3 and 6 to account for the possible confounding life-course and period patterns.

Table 3 Cohort pattern in the association between birthweight and health outcomes: unstandardized regression coefficients from multilevel growth models

	Self-rated health			Health summary index		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Age	0.025*** (0.001)	0.025*** (0.001)	0.015*** (0.004)	0.047*** (0.001)	0.047*** (0.001)	0.032*** (0.005)
Age squared	0.000*** (0.000)	0.000*** (0.000)	0.000*** (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.001*** (0.000)
Female	0.148*** (0.016)	0.147*** (0.016)	0.147*** (0.015)	0.140*** (0.019)	0.139*** (0.019)	0.141*** (0.018)
Black	0.304*** (0.016)	0.304*** (0.016)	0.213*** (0.016)	0.008 (0.020)	0.009 (0.020)	−0.038* (0.019)
Hispanic	0.099** (0.040)	0.100* (0.040)	0.050 (0.037)	−0.050 (0.050)	−0.051 (0.050)	−0.069 (0.048)
1960 cohort	−0.039 (0.021)	−0.209** (0.076)	−0.249** (0.075)	0.098** (0.033)	−0.253* (0.119)	−0.328** (0.119)
1970 cohort	0.038 (0.022)	0.007 (0.080)	0.049 (0.091)	0.317*** (0.033)	0.108 (0.108)	0.098 (0.125)
1980 cohort	0.090*** (0.024)	−0.067 (0.086)	−0.213 (0.113)	0.351*** (0.037)	0.066 (0.111)	−0.267 (0.148)
Birth weight	−0.048 (0.030)	−0.138* (0.055)	−0.104 (0.055)	−0.099** (0.036)	−0.350*** (0.095)	−0.295** (0.091)
×1960 cohort		0.183* (0.079)	0.175* (0.074)		0.380** (0.124)	0.314** (0.120)
×1970 cohort		0.034 (0.083)	0.004 (0.078)		0.228* (0.112)	0.165 (0.113)
×1980 cohort		0.170 (0.090)	0.177* (0.087)		0.309** (0.113)	0.265* (0.122)
x Age			0.000 (0.002)			−0.001 (0.004)
Highest grade completed			−0.060*** (0.003)			−0.019*** (0.004)
Adult income-to-needs			−0.008*** (0.001)			−0.006*** (0.001)
working			−0.091*** (0.008)			−0.085*** (0.010)
Disabled			0.671*** (0.022)			0.666*** (0.025)
Period dummies	No	No	Yes	No	No	Yes
Intercept	2.281	2.365	3.135	0.565	0.796	1.110
BIC	180,328.00	180,354.40	178,776.40	67,946.79	67,967.37	67,099.16
Number of groups	8342	8342	8342	6108	6108	6108
Sample size	80,018	80,018	80,018	33,054	33,054	33,054

*Significant at 5% level

**Significant at 1% level

***Significant at 0.1%

Table 4 Cohort pattern in the association between early-life disease index and health outcomes: unstandardized regression coefficients from multilevel growth models

	Self-rated health			Health summary index		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Age	0.025*** (0.001)	0.025*** (0.001)	0.014*** (0.002)	0.046*** (0.001)	0.046*** (0.001)	0.030*** (0.002)
Age squared	0.000*** (0.000)	0.000*** (0.000)	0.000*** (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.001*** (0.000)
Female	0.132*** (0.013)	0.133*** (0.013)	0.138*** (0.013)	0.090*** (0.012)	0.091*** (0.012)	0.089*** (0.012)
Black	0.333*** (0.014)	0.333*** (0.014)	0.235*** (0.014)	0.060*** (0.013)	0.059*** (0.013)	0.019 (0.013)
Hispanic	0.105** (0.033)	0.106** (0.033)	0.048 (0.031)	0.020 (0.030)	0.022 (0.030)	0.000 (0.029)
1960 cohort	−0.006 (0.019)	−0.014 (0.021)	−0.084** (0.028)	0.106*** (0.023)	0.090*** (0.025)	−0.032 (0.031)
1970 cohort	0.075*** (0.019)	0.057** (0.021)	−0.086 (0.048)	0.273*** (0.023)	0.225*** (0.025)	−0.015 (0.047)
1980 cohort	0.103*** (0.020)	0.099*** (0.023)	−0.137* (0.066)	0.281*** (0.026)	0.267*** (0.028)	−0.086 (0.064)
Early life disease index	0.167*** (0.007)	0.143*** (0.018)	0.140*** (0.016)	0.301*** (0.006)	0.227*** (0.023)	0.237*** (0.022)
×1960 cohort		0.023 (0.023)	0.009 (0.022)		0.051 (0.028)	0.084** (0.027)
×1970 cohort		0.040 (0.021)	0.023 (0.020)		0.112*** (0.025)	0.191*** (0.025)
×1980 cohort		0.018 (0.022)	0.021 (0.021)		0.058* (0.025)	0.195*** (0.027)
×Age			0.001 (0.001)			0.010*** (0.001)
Highest grade completed			−0.065*** (0.003)			−0.018*** (0.003)
Adult income-to-needs			−0.007*** (0.001)			−0.004*** (0.001)
working			−0.089*** (0.007)			−0.101*** (0.008)
Disabled			0.654*** (0.018)			0.642*** (0.019)
Period dummies	No	No	Yes	No	No	Yes
Intercept	2.090	2.098	2.958	0.327	0.353	0.696
BIC	234,261.80	234,292.60	231,935.80	120,182.90	120,185.90	118,465.70
Number of groups	11,352	11,352	11,352	11,338	11,338	11,338
Sample size	105,981	105,981	105,981	59,072	59,072	59,072

