Sex and Race Disparities in Health: Cohort Variations in Life Course Patterns

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This study assesses changes in sex and race disparities in health over the life course and across cohorts by conducting growth curve analyses of nationally representative longitudinal data that spans 15 years. It finds that changes in disparities in depressive symptoms, disability and self-assessments of health across the life course are cohort-related phenomena: (1. there is significant inter-cohort heterogeneity in health trajectories; (2. intra-cohort sex and race inequalities exist in levels of health but not in growth rates of all health problems; (3. there are inter-cohort variations in the intra-cohort heterogeneity – sex and race gaps change across cohorts in levels of health. Changes in the sex gap in growth rates of depression are also strongly contingent upon cohort membership.

The process by which health deteriorates or is maintained as individuals age and its relationship to demographic statuses such as sex and race have attracted great attention in recent social and epidemiologic research on aging and the life course. However, our understanding of the links between social factors and health has been limited by a neglect of the cohort succession process. Most previous studies hinge on the cross-sectional relationship between age and health that confound aging and cohort effects. Findings have also been inconsistent regarding whether social disparities in health grow or diminish over the life course. The essential assumption of previous studies that omit cohort analysis is that sex and race differences in age patterns of health observed from cross-sectional data represent true intra-individual developmental trajectories of change over time that are equal across various birth cohorts. This assumption may not be tenable in light of the social changes and vastly different historical and life experiences of birth cohorts during the 20th century that bear important consequences for cohort differences in mental and physical health. The question of whether changes in social attainment, health capital and the roles of women and blacks have made successive birth cohorts less susceptible to sex and race inequalities in health merits further study.

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This study aims to advance sociological understanding of the mechanisms generating sex and race inequalities in health over the life course by systematically examining inter-cohort and intra-cohort variations in health trajectories. It uses a four-wave nationally representative longitudinal survey of adults 25 years and older and employs growth curve models to effectively disentangle the aging and cohort effects. The central question we address is how taking cohort effects into account modifies previous understandings of changes in sex and race inequalities in health over the life course. Specifically, we examine three related questions: (1. Are there inter-cohort variations in health trajectories over the life course? (2. Do sex and race disparities in health change over the life course within cohorts? (3. Are there also inter-cohort differences in the patterns of intra-cohort sex and race disparities in health trajectories? An assessment of the distinct roles of aging and cohort effects in the study of social inequalities in health may provide a better understanding of biological and social structural factors that condition health inequalities over the life course and help researchers better evaluate the explanatory power of competing theories. The identification of cohort-specific patterns of inequalities in psychiatric morbidity and physical functioning may also have important implications for public health and finance as the largest cohort in U.S. history – the Baby Boomers – reaches age 65 after 2010.

Theoretical Hypotheses

Inter-cohort Variations over the Life Course

Life course research on health has long concerned itself with age patterns because age has been shown to be among the most important sources of variations in vital rates and has frequently been used to understand the etiology of diseases (Hobcraft, Menken and Preston 1982). However, there has been insufficient attention to birth cohort variations in examinations of health changes over the life course. We understand much less about the way health trajectories change with cohorts. Most prior studies are also cross-sectional and cannot be used to infer intra-cohort health change with age. And most extant longitudinal studies have not successfully disentangled age and cohort effects, due to limitations in data or modeling strategies (Yang 2007). However, it is essential to distinguish aging and cohort effects both conceptually and analytically. Age effects reflect processes of aging internal to individuals and represent developmental changes over the life course. Cohort effects reflect social change and subsume the effects of early life conditions and the continuous accumulation of exposures to biological and social risk factors for health throughout the life course (Ryder 1965). By ignoring cohort effects in the analysis of life course patterns of health, one implicitly assumes that the rates at which health changes

with age are equally applicable across all cohorts. This assumption greatly simplifies analysis and may be reasonable from the perspective that there is substantial continuity across adjacent cohorts. However, it must be subject to tests in light of the many social and historical forces that have produced significant cohort differences in aging experiences (Uhlenberg and Riley 1996).

> Hypothesis 1 – Inter-cohort change: There are substantial inter-cohort variations in health trajectories over the life course, with more recent cohorts having higher levels of health and lower growth rates in health problems with age.

The rationale for this hypothesis begins with the observation that social demographic and historical changes may have had profound and enduring effects on cohorts' health. It has been argued that throughout the 20th century, individuals' health capital – physiological robustness and capacity of vital organ systems – has improved with the year of birth, with more recent cohorts faring substantially better in their initial endowments at birth and having lower depreciation rates in that stock of health capital (Fogel 2004). Improved physiological capacities in later cohorts also bode well for the effectiveness of medical treatments. In fact, recent demographic research shows that better nutrition and reduced inflammatory infection in utero and during infancy have reduced the risks for major chronic diseases in adulthood (Barker 1998) and led to less severe disabilities for successive birth cohorts (Crimmins, Reynolds and Saito 1999). Improvements in physical functioning in more recent cohorts are also likely given evidence of continuous cohort improvements in education, general health, quality of life and smoking cessation (Haug and Folmar 1986; Hughes and O'Rand 2004; Pampel 2005). In contrast, early life experiences such as childhood poverty and traumas associated with the Great Depression and world wars have been shown to negatively affect the mental and physical health of earlier cohorts (Elder 1974; O'Rand and Hamil-Leuker 2005).

On the other hand, it is possible that the health improvements across cohorts are constrained to some extent by economic, cultural and lifestyle changes. For example, there have been substantial changes in family structures such as decreases in marriages and increases in divorces in more recent cohorts (Popenoe 1993; Waite 1995). The larger size of the baby boom cohorts has also increased labor market competition and life stress during early adulthood that persists into later ages (Easterlin 1987). Additionally, there have been evident increases in lifestyles harmful to cardiovascular health and increases in obesity in later born cohorts (Cabrera et al. 2003; Flegal et al. 2002). All these risk factors may have dampened

the positive cohort effects on health. Studies of depressive symptoms show evidence of more depression in War Babies (1935-45) than in earlier cohorts (Kasen et al. 2003; Yang 2007). Earlier cohorts (1900-1905 and pre-1900 cohorts) have also been found to have better perceived or self-rated health in late life than their successors (1906-1917 cohort) (Idler 1993). Because there are both positive and deleterious forces affecting the life courses of more recent cohorts, an empirical question is whether the cohort trend of improving health can be generalized to the entire adult life course and to cohorts born after WWII such as the Baby Boomers.

Sex and Race Disparities in Health Trajectories

Previous social and epidemiologic research has found that sex and race manifest strong relationships with health and the way health changes over the life course. Whereas the female and black disadvantage in health is a general observation, the patterns of sex and race disparities in health trajectories are much less clear. Although most extant studies suggest that the salience of sex and race in affecting health varies across age, they have reported different directions for such variations. A key factor that has led to the inconsistent understanding about aging and stratification patterns is the same one that plagues studies of health trajectories in the overall population – the confounding of aging and cohort effects. It remains unknown as to whether discrepant findings on changes in sex and race gaps in health with age are actually due to birth cohort differences because no prior studies have simultaneously examined and effectively distinguished aging and cohort effects using longitudinal research designs. We argue that taking cohort effects into account can largely resolve the inconsistencies in previous findings and can provide a better test of competing theories, whereas ignoring cohort variations may bias the findings on age-related inequalities. In light of the *inter-cohort change hypothesis*, we further test the *intra-cohort inequality hypothesis* that sex and race disparities in health change significantly over the life course, independent of cohort differences. We directly test the implicit assumption on which prior studies are based, that is, health inequalities by sex and race change over the life course in the same way within each cohort and the intra-cohort patterns reflect aging-related phenomena that are universal across all cohorts.

Previous studies suggest two opposing directions of change in sex and race gaps in health with age. Some found that these gaps widen with age (Mirowsky 1996; Maddox and Clark 1992). Others found that these gaps converge with age (George 1992; Ross and Bird 1994), and the racial gap even exhibits a crossover in old age (Johnson 2000; Land et al. 1994). The dominant explanation for increasing health disparities with age is

the cumulative advantage/disadvantage theory. Originating from studies of scientific careers and status attainment models (Merton 1968), the cumulative advantage theory has recently proliferated in life course studies of changes in the effects of personal and structural characteristics on various outcomes. The theory suggests that the effects of early advantage or disadvantage accumulate over the life course, thereby increasing heterogeneity within cohorts (O'Rand 2003). In the context of aging and health, it predicts that inequalities in social status and consequently in health status early in life amplify with age and differentiate individuals further as they age. Because this perspective emphasizes intra-cohort differentiation, a proper test should control for cohort differences that might otherwise be confounded with aging effects.

> Hypothesis 2a: Sex and race disparities in health increase over the life course within cohorts, suggesting positive effects of sex and race on growth rates of health problems and diverging health trajectories, net of cohort effects.

The prevailing explanation for decreasing health disparities with age is the age-as-leveler theory. It suggests that the health gaps narrow across the life course due to the equalization of resources in later life and the selective survival of elite minorities who have acquired immunity against hardships in life (House et al. 1994; Preston, Hill and Drevenstedt 1998). Accordingly, we have:

> Hypothesis 2b: Sex and race disparities in health decrease over the life course within cohorts, suggesting negative effects of sex and race on growth rates of health problems and converging health trajectories, net of cohort effects.

Most extant cross-sectional or short-term longitudinal analyses of sex and race differences in health are concerned with individual level as opposed to aggregate data, but these analyses provide no consistent support for either perspective (Ferraro and Farmer 1996; Kelley-Moore and Ferraro 2004). A secondary explanation for the diverging health gaps with age has been the double jeopardy hypothesis that old age and racial/ethnic minority status interact to widen black and white health differences, and it has also been extended to a triple jeopardy hypothesis that considers the compounding effects of sexism (Ferraro and Farmer 1996). Several recent studies more rigorously examined how sex and race effects on health change over the life course using better statistical models of age

interactions with sex and race (Ferraro and Farmer 1996; Kelley-Moore and Ferraro 2004; Mendes de Leon et al. 2005). They show no apparent increases or decreases in sex or race gaps and hence no support for the cumulative advantage, the double/triple jeopardy or the age-as-leveler theory when other social factors are controlled.

One major limitation of these studies is that they do not test for cohort effects. A recent longitudinal investigation of SES (especially education) gaps in health is improved in this regard since it adjusts for cohort effects in growth curve models (Lynch 2003). The study finds evidence for increasing education effects with age within cohorts net of inter-cohort differences, thus supporting the cumulative advantage theory. Like education, sex and race are also important aspects of the stratification system and thus provide similar information about the social structural contexts within which health is protected or put at risk. As such, sex and race effects may also follow the same pattern. As ascribed demographic characteristics, however, sex and race are more distal factors that exert their influences earlier than education or income do, and they have a more pervasive impact on an individual's life chances. Therefore, it is also possible that this finding does not apply to changes in sex and race inequalities.

Another limitation of extant studies is their focus on narrow age ranges. The fact that sex and racial inequalities in health are present early on and throughout the adult life course suggests the importance of examining the entire adult age range, not just middle or older ages. Including all ages allows observation of the life course stages during which health inequalities are most substantial. Furthermore, most studies are confined to physical health. It is not clear whether the patterns hold for mental health which is subject to different risk factors. In addition, not all studies account for selection effects. Findings based on surviving survey respondents who tend to be healthier than those lost to follow-up due to death or nonresponse may downwardly bias the estimated health disparities.

Inter-cohort Variations in Sex and Race Disparities in Health Trajectories

The inter-cohort change hypothesis refers to changes in overall cohort means, and the intra-cohort inequality hypothesis refers to whether sex and race groups within cohorts become increasingly heterogeneous with age. It is logical to further ask how the patterns of within cohort heterogeneity differ across cohorts.