*Significant at 5% level

**Significant at 1% level

***Significant at 0.1%

Table 5 Cohort pattern in the association between mother's education and health outcomes: unstandardized regression coefficients from multilevel growth models

	Self-rated health			Health summary index		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Age	0.025*** (0.001)	0.025*** (0.001)	0.013*** (0.002)	0.045*** (0.001)	0.045*** (0.001)	0.032*** (0.003)
Age Squared	0.000*** (0.000)	0.000*** (0.000)	0.000*** (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.001*** (0.000)
Female	0.157*** (0.011)	0.157*** (0.011)	0.155*** (0.010)	0.125*** (0.013)	0.125*** (0.013)	0.121*** (0.013)
Black	0.258*** (0.012)	0.258*** (0.012)	0.192*** (0.011)	0.003 (0.015)	0.003 (0.015)	−0.032* (0.014)
Hispanic	0.097*** (0.026)	0.097*** (0.026)	0.056* (0.024)	0.011 (0.033)	0.010 (0.033)	−0.006 (0.032)
1960 cohort	−0.030* (0.014)	−0.063* (0.026)	−0.134*** (0.030)	0.104*** (0.023)	−0.032 (0.048)	−0.160** (0.052)
1970 cohort	0.085*** (0.016)	0.028 (0.031)	−0.132** (0.047)	0.326*** (0.024)	0.208*** (0.046)	−0.063 (0.066)
1980 cohort	0.132*** (0.018)	0.086* (0.039)	0.045** (0.066)	0.347*** (0.027)	0.204*** (0.050)	−0.185* (0.085)
Mother's education	−0.071*** (0.005)	−0.085*** (0.008)	−0.046*** (0.008)	−0.021*** (0.006)	−0.075*** (0.016)	−0.059*** (0.015)
×1960 cohort		0.017 (0.011)	0.020 (0.011)		0.066** (0.020)	0.051* (0.020)
×1970 cohort		0.026* (0.012)	0.036** (0.012)		0.057** (0.019)	0.037 (0.020)
×1980 cohort		0.021 (0.014)	0.045** (0.014)		0.066*** (0.019)	0.040 (0.022)
×Age			−0.001 (0.000)			−0.002** (0.001)
Highest grade completed			−0.065*** (0.002)			−0.020*** (0.003)
Adult income-to-needs			−0.007*** (0.001)			−0.005*** (0.001)
Working			−0.098*** (0.006)			−0.106*** (0.008)
Disabled			0.701*** (0.017)			0.642*** (0.020)
Period dummies	No	No	Yes	No	No	Yes
Intercept	2.366	2.392	3.159	0.496	0.607	0.963
BIC	317,208.40	317,238.30	314,031.70	119,899.20	119,918.00	118,393.80
Number of groups	17,209	17,209	17,209	12,031	12,031	12,031
Sample size	141,142	141,142	141,142	58,203	58,203	58,203

*Significant at 5% level

**Significant at 1% level

***Significant at 0.1%

Table 6 Cohort pattern in the association between childhood income-to-needs ratio and health outcomes: unstandardized regression coefficients from multilevel growth models