A large body of sociological research has shown that sex and race inequalities in health largely result from differences in power, prestige, social status, socially learned lifestyles, behaviors, roles and stress (Verbrugge 1989; Williams 2005). The secular trends of these risk factors may not have occurred in a parallel manner across male and female cohorts,

or across black and white cohorts. Changes in sex- and race-specific exposures to these factors across birth cohorts may thus contribute to inter-cohort variations in intra-cohort sex and race disparities in health trajectories over the life course. Cohort membership, therefore, provides an important social structural context that conditions the cumulative advantage/disadvantage or leveling process. The inter-cohort difference in intra-cohort inequality hypothesis states that patterns of change in sex and race disparities in health trajectories vary by cohort.

Women and blacks who came of age during and after the women's and civil rights movements of the 1960s may have experienced more favorable changes in social conditions affecting health. More recent cohorts of women and blacks have greater improvements in education than men and whites do (Hughes and O'Rand 2004). There are also large cohort differences in gender role expectations, with more recent cohorts having less restrictive and more liberal attitudes toward women's non-domestic roles (Brooks and Bolzendahl 2004). Increases in female employment differentiate cohorts of women with respect to socioeconomic achievement and family structure (Spain and Bianchi 1996). Women of the Baby Boomer cohort, for example, have similar labor force participation patterns with men and have better mental health and fewer depressive symptoms than their predecessors (Jackson 2004). One study also suggests that, in more recent Swedish cohorts, there is a decreasing sex gap in morbidity and cardiovascular disease risk factors due to increasing blood pressure, weight and body mass in male cohorts and no significant increases in female cohorts (Cabrera et al. 2003). Studies have also found diverse patterns of smoking across cohorts and the presence of sex differences in cigarette use. Earlier male cohorts smoked cigarettes in larger numbers than female cohorts, but the sex gap narrowed with the diffusion of smoking in later female cohorts and increases in smoking cessations in more recent cohorts for both sexes (Pampel 2005). If sex and race gaps in major risk factors for health have decreased in more recent cohorts, then the corresponding health gaps may diminish. This suggests:

> Hypothesis 3a: Sex and race gaps in levels of health narrow in more recent cohorts.

On the other hand, a study suggests increasing black-white differentials in functional health, with more recent cohorts of elderly people having wider gaps than earlier cohorts (Gibson 1994). If risk factors for women and blacks in successive cohorts have increased, then there would be larger health gaps across cohorts. This suggests:

> Hypothesis 3b: Sex and race gaps in levels of health widen in more recent cohorts.

Recent studies of educational health disparities further indicate that the trends of increasing education effects over the life course (or larger educational gaps in older ages) have strengthened in more recent cohorts (House, Lantz and Herd 2005; Lynch 2003), suggesting a stronger cumulative advantage process at work for more recent cohorts. We, therefore, test:

> Hypothesis 3c: Sex and race gaps in growth rates of health problems change across cohorts: the degree of divergence (Hypothesis 2a) or convergence with age (Hypothesis 2b) changes in more recent cohorts

Extant evidence from longitudinal data is scant with regard to specific patterns of cohort changes in sex and race disparities in health. We need a better understanding of the nature and social context of sex and race health inequalities through additional studies that include multiple birth cohorts, multiple follow-up measures spanning longer time intervals and different health outcomes. We also need a simultaneous assessment of the aging and cohort effects to appropriately test competing hypotheses about the distinct roles of age and cohort in moderating social inequalities and to help resolve inconsistencies in previous findings. The present study addresses this gap by systematically examining inter-cohort and intra-cohort variations in sex and race disparities in depression, physical disability, and self-assessments of health over the adult life course using longitudinal growth curve analyses. The analyses adjust for the effects of major social and health risk factors that have been shown to correlate with depression, physical disability and self-rated health – including SES (education and family income) (House et al. 1994), marital status (George 1996), health statuses such as chronic conditions and body weight (Cabrera et al. 2003), and the health behavior of smoking (Das 2003). Because we hypothesize that there are cohort variations in these risk factors, we expect that accounting for these factors partially explains the cohort, sex and race effects on health trajectories. Studies of comorbidity suggest a positive association between depression and physical illness (Yang 2006). Accordingly, we also include depression, disability and self-rated health as covariates in the analysis of each outcome.

Data and Methods

The data are from the Americans' Changing Lives Study, a long-term nationally representative longitudinal survey of the adult non-institutionalized population (House et al. 2005). The ACL study uses an accelerated longitudinal design wherein an initial sample of 3,617 individuals from a broad

array of ages (25 and older), and thus of multiple birth cohorts (before 1905 to 1964), were interviewed in 1986 and monitored with three follow-up surveys in 1989, 1994 and 2001/2002. This design is especially useful for testing aging- and cohort-related hypotheses because it allows (1. the distinction between intra-individual change with age within cohorts and inter-cohort differences, and (2. a more rapid accumulation of information on age and cohort changes than a single cohort study.

Measures

There are three dependent variables. *Depressive symptoms* are measured by a standardized index created by the ACL study from an 11-item Center for Epidemiologic Studies depression scale of self-reported symptoms of depression such as feeling depressed, lonely, sad, etc. – the reliability and validity of which have been well established (Radloff 1977). *Physical disability* is indicated by level of difficulty in performing Activities of Daily Living and of Instrumental ADL such as being confined to bed or a chair and climbing a few flights of stairs. A summary index was created by the ACL to indicate an increasing level of functional disability, with 1, 2, 3 and 4 equal to no, least severe, moderately severe and most severe functional impairment, respectively. *Self-rated health* is measured by a scale indicating perception of general health, with 1 = poor, 2 = fair, 3 = good, 4 = very good and 5 = excellent.

Age at baseline interview ranges from 25 to 95+.[1] Using age and the baseline year of 1986, we grouped respondents into seven 10-year *birth cohorts*. This operationalization of cohort grouping is conventional in demographic analysis and distinguishes cohorts in a way that is qualitatively meaningful: cohorts 0–6 refer to those born before 1905, Young Progressives (1905-14), Jazz Age Babies (1915-24), Depression Kids (1925-34), War Babies (1935-44), and Baby Boomers (1945-54, 1955-64), respectively (Hughes and O'Rand 2004; Yang 2007).[2] At each follow-up, surviving respondents in each cohort aged together, yielding cohort-specific age trajectories.

The other key variables include respondent's *sex* (1 = female; 0 = male) and *race* (1 = black; 0 = white).[3] Control variables include *years of education* and *family income* in 2001 dollars, *marital status* (1 = not married or those who are divorced, separated, widowed or never married; 0 = married), number of *chronic illnesses* (arthritis, lung disease, hypertension, heart attack, diabetes, cancer, foot problems, stroke, broken bones and urine beyond control), Quetelet *Body Mass Index* and *smoking* (the number of cigarettes one usually smokes in a day). Based on medical guidelines, we recoded the continuous BMI score into categories of underweight (< 18.5), overweight ($25 < 30$), and obese (30+), with normal ($18.5 < 25$) being the

reference group. Respondent's birth cohort membership, sex, race, and education are time-constant covariates. All other variables are time-varying covariates with measurements at four interviews.

Samples

The sample consists of all black and white respondents at the baseline (N = 3,497 in 1986) and at subsequent waves for which their data on all variables are available: N = 2,780 in 1989, N = 2,331 in 1994, and N = 1,566 in 2001. The fourth wave included fewer than 100 proxy interviews that were treated as self-reported interviews. The majority of attrition in subsequent waves is due to non-response and death. The number of non-respondents is 557 at wave two, 476 at wave three, and 607 at wave four; the number of those who died is 160 by wave two, increases to 530 by wave three, and 1,153 by the final wave. A small number of observations in the latter two waves are excluded due to missing values for one or more covariates. Together, these yield 10,174 person-year observations.

The ACL study uses a complex sampling design that oversampled blacks and adults over 60 years of age. A composite weight variable was developed for each wave to adjust sample distributions for the probabilities of selection within households, geographical and race group differences, differential nonresponse and post-stratification (to match the demographic distributions of the known population as estimated by the 1985 U.S. Census). All statistical analyses employed this composite sampling weight to produce unbiased estimates.[4] Table 1 shows the summary statistics of all variables used in the analyses for all waves combined.

Analytic Methods

We employ hierarchical linear models for longitudinal data, or growth curve models, to simultaneously estimate intra-cohort age trajectories of health and inter-cohort differences in age trajectories of health (Raudenbush and Bryk 2002; Yang 2007). The panel data have two levels, with repeated measurements at level one being nested within individuals at level two. At level 1, the response variable y_{ti} for person i at time t is modeled as a function of age (A). Accordingly, the model can be specified as the following:

$$\text{Level-1 Model: } Y_{ti} = \beta_{0i} + \beta_{1i} A_{ti} + \beta_{2i} A_{ti}^2 + e_{ti}$$

We estimated both a simple linear age model and a quadratic age model and found that the latter fits the data substantially better. Consequently, the present model includes both linear A and quadratic terms A^2 of age. The coefficients β_{0i}, β_{1i} and β_{2i} represent the intercept or mean level, the

Table 1: Weighted Descriptive Statistics of All Variables in the Analyses

Variable	Mean	SD	Min	Max
CES-D	-.14	.95	-1.18	4.74
Disability	1.29	.73	1	4
Self-rated health	3.61	1.03	1	5
Age	51.25	15.66	25	95
Birth cohort	—	—	0	6
Sex (female = 1)	.54	.50	0	1
Race (black = 1)	.11	.31	0	1
Died	.15	.35	0	1
Nonresponse	.18	.38	0	1
Education	12.62	2.89	0	17
Family income (in thousands)	53.40	60.09	0	2750
Marital status (not married = 1)	.31	.46	0	1
Chronic illness	1.06	1.24	0	8
BMI (ref. = Normal)				
Underweight (BMI $<$ 18.5)	.17	.38	0	1
Overweight (25 $\leqslant$ BMI$<$30)	.30	.46	0	1
Obese (BMI $\geqslant$ 30)	.14	.35	0	1
Smoking	4.99	10.22	0	50

Notes: Unweighted N = 10,174
Source: ACL 1986–2001/2

linear growth rate, and the quadratic growth rate of health with age, respectively. The random within-person error term, e_{ti}, is assumed to be normally distributed with means of 0 and variance of σ^2.[5] At level 2, each parameter of the age trajectories is further modeled as a function of person-level attributes: birth cohort (*C*) and its quadratic function C^2, the inclusion of which is determined by model fit, as is the case with age (*A*), sex (*S*), race (*R*) and the interaction effects of sex and cohort (*SC*) and race and cohort (*RC*). Level-2 Model: for the intercept:

$$\beta_{0i} = \gamma_{00} + \gamma_{01}C_i + \gamma_{02}C_i^2 + \gamma_{03}S_i + \gamma_{04}R_i + \gamma_{05}SC + \gamma_{06}RC + u_{0i}$$

for the linear growth rate:

$$\beta_{1i} = \gamma_{10} + \gamma_{11}C_i + \gamma_{12}C_i^2 + \gamma_{13}S_i + \gamma_{14}R_i + u_{1i}$$

In the model for the intercept, β_{0i}, $\gamma_{01} - \gamma_{06}$ are coefficients for the effects of *C, C², R, SC* and *RC*. In the model for linear growth rate, β_{1i}, $\gamma_{11} - \gamma_{14}$ are coefficients for the effects of *C, C²* and *R* on the linear rate of change with age (effects of *SC* and *RC* are not significant and omitted). A similar model for the quadratic growth rate is tested. Control variables are entered at

level-1 for time varying covariates and level-2 for education and attrition dummies. All continuous variables are centered in order for the intercept to be substantively meaningful (Yang 2007).