	Self-rated health			Health summary index		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Age	0.025*** (0.001)	0.025*** (0.001)	0.017*** (0.003)	0.049*** (0.001)	0.049*** (0.001)	0.037*** (0.004)
Age squared	0.000*** (0.000)	0.000*** (0.000)	0.000*** (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.001*** (0.000)
Female	0.146*** (0.015)	0.146*** (0.015)	0.147*** (0.014)	0.141*** (0.018)	0.140*** (0.018)	0.143*** (0.017)
Black	0.188*** (0.017)	0.168*** (0.017)	0.138*** (0.016)	−0.032 (0.020)	−0.041* (0.020)	−0.067** (0.020)
Hispanic	0.062 (0.037)	0.057 (0.037)	0.031 (0.035)	−0.030 (0.047)	−0.029 (0.047)	−0.047 (0.045)
1960 cohort	−0.045* (0.020)	−0.105** (0.033)	−0.151*** (0.038)	0.100** (0.033)	0.050 (0.056)	−0.056 (0.060)
1970 cohort	0.050* (0.021)	−0.064 (0.035)	−0.144* (0.060)	0.334*** (0.033)	0.284*** (0.052)	0.044 (0.078)
1980 cohort	0.101*** (0.022)	−0.077* (0.033)	−0.187* (0.080)	0.349*** (0.035)	0.244*** (0.052)	−0.082 (0.101)
Childhood income-to-needs	−0.054*** (0.004)	−0.102*** (0.009)	−0.069*** (0.009)	−0.016*** (0.004)	−0.046** (0.014)	−0.037** (0.013)
× 1960 cohort		0.029* (0.012)	0.031** (0.011)		0.020 (0.018)	0.007 (0.017)
× 1970 cohort		0.052*** (0.012)	0.049*** (0.011)		0.021 (0.016)	0.003 (0.016)
× 1980 cohort		0.074*** (0.010)	0.060*** (0.010)		0.040** (0.015)	0.008 (0.016)
× age			−0.000 (0.000)			−0.002*** (0.001)
Highest grade completed			−0.056*** (0.003)			−0.019*** (0.004)
Adult income-to-needs			−0.008*** (0.001)			−0.005*** (0.001)
Working			−0.092*** (0.007)			−0.094*** (0.010)
Disabled			0.668*** (0.021)			0.635*** (0.023)
Period dummies	No	No	Yes	No	No	Yes
Intercept	2.430	2.540	3.193	0.548	0.628	0.954
BIC	199,083.60	199,062.30	197,383.80	76,284.81	76,306.09	75,350.67
Number of groups	9427	9427	9427	6954	6954	6954
Sample size	87,565	87,565	87,565	36,762	36,762	36,762

*Significant at 5% level

**Significant at 1% level

***Significant at 0.1%

Table 7 Cohort pattern in the association between parental smoking before age 17 and health outcomes: unstandardized regression coefficients from multilevel growth models

	Self-rated health			Health summary index		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Age	0.025*** (0.001)	0.025*** (0.001)	0.009*** (0.002)	0.046*** (0.001)	0.046*** (0.001)	0.023*** (0.003)
Age squared	0.000*** (0.000)	0.000*** (0.000)	0.000*** (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.001*** (0.000)
Female	0.149*** (0.014)	0.150*** (0.014)	0.153*** (0.013)	0.123*** (0.014)	0.123*** (0.014)	0.120*** (0.013)
Black	0.314*** (0.014)	0.311*** (0.014)	0.216*** (0.014)	0.016 (0.015)	0.015 (0.015)	−0.027 (0.015)
Hispanic	0.102** (0.033)	0.104** (0.033)	0.045 (0.031)	0.002 (0.033)	0.003 (0.033)	−0.022 (0.032)
1960 cohort	0.005 (0.019)	−0.040 (0.032)	−0.149*** (0.036)	0.123*** (0.024)	0.055 (0.040)	−0.116*** (0.044)
1970 cohort	0.129*** (0.019)	0.052 (0.030)	−0.154** (0.053)	0.346*** (0.024)	0.267*** (0.037)	−0.072 (0.058)
1980 cohort	0.177*** (0.021)	0.088** (0.031)	−0.236** (0.070)	0.376*** (0.027)	0.325*** (0.039)	−0.165* (0.076)
Parental smoking Before 17	0.124*** (0.014)	0.033 (0.029)	0.039 (0.027)	0.077*** (0.014)	−0.015 (0.039)	0.008 (0.038)
× 1960 cohort		0.068 (0.040)	0.076* (0.037)		0.106* (0.049)	0.132** (0.048)
× 1970 cohort		0.123** (0.039)	0.106** (0.036)		0.129** (0.046)	0.180*** (0.046)
× 1980 cohort		0.158*** (0.040)	0.145*** (0.039)		0.074 (0.047)	0.163** (0.050)
× Age			0.003** (0.001)			0.009*** (0.002)
Highest grade completed			−0.064*** (0.003)			−0.021*** (0.003)
Adult income-to-needs			−0.007*** (0.001)			−0.005*** (0.001)
Working			−0.091*** (0.007)			−0.106*** (0.008)
Disabled			0.667*** (0.019)			0.643*** (0.019)
Period dummies	No	No	Yes	No	No	Yes
Intercept	2.066	2.126	2.939	0.383	0.442	0.827
BIC	235,483.90	235,500.80	233,098.70	122,696.30	122,720.50	121,104.90
Number of groups	11,405	11,405	11,405	11,393	11,393	11,393
Sample size	106,311	106,311	106,311	59,276	59,276	59,276