The HLM-growth curve methodology has the advantage of allowing data that are unbalanced in time (Raudenbush and Bryk 2002). That is, it incorporates all individuals with data for the estimation of trajectories, regardless of the number of waves he or she contributes to the person-year data set. Compared to alternative modeling techniques, this substantially reduces the number of cases lost to follow-up due to mortality or nonresponse. Even though previous studies using growth curve models include the deceased and nonrespondents in the analysis (Lynch 2003; Yang 2007), they do not automatically resolve the sample selection problem unless they distinguish the loss-to-follow-up from those with complete data for all waves. If mortality and nonresponse are significantly correlated with worse health and other key covariates of health such as age and SES, parameter estimates of health trajectories may be biased if they are not controlled. To fully assess the potential influence of selection due to death or nonresponse, we control for the effects of attrition by including dummy variables indicating the deceased and nonrespondents in the level-2 models. All statistical analyses are performed using SAS 9.1.

Results and Findings

We test three cohort-related hypotheses by estimating growth curve models of cohort, aging, sex, and race effects for each of the three health outcomes. We summarize the modeling results qualitatively in Table 2 for each hypothesis. We present the model effect coefficients, significance tests, and model fit statistics using the Bayesian Information Criteria in tables 3 through 5 for depressive symptoms, physical disabilities and self-assessments of health, respectively. Models 1–4 report decreasing BIC statistics, thus indicating improved model fit as the successive models are estimated. We also graphically display in figures 1 through 5 cohort differences in mean levels and growth trajectories of health that are statistically significant.

Inter-cohort Change

Results support Hypothesis 1 (see Table 2): there are significant cohort variations in both mean levels (β_{0i}) and growth rates (β_{1i} and β_{2i}) of health problems, but the directions of change differ by the health outcome examined. Specifically, the Model 1s in tables 3–5 show that more recent cohorts have lower mean levels of physical disability and higher mean levels of self-rated health, but suffer from higher mean levels of depression. This cohort effect (γ_{01}) is linear for the depression and self-rated health

Table 2: A Summary of Tests of Cohort-Related Hypotheses

Hypothesis	Effect on Health	Result		
		Depression	Disability	Self-Rated Health
1. Inter-cohort Change	Cohort			
	on mean level	+	–	+
	on growth rate	–	–	–
2. Intra-cohort Inequality	Sex (female = 1)			
	on mean level	+	+	–
	on growth rate	–	NS	NS
	Race (black = 1)			
	on mean level	+	+	–
	on growth rate	NS	NS	NS
3. Inter-cohort Difference in Intra-cohort Inequality	Cohort * Sex			
	on mean level	+	–	NS
	on growth rate	NS	NS	NS
	Cohort * Race			
	on mean level	+	–	–
	on growth rate	NS	NS	NS

Note: NS indicates the effect coefficient is not statistically significant.

models, with each successive cohort having a .03-unit increase in the standardized CES-D score and a .10-unit increase in self-rated health on average ($p < .001$). The cohort effect is quadratic for the disability model, with each successive cohort having a .28-unit lower disability score (γ_{01}) that decreases at an increasing rate of .02 (γ_{02})($p < .001$). The Model 1s also show that health problems increase significantly with age, as indicated by the intercept coefficients for the linear (γ_{10}) and quadratic growth rates (γ_{20}) for all three health outcomes. However, such quadratic age patterns are not universal across cohorts and, therefore, are not entirely developmental in nature but affected by social context as defined by cohort membership. More recent cohorts show smaller increases in depressive symptoms (γ_{11} = -.06, $p < .01$) and physical disability (γ_{11} = -.25, γ_{12} = .02, $p < .001$) with age, but slightly faster decreases in perception of good health (γ_{11} = -.03, $p < .05$) (see Figure 5 for additional results on disability and self-rated health).

The Model 1s and all subsequent models control for selection effects due to death and nonresponse in the follow-up surveys. The deceased have significantly more depressive symptoms, disabilities, and worse self-rated health than those who completed all surveys. The nonrespondents have more depression but do not differ from other survivors in disability or self-rated health. Adjusting for the attrition status produced estimates of age trajectories and cohort effects that would otherwise be biased.

Intra-cohort Inequality

Results only partially support the *intra-cohort inequality hypothesis* (see Table 2): there are significant intra-cohort sex and race inequalities in health trajectories, net of cohort effects. The Model 2s in tables 3–5 universally report significant and positive sex and race effects on the mean levels of health: women and blacks have on average more depressive symptoms, physical disabilities, and assess their health to be worse than men and whites do. Table 3 also shows that the sex gap in depression decreases with age, as indicated by the negative sex effect on the growth rate in Model 2 (γ_{13}= -.06, $p < .10$) that becomes more significant in models 3 and 4 with controls of other factors. This supports Hypothesis 2b that health gaps converge with age, as predicted by the age-as-leveler theory.

The Model 2s in tables 4 and 5 show no significant sex and race effects on growth rates of disability and self-rated health, thus providing no support for Hypothesis 2a or 2b when the inter-cohort change effect is controlled. In additional analyses, we tested the same sex and race effects change with age without controlling for cohort effects and found that both sex and race effects on disability increase with age and that sex effects on self-rated health also increase with age, as predicted by the cumulative advantage theory. Therefore, the life course changes in sex and race gaps in health that were found in previous studies are actually due largely to cohort differences. Taking cohort effects into account has substantially modified the existing understanding of the relationships between sex/race inequalities and aging. We found evidence for a converging sex gap in depression trajectories, but constant sex and race gaps in physical and self-rated health trajectories within cohorts.

Inter-cohort Difference in Intra-cohort Inequality

As for the third hypothesis that intra-cohort sex and race gaps in health trajectories vary across cohorts, there is support for inter-cohort variations in intra-cohort sex and race differences in mean health levels, but not in growth rates of health problems except for depression (see Table 2). We tested the hypothesis in the Model 3s, which include cohort-by-sex and cohort-by-race interaction effects for the models of intercept or mean levels (β_{0i}) and the models of growth rates (β_{1i} and β_{2i}).

Table 3 shows positive cohort-by-sex interaction (γ_{05} = .02) and cohort-by-race interaction (γ_{06} = .04, $p < .05$) effects for the mean level of depression. Controlling for other risk factors, the cohort-by-sex interaction effect increases in size and becomes significant in Model 4. This supports Hypothesis 3b that sex and race gaps widen across cohorts. Figure 1 shows the predicted mean levels of CES-D scores by cohort for each sex and race based on estimates of Model 4, adjusting for age and all

Table 3: Growth Curve Model Estimates of Cohort, Aging, Sex and Race Effects on Depressive Symptoms

		Model 1	Model 2	Model 3	Model 4
Fixed Effects Parameters		Coef. (t ratio)	Coef. (t ratio)	Coef. (t ratio)	Coef. (t ratio)
For Intercept	β_{0i}				
Intercept	γ_{00}	-.34***	-.53***	-.45***	.16
Cohort	γ_{01}	.03***	.04***	.02	.06*
		(3.45)	(3.52)	(1.01)	(2.15)
Sex (Female = 1)	γ_{03}		.20***	.13#	-.07
			(6.66)	(1.93)	(-1.19)
Race (Black = 1)	γ_{04}		.32***	.16#	-.12
			(8.84)	(1.86)	(-1.49)
Cohort * Sex	γ_{05}			.02	.03*
				(1.09)	(2.31)
Cohort * Race	γ_{06}			.04*	.07***
				(2.10)	(3.85)
For Linear Growth Rate	β_{1i}				
Intercept	γ_{10}	.13**	.17***	.17***	.05
		(2.93)	(3.33)	(3.33)	(.72)
Cohort	γ_{11}	-.06***	-.06***	-.06***	-.03**
		(-5.84)	(-5.91)	(-5.84)	(-2.63)
Sex (Female = 1)	γ_{13}		-.06#	-.06*	-.08*
			(-1.95)	(-2.02)	(-2.57)
Race (Black = 1)	γ_{14}		.03	.02	-.01
			(.74)	(.56)	(-.41)
For Quadratic Growth Rate	β_{2i}				
Intercept	γ_{20}	.09***	.09***	.09***	.09***
		(4.69)	(4.54)	(4.55)	(4.78)
Control Variables					
Died		.37***	.38***	.37***	.07*
		(9.13)	(9.43)	(9.32)	(2.07)
Nonresponse		.20***	.16***	.16***	.09**
		(5.87)	(4.88)	(4.70)	(3.13)
Education					-.04***
					(-8.49)
Family income					-.00*
					(-2.53)
Marital status (not married = 1)					.24***
					(10.93)
Chronic illness					.07***
					(7.29)
BMI (reference = normal)					
Underweight					.22***
					(3.36)

Table 3 ***continued***

		Model 1	Model 2	Model 3	Model 4
Fixed Effects Parameters		Coef. (t ratio)	Coef. (t ratio)	Coef. (t ratio)	Coef. (t ratio)
Overweight					-.07*** (-3.49)
Obese					-.07* (-2.57)
Smoking					.00*** (3.97)
Disability					.20*** (15.15)
Self-rated health					-.17*** (-16.95)
Random Effects – Variance Components					
Level-1: within-person	σ^2	.38***	.38***	.38***	.36***
Level-2: ln intercept	r_{u0}	.44***	.41***	.41***	.27***
ln growth rate	r_{u1}	.16***	.16***	.16***	.14***
Goodness-of-fit (BIC)		28100.5	27975.8	27973.0	26773.5

*p < .05 **p < .01 ***p < .001 (two-tailed test) #p < .10 (one-tailed test)

other factors. More recent cohorts have higher mean levels of depression. Sex and race differences are small in the earliest two cohorts, with male and white cohorts having slightly more depression, but large in more recent cohorts, with female and black cohorts having substantially more depression. The reversal and widening of the gaps is due to steeper increases in mean CES-D scores for female and black cohorts in each successive cohort.

In contrast, Model 3 in Table 4 shows negative cohort-by-sex interaction (γ_{05} = -.04, p < .05) and cohort-by-race interaction (γ_{06} = -.03, p < .05) effects for the mean level of disability, supporting Hypothesis 3a that sex and race gaps narrow across cohorts. Figure 2 shows the patterns of quadratic cohort declines in predicted mean levels of disability by race from Model 3. Female and black disadvantages in physical functioning are roughly constant in earlier cohorts but decrease in more recent cohorts born after 1935. Model 4 shows that cohort changes in the race gap are largely explained away by other social and biological risk factors while those in the sex gap remain significant.

Table 5 shows that the cohort-by-sex interaction and cohort-by-race interaction effects are not significant for the mean level of self-rated health in Model 3, but the latter becomes significant in Model 4 with adjustment

Table 4: Growth Curve Model Estimates of Cohort, Aging, Sex and Race Effects on Functional Disability