*Significant at 5% level

**Significant at 1% level

***Significant at 0.1%

Results

Because self-rated health and health summary index are collected from different waves of the surveys, we report the descriptive statistics of these two health outcomes separately. Because some variables are time varying, we report the descriptive statistics for the observations instead of individuals. Tables 1 and 2 present the statistics for the self-rated health sample. The average age of the sample is 35.44, but this varies across cohorts with the older cohorts having older average ages. Self-rated health has a mean of 2.23 out of 5 (the lowest score possible), and the 1950 birth cohort reports the poorest self-rated health with a mean of 2.31. In terms of trends in five early-life factors, the proportion of individuals with a birthweight of more than 5.5 lb does not significantly change across birth cohorts, but younger birth cohorts score higher on the early-life disease index (i.e., worse early life health), have mothers with higher educational attainment, have a higher family income-to-needs ratio in childhood, and have parents with lower smoking rates before age 17. More recent birth cohorts also have relatively higher educational attainment in adulthood but lower adult income-to-needs ratios; this may partially reflect their early career stage compared to older birth cohorts. Table 2 displays descriptive statistics for the health summary index sample. The mean age is 39.71, and the mean of the health summary index is 0.65. The 1950 cohort has the highest mean for the dependent variable at 0.98. Overall health improves across these four birth cohorts, but this may be partially due to age effects. With regard to the cohort pattern in the explanatory variables, it is similar to that in Table 1.

Early Life Health, Nutrition, and Disease

Table 3 displays the results of the association between birthweight and two health outcomes. Birthweight does not have a significant impact on self-rated health in Model 1 but appears significant when interacted with birth cohort dummies in Model 2. Individuals in the 1950s birth cohort with a birthweight of more than 5.5 lb report 0.138 units lower in poor self-rated health compared to those who weighed less at birth. Cohort interactions with birthweight are significant for the 1960s birth cohort in Model 2 and the 1960s and 1980s birth cohorts in Model 3, which adds the interaction between birthweight and age, adult SES, disability status, and survey-year dummies. These factors are controlled to ascertain that age, period patterns, and possible confounding factors in adulthood do not contribute to the changing association between birthweight and self-rated adult health across birth cohorts. The positive signs of these interactions indicate a weakening relationship between birthweight and self-rated health in the later birth cohorts. Models 4 through 6 in Table 3 present the impact of birthweight on the health summary index. After adjusting for confounded age effects, all later birth cohorts have more health problems than the 1950s birth cohort. Birthweight of more than 5.5 lb is associated with a 0.099 unit reduction in the number of health problems. Positive cohort interactions once again indicate a weakening relationship between birthweight and the health summary index across birth cohorts.

In Table 4, we test how the association between early-life disease and adult health may change across birth cohorts. The early-life disease index is significantly associated with 0.167 units higher in poor self-rated health and 0.301 units higher in the number of health conditions in adulthood. The association strengthens across birth cohorts as shown by the positive cohort interactions in Model 3 and Model 6, although they are only significant for the health summary index. The age interaction with the early-life disease index is also significant and positive, which indicates that its impact on adult health increases over the life course.

Parental SES and Health Behavior

Table 5 summarizes the results of mother's education on the two health outcomes. For both self-rated health and the health summary index, the main effect of mother's education is significantly negative for all six models, which implies mother's education is beneficial for adult children's health. In Model 2 on self-rated health, the 1970s birth cohort interaction with mother's education is significant, while in Model 3 both the 1970s and 1980s birth cohorts' interactions with mother's education are significant and positive. Since a mother's education is negatively associated with worse health outcomes, the positive interaction suggests the impact of mother's education weakens across cohorts. For the health summary index, all three cohorts' interactions with mother's education are positive and significant in Model 5, but in Model 6, which includes adult sociodemographic characteristics, the age interaction and survey-year dummies, only the 1960s cohort interaction is still statistically significant. The significant negative interaction between mother's education and age suggests the effect of mother's education on the health summary index strengthens over the life course.

Table 6 displays the cohort pattern in the link between childhood income-to-needs ratio and adult health. Each one-unit increase in childhood income-to-needs ratio is associated with a 0.054 unit reduction in poor self-rated health and a 0.016 unit reduction in the number of health conditions. Similar to mother's education, the impact of childhood income-to-needs ratio on adult self-rated health weakens across cohorts. For the health summary index, the impact of this childhood economic indicator is significantly weaker among the 1980s cohort compared to the 1950s cohort. In Model 6, a significantly negative interaction between this economic indicator and age suggests its impact on adult health increases over the life course.

Table 7 presents results for the link between parental smoking before age 17 and health across birth cohorts. Respondents who had at least one parent who smoked during their childhoods have 0.124 units higher in poor self-rated health than those whose parents did not smoke. Cohort interactions with parental smoking are significant for the 1970s and 1980s cohorts in Model 2 and all three cohorts in Model 3. The positive cohort interactions suggest that the association between parental smoking and self-rated health is stronger in later birth cohorts. For the health summary index, Model 4 shows parental smoking is associated with a 0.077 unit increase in the number of health problems. In Model 6, cohort interactions for all three cohorts are positive and significant, which suggests the association between

parental smoking and the health summary index strengthens across birth cohorts. The age interactions with parental smoking in models 3 and 6 are positive and significant, which suggests that the harmful impact of parental smoking on adult health strengthens over the life course.

In order to more visually portray the changing effects of these five early-life factors on adult health across birth cohorts, Fig. 1 demonstrates how the predicted value of self-rated health and health summary index may change across birth cohorts. In order to reduce fluctuations in the cohort trend, we treat cohort as a continuous variable and interact it with early-life factors. The predicted values of health outcomes are based on models adjusted for adulthood SES, confounding age, and period patterns like models 3 and 6 in the tables. Self-rated health is on the left, while health summary index on the right. Each row represents one early life factor. For each early life factor, we choose two levels (e.g., mother without high school degree vs. mother with college degree). The widening gap in predicted outcomes between two levels for each life factor would indicate that the effect of this early factor on adult health outcomes increases across birth cohorts. A narrowing gap would indicate a decreasing effect across cohorts. It is clear that the effects of birthweight, mother's education, and childhood family income-to-needs ratio decrease across birth cohorts, while the effects of the early-life disease index and parental smoking before age 17 increase across cohorts.

Discussion and Conclusion

This study investigates the changing effect of early-life conditions on later-life health across the 1950s to 1980s birth cohorts in the United States. We find divergent patterns by specific early-life factors. The association between birthweight and two adult health outcomes (self-rated health and health summary index) weakens in later birth cohorts. So does mother's education and childhood family income-to-needs ratio. But the association strengthens for early-life disease index and parental smoking status. These findings suggest that cohort effect is multidimensional, and whether cohort effect weakens or strengthens depends on which dimensions of early-life are considered. Prior literature that focuses on disentangling the average effects of period and cohort may miss the multidimensionality characteristics of the cohort effect.

The question is why the effects of some early-life factors strengthen while others weaken. It seems they are not simply related to the temporal trends in the prevalence or levels of these factors. For example, lower prevalence of parental smoking before children turning age 17 over time does not reduce its impact on offspring health; improving mother's educational attainment and family income-to-needs ratio is not translated into increasing impact on offspring health across cohorts. Proportion of individuals with birthweights of more than 5.5 lb does not significantly change across birth cohorts, but the association between birth weight and adult health weakens across cohorts. The only exception here is that the increasing prevalence of early-life diseases is accompanied by its increasing importance on adult health.