Fixed Effects Parameters		Model 1 Coef. (t ratio)	Model 2 Coef. (t ratio)	Model 3 Coef. (t ratio)	Model 4 Coef. (t ratio)
For Intercept	β_{0i}				
Intercept	γ_{00}	2.00***	1.85***	1.73***	1.50***
Cohort	γ_{01}	-.28***	-.27***	-.24***	-.17***
		(-8.59)	(-8.39)	(-6.92)	(-4.58)
Cohort^2	γ_{02}	.02***	.02***	.02***	.02***
		(4.86)	(4.81)	(4.63)	(5.76)
Sex (Female = 1)	γ_{03}		.17***	.30***	.19***
			(6.71)	(5.83)	(4.00)
Race (Black = 1)	γ_{04}		.12***	.25***	.06
			(4.06)	(3.91)	(.98)
Cohort * Sex	γ_{05}			-.04**	-.03*
				(-2.88)	(-2.49)
Cohort * Race	γ_{06}			-.03*	-.01
				(-2.24)	(-.61)
For Linear Rate of Change	β_{1i}				
Intercept	γ_{10}	.83***	.78***	.77***	.73***
		(11.24)	(10.12)	(10.07)	(8.43)
Cohort	γ_{11}	-.25***	-.25***	-.25***	-.28***
		(-6.00)	(-5.93)	(-5.91)	(-6.63)
Cohort^2	γ_{12}	.02***	.02***	.02***	.02***
		(3.82)	(3.80)	(3.81)	(4.62)
Sex (Female = 1)	γ_{13}		.05$^{\#}$	.05$^{\#}$	.04
			(1.74)	(1.72)	(1.51)
Race (Black = 1)	γ_{14}		.07$^{\#}$	.07$^{\#}$	.06
			(1.89)	(1.92)	(1.61)
For Quadratic Rate of Change	β_{2i}				
Intercept	γ_{20}	.31***	.30***	.30***	.33***
		(6.51)	(6.38)	(6.33)	(7.31)
Cohort	γ_{21}	-.06***	-.06***	-.06***	-.06***
		(-5.62)	(-5.52)	(-5.47)	(-6.13)
Control Variables					
Died		.44***	.46***	.47***	.35***
		(14.79)	(15.31)	(15.50)	(12.55)
Education					-.02***
					(-7.07)
Family income					-.00$^{\#}$
					(-1.84)
Marital status (not married = 1)					.04*
					(2.39)

Table 4 *continued*

		Model 1	Model 2	Model 3	Model 4
Fixed Effects Parameters		Coef. (t ratio)	Coef. (t ratio)	Coef. (t ratio)	Coef. (t ratio)
Chronic illness					.16*** (23.03)
BMI (reference = normal)					
Underweight					.22*** (4.38)
Overweight					-.02 (-1.47)
Obese					.05* (2.25)
Smoking					-.00 (-1.59)
CES-D					.11*** (14.50)
Self-rated health					-.18*** (-25.33)
Random Effects – Variance Components					
Level-1: within-person	σ^2	.22***	.22***	.22***	.22***
Level-2: In intercept	r_{u0}	.30***	.29***	.29***	.21***
In growth rate	r_{u1}	.15***	.15***	.15***	.14***
Goodness-of-fit (BIC)		22505.4	22459.6	22455.9	21838.0

*p < .05 **p < .01 ***p < .001 (two-tailed test) #p < .10 (one-tailed test)

of control variables (γ_{06} = -.04, p < .05). Figure 3 presents the predicted cohort differences in mean self-rated health by race adjusting for all other factors. Similar to the results on depression that support Hypothesis 3b, a divergence in the race gap occurs for more recent cohorts born after 1925 due to smaller increases for black cohorts compared to white cohorts in self-rated health.

The cohort-by-sex and cohort-by-race interaction effects are not significant in the models of growth rates when the cohort, sex, and race effects are controlled and thus are omitted. Combining results from the models of intercept and growth rates, we find that significant inter-cohort differences exist in changes of intra-cohort sex gaps in depression but not in changes of intra-cohort sex or race gaps in disability or self-rated health. This is expected given the results from the tests of Hypothesis 2a and b, which indicate that a significant intra-cohort sex gap in growth rates is found only in depression, controlling for cohort effects. Figure 4 shows the predicted sex-specific age trajectories of depression for select cohorts based on Model 4. Consistent with previous findings from Yang (2007),

Table 5: Growth Curve Model Estimates of Cohort, Aging, Sex and Race Effects on Self-Rated Health

Fixed Effects Parameters		Model 1 Coef. (t ratio)	Model 2 Coef. (t ratio)	Model 3 Coef. (t ratio)	Model 4 Coef. (t ratio)
For Intercept	β_{0i}				
Intercept	γ_{00}	3.15***	3.28***	3.26***	3.34***
Cohort	γ_{01}	.10*** (9.31)	.10*** (9.30)	.11*** (6.75)	.03 (1.02)
Sex (Female = 1)	γ_{03}		-.13*** (-4.02)	-.09 (-1.24)	.02 (.50)
Race (Black = 1)	γ_{04}		-.25*** (-6.52)	-.25** (-2.89)	.07 (.91)
Cohort * Sex	γ_{05}			-.01 (-.63)	-.01 (-.92)
Cohort * Race	γ_{06}			.00 (.07)	-.04* (-2.15)
For Linear Growth Rate	β_{1i}				
Intercept	γ_{10}	-.08 (-1.58)	-.09# (-1.69)	-.09# (-1.72)	-.07 (-1.03)
Cohort	γ_{11}	-.03* (-2.37)	-.02* (-2.23)	-.02* (-2.21)	-.03* (-2.75)
Sex (Female = 1)	γ_{13}		.04 (1.12)	.04 (1.16)	.04 (1.13)
Race (Black = 1)	γ_{14}		-.08# (-1.80)	-.08# (-1.80)	-.05 (-1.12)
For Quadratic Rate of Change	β_{2i}				
Intercept	γ_{20}	.09*** (4.50)	.10*** (4.59)	.10*** (4.59)	.07*** (3.52)
Control Variables					
Died		-.48*** (-11.44)	-.48*** (-11.54)	-.48*** (-11.58)	-.29*** (-7.90)
Education					.05*** (9.76)
Family income					.00*** (3.31)
Marital status (not married = 1)					.02 (.83)
Chronic illness					-.26*** (-29.20)
BMI (reference = normal)					
Underweight					-.46*** (-6.72)
Overweight					-.05* (-2.21)