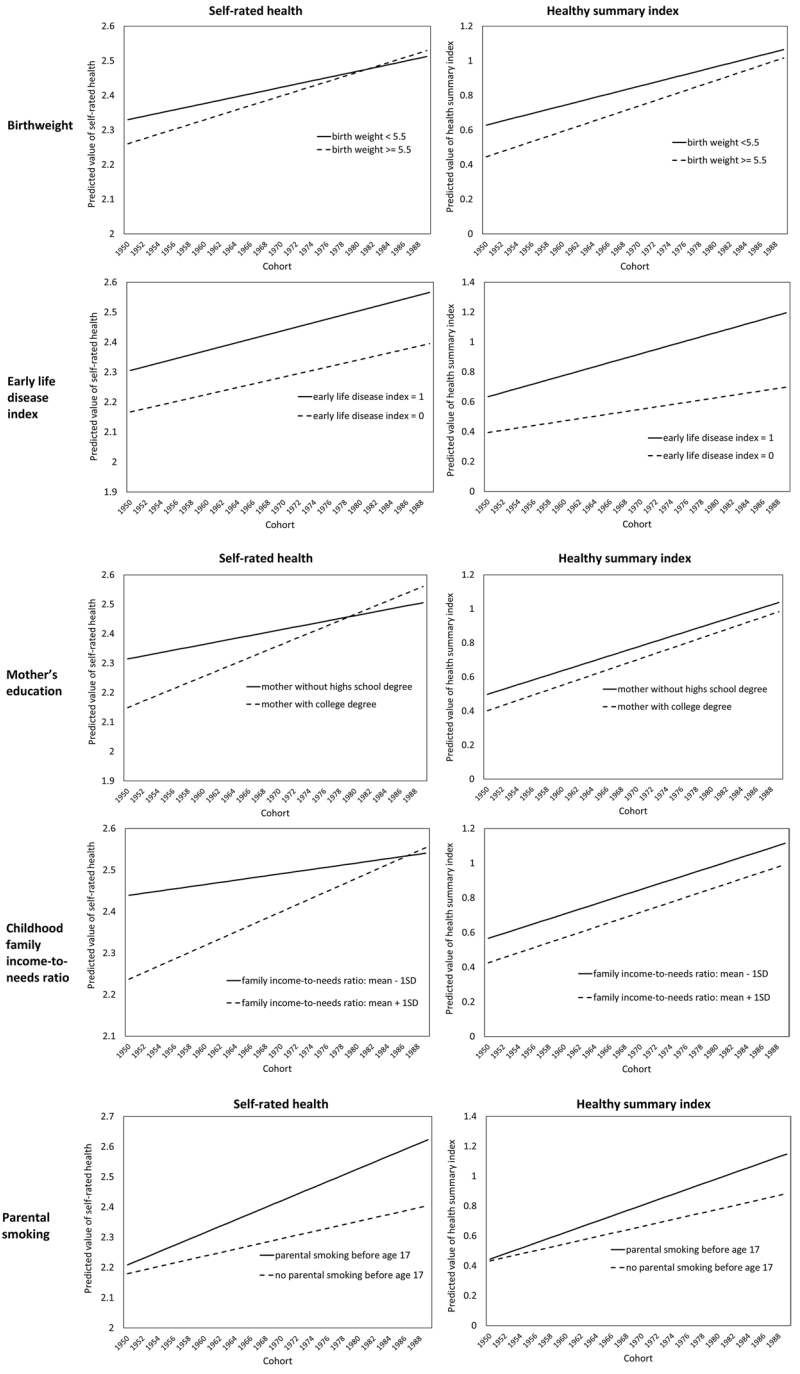

Fig. 1 Trends in self-rated health and health summary index across cohorts by early-life factors

These findings reveal the complexity of the social-to-biological embodiment across the life course, and underline the importance of understanding the effects of early-life factors within historical contexts. Macro social, economic, policy, technological, and medical changes can alter the impact of early-life conditions on adult health. On the one hand, as more medical care advancements improve the survival and health of preterm and low-birthweight infants (e.g., Goldenberg and Culhane 2007; Phibbs et al. 2007), they may continue to experience better health and lower risk of mortality and morbidity in adulthood. Thus, the detrimental impact of low birthweight on adult health declines across cohorts. We do not find support for the argument that increasing survival of low-birthweight babies due to medical advances would amplify the negative consequence of low birthweight on adult health, consistent with Hack et al. (1995).

On the other hand, children are increasingly experiencing a variety of diseases (e.g., asthma, allergy, and cancer) and health risk factors (e.g., obesity) (e.g., Beggs and Bambrick 2005; Charpin and Gouitaa 2001; Schmidt 1998; Wang and Lim 2012). Obesogenic environment and sedentary lifestyle are the major causes for the rising obesity prevalence. Tobacco, food additives, certain medications, viral infections, and industrial and agricultural chemicals contribute to the rise in cancer incidence rates among children (Schmidt 1998). Atmospheric carbon dioxide concentration may be the cause for the growth of respiratory diseases (Beggs and Bambrick 2005). These worsening health profiles in childhood strengthen the link to later health across cohorts.

The impact of family socioeconomic background on adult children's health has generally weakened for cohorts born in the second half of the twentieth century partially due to many government programs to offset early-life SES disadvantages. As more government programs to provide childcare assistance and prevent behavioral and social problems were established (Crosby et al. 2005; Gennetian et al. 2004; Bloom and Michalopoulos 2001), the impact of low SES status during childhood on children's health has decreased across cohorts, which further weakens its impact on their health when they turn into adults. Whether these programs also weaken the link between family background and adult SES is a bit mixed as shown in Appendix Tables 8 and 9. On the one hand, mother's education becomes increasingly important for adult children's education attainment, and family income becomes increasingly important for adult children's income, consistent with Chetty et al. (2014). On the other hand, the impact of family income on adult children's educational attainment does weaken across cohorts. Therefore, the weakening link between family SES background and adult children's health is probably only minimally mediated through adult children's SES. It is mostly due to the weakening link between family SES and childhood health as a result of those government programs. The increasing importance of attained status (e.g., education, income and occupation) on adult health may also further dilute the impact of ascribed status (e.g., family socioeconomic background) (e.g., Meara et al. 2008; Olshansky et al. 2012; Pappas et al. 1993).

But one aspect of family background has been increasingly more important for adult children's health, that is whether parents are smokers. Even though the number of smokers has steadily declined in the past several decades, tobacco companies have

Table 8 Cohort pattern in the association between mother's education and adult ses outcomes: unstandardized regression coefficients from multilevel growth models

	Adult education	Adult income
	Model 1	Model 2
Age	0.027***	0.102***
	(0.001)	(0.003)
Age squared	−0.000***	0.000***
	(0.000)	(0.000)
Female	0.231***	−0.131***
	(0.029)	(0.032)
Black	−0.762***	−1.342***
	(0.030)	(0.035)
Hispanic	−0.712***	−0.549***
	(0.066)	(0.075)
1960 cohort	0.175***	−0.116
	(0.042)	(0.077)
1970 cohort	0.420***	−0.205*
	(0.051)	(0.096)
1980 cohort	0.076	−0.469***
	(0.074)	(0.111)
Mother's education	0.083***	0.291***
	(0.006)	(0.022)
×1960 cohort	−0.018	0.033
	(0.010)	(0.033)
×1970 cohort	0.075***	0.047
	(0.013)	(0.038)
×1980 cohort	0.332***	0.063
	(0.024)	(0.041)
Intercept	13.219	4.643
BIC	282,190.5	770,553.7
Number of groups	17,782	17,782
Sample size	163,930	163,930

*Significant at 5% level

**Significant at 1% level

***Significant at 0.1%

used new chemical fillers to increase tobacco addiction (e.g., Keithly et al. 2005; Rabinoff et al. 2007; Stepanov et al. 2011). These strong chemical components in cigarettes have contributed to worse health outcomes among smokers and put their family members at a higher risk of diseases related to exposure to tobacco (e.g., Edwards et al. 2017; Thun et al. 2013). This is probably one of the reasons why the impact of parental smoking on the health of offspring has increased in more recent cohorts. Another possible explanation is that smoking parents increasingly become a selected group with both material and cultural disadvantages. They may become more concentrated at the bottom of social stratification (e.g., income, wealth, education, occupation) and have worse lifestyles and health behaviors, which means that having a smoking parent may become an indicator of more adverse childhood

Table 9 Cohort pattern in the association between childhood income-to-needs ratio and adult ses outcomes: unstandardized regression coefficients from multilevel growth models

	Adult education Model 1	Adult income Model 2
Age	0.031*** (0.001)	0.095*** (0.003)
Age squared	−0.000*** (0.000)	−0.000 (0.000)
Female	0.212*** (0.037)	−0.219*** (0.036)
Black	−0.202*** (0.042)	−0.872*** (0.042)
Hispanic	−0.383*** (0.091)	−0.298** (0.089)
1960 cohort	0.009 (0.084)	−0.236** (0.079)
1970 cohort	0.613*** (0.088)	−0.432*** (0.087)
1980 cohort	1.303*** (0.079)	−0.032 (0.079)
Childhood Income-to-needs	0.483*** (0.022)	0.287*** (0.020)
×1960 cohort	0.017 (0.031)	0.104*** (0.029)
×1970 cohort	−0.061* (0.030)	0.145*** (0.029)
×1980 cohort	−0.184*** (0.025)	−0.060* (0.025)
Intercept	12.093	4.242
BIC	181,677.9	470,694.4
Number of groups	9590	9590
Sample size	101,809	101,809

*Significant at 5% level

**Significant at 1% level

***Significant at 0.1%

circumstances in recent cohorts. Even though the impacts of mother's education and family income on adult children's health have declined across cohorts, increasing selection into smoking group from these disadvantaged family backgrounds can amplify the impact of parental smoking on offspring health. To account for the influences of these compositional changes, we control for mother's education and family income in the model of parental smoking as shown in Appendix Table 10. The sample size decreases by over 40%, so we need to interpret these results with cautions. These two indicators of family SES background explain to some extent the increasing effect of parental smoking on adult children's self-rated health, but not much on their health summary index. Therefore, increasing selection into smoking

Table 10 Cohort pattern in the association between parental smoking before age 17 and health outcomes controlling for mother's education and family income-to-needs ratio: unstandardized regression coefficients from multilevel growth models

	Self-Rated Health		Health Summary Index	
	Model 1	Model 2	Model 3	Model 4
Age	0.022***	0.022***	0.041***	0.041***
	(0.001)	(0.001)	(0.002)	(0.002)
Age squared	0.000	0.000	0.001***	0.001***
	(0.000)	(0.000)	(0.000)	(0.000)
Female	0.169***	0.164***	0.149***	0.148***
	(0.017)	(0.017)	(0.018)	(0.018)
Black	0.216***	0.172***	−0.039*	−0.044*
	(0.018)	(0.020)	(0.019)	(0.021)
Hispanic	0.046	0.029	−0.041	−0.045
	(0.047)	(0.047)	(0.048)	(0.048)
1960 cohort	−0.061	−0.052	0.018	0.019
	(0.041)	(0.041)	(0.056)	(0.056)
1970 cohort	0.061	0.088*	0.177**	0.263
	(0.039)	(0.040)	(0.066)	(0.054)
1980 cohort	0.107**	0.153***	0.285***	0.292*
	(0.039)	(0.040)	(0.055)	(0.056)
Parental smoking before 17	0.019	0.019	−0.015	−0.016
	(0.039)	(0.039)	(0.056)	(0.056)
×1960 cohort	0.089	0.085	0.147*	0.146*
	(0.051)	(0.051)	(0.070)	(0.070)
×1970 cohort	0.102*	0.087	0.177**	0.175**
	(0.050)	(0.050)	(0.066)	(0.066)
×1980 cohort	0.141**	0.110*	0.172*	0.168*
	(0.052)	(0.052)	(0.069)	(0.069)
×Age	0.002	0.002	0.009***	0.009***
	(0.001)	(0.001)	(0.002)	(0.002)
Highest grade completed	−0.052***	−0.045***	−0.018***	−0.017***
	(0.003)	(0.004)	(0.004)	(0.004)
Adult income-to-needs	−0.008***	−0.007***	−0.005***	−0.005***
	(0.001)	(0.001)	(0.001)	(0.001)
Working	−0.086***	−0.085***	−0.089***	−0.089***
	(0.008)	(0.008)	(0.010)	(0.010)
Disabled	0.672***	0.672***	0.626***	0.626***
	(0.025)	(0.025)	(0.025)	(0.025)
Mother's education		−0.036***		−0.007
		(0.007)		(0.009)
Childhood income-to-needs		−0.017***		−0.001
		(0.005)		(0.005)
Intercept	2.982	3.007	0.793	0.798
BIC	137,003.1	136,975.7	67,233.68	67,253.55
Number of groups	6319	6319	6145	6145
Sample size	62,147	62,147	33,306	33,306

*Significant at 5% level

**Significant at 1% level

***Significant at 0.1%

group from lower SES background may explain a modest amount. But other more adverse childhood circumstances (e.g., worse parental health, lifestyles and health behaviors) associated with this selection process can still impact the health status of children, either during birth or after birth, and lead to the increasing effect of parental smoking on adult children's health.

We further conduct several sensitivity and group-specific analyses. We try different categorizations of cohorts (e.g., Baby Boomers, early-Generation Xers, late-Generation Xers, and early-Generation Yers), which do not substantially change the main results. Separate analyses are conducted for black and white respondents. The main patterns do not differ by black and white. The Hispanic subsample is too small for a separate analysis. We also further break down analysis by gender and do not find substantial gender differences.

This study has several limitations. First, the age ranges across the four birth cohorts are not balanced. The 1980s birth cohort is still relatively young and may not be old enough to experience serious health problems. We do include the interaction between age and explanatory variables to ascertain the cohort pattern is not driven or confounded by the life-course pattern. In some situations, after controlling for life-course pattern, cohort pattern actually becomes even more salient. This is consistent with Lynch (2003), which finds each pattern is suppressed when the other is ignored. In additional analysis not shown here, we restricted the age of respondents to 25–45 years old in order to increase the extent of age overlap across cohorts. The results in this restricted sample do not differ from the main results (tables available upon request). Therefore, even though the age ranges across birth cohorts are not balanced, this issue should not have driven the cohort patterns found in this research. Second, we only have a limited number of cohorts and early-life factors, but we are not aware of a better dataset. Future studies should further test other early-life factors when data become available. Third, we attempt to understand the heterogeneity in the changing impacts of early-life factors in the macro social, medical, technological, and policy context, and provide some possible explanations. We are not able to directly test these explanations with the current data, which, however, is beyond the scope of this paper. Future research can further employ other appropriate data to test the robustness of the findings and test the potential mechanisms contributing to these findings.

This study produces some novel results on the changing effect of early-life conditions on adult health across birth cohorts. Our findings suggest that the effects of nutrition and family SES on adult health weaken, while the effects of diseases and disease risk factors (e.g., smoking) strengthen across birth cohorts. Therefore, while some dimensions of early-life factors take less of a toll on health, with the reemergence of infectious diseases and rising obesity, other dimensions of early-life conditions will pose long-term health consequences for the young and future birth cohorts. The steadily declining smoking rate in the past several decades is regarded as a success story among U.S. health scientists and policy makers. However, its negative impact on the offspring of smokers has steadily strengthened. Future research should extend these analyses and reveal the origins of health across birth cohorts. With more evidence-based studies, social and health scientists can gain a better understanding about the mechanism of health and how it changes across birth

cohorts. This insight is important for healthcare providers and policy makers seeking to develop age-appropriate strategies and policies to improve health.

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Appendix

See Tables 8, 9 and 10.

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