Table 5 *continued*

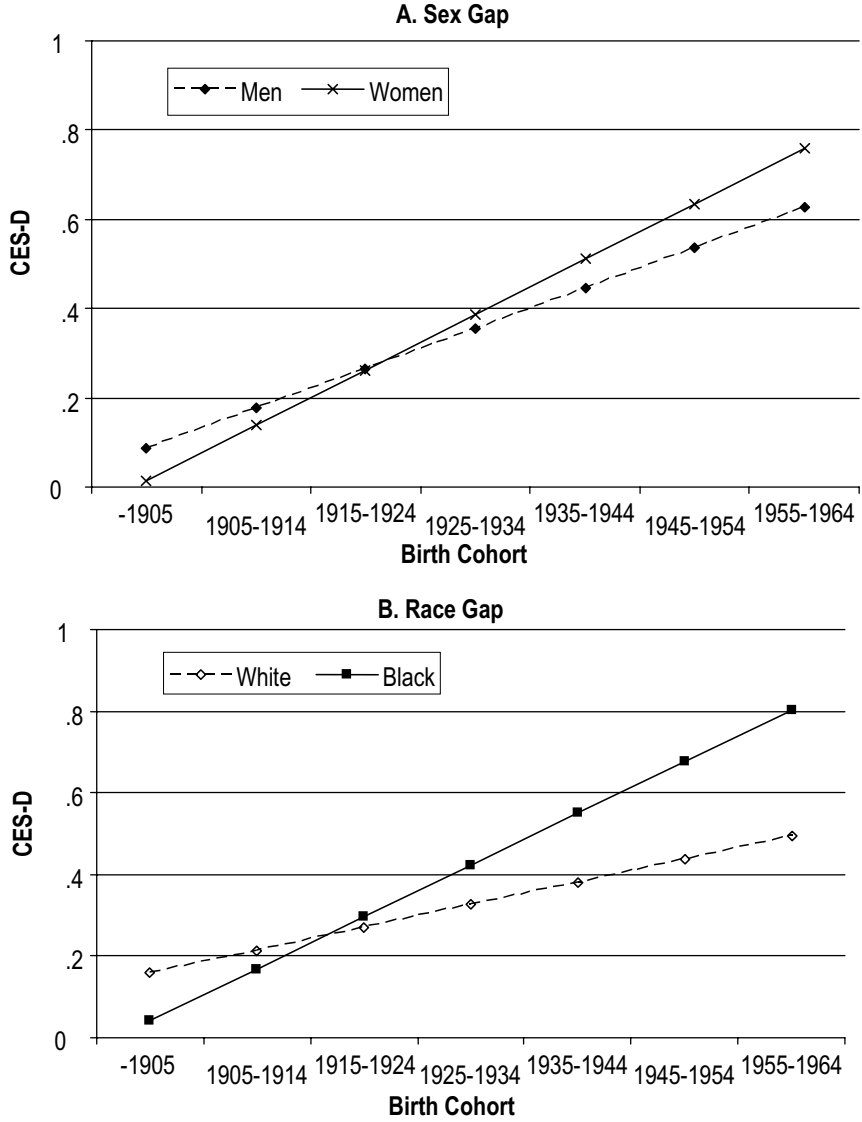
		Model 1	Model 2	Model 3	Model 4
Fixed Effects Parameters		Coef. (t ratio)	Coef. (t ratio)	Coef. (t ratio)	Coef. (t ratio)
Obese					-.24*** (-7.75)
Smoking					-.01*** (-4.39)
CES-D					-.17*** (-17.31)
Disability					-.32*** (-24.98)
Random Effects – Variance Components					
Level-1: within-person	σ^2	.38***	.38***	.38***	.37***
Level-2: In intercept	r_{u0}	.52***	.51***	.51***	.33***
In growth rate	r_{u1}	.24***	.24***	.24***	.24***
Goodness-of-fit (BIC)		28592.3	28543.8	28538.9	27416.5

*p < .05 **p < .01 ***p < .001 (two-tailed test) #p < .10 (one-tailed test)

whereas the gross age effects in Model 1 indicate increases in depression with age, the net depression trajectories show decreases with age within cohorts after adjusting for the effects of SES, marital status and physical illness. Furthermore, in support of Hypothesis 3c, the within-cohort sex gap does not uniformly increase or decrease across the life course but varies by cohort. For instance, the sex gap first diverges in the earliest cohort born before 1905, with men having more depressive symptoms than women, then crosses over in the 1915-24 cohort, converges in the War Babies, and becomes largely constant in the Baby Boomers. Overall, this shows a trend of decreasing degrees of or lessening convergence in female and male depression trajectories in more recent cohorts.

Intra-cohort sex and race gaps in disability and self-rated health do not significantly change with age. Therefore, these gaps in growth trajectories are constant within cohorts. Figure 5 displays the predicted cohort differences in growth trajectories in disability and self-rated health based on the Model 4s. Adjusting for all other factors reduces the magnitudes of the cohort effect coefficients in the intercept model for disability and diminishes the significance level in the intercept model for self-rated health. Therefore, inter-cohort variations in mean levels of disability and self-assessments of health can be largely explained by cohort differences in SES, marital status, chronic illness, obesity and mental health. With self-

Figure 1: Predicted Mean Levels of CES-D Score by Birth Cohort: Sex and Race Gaps



assessments of health, cohort differences in patterns of smoking also play a role. The net effect of inter-cohort change that supports Hypothesis 1 is still evident: controlling for all other factors, more recent cohorts have, on average, less disability and better self-rated health, and disability increases less steeply with age in more recent cohorts. At the same time, however, Figure 5 shows that successive cohorts have higher levels of disability than their predecessors do at the same ages. This may be due to an earlier onset of debilitating conditions in recent cohorts, such as obesity

Figure 2: Predicted Mean Levels of Disability by Birth Cohort: Sex and Race Gaps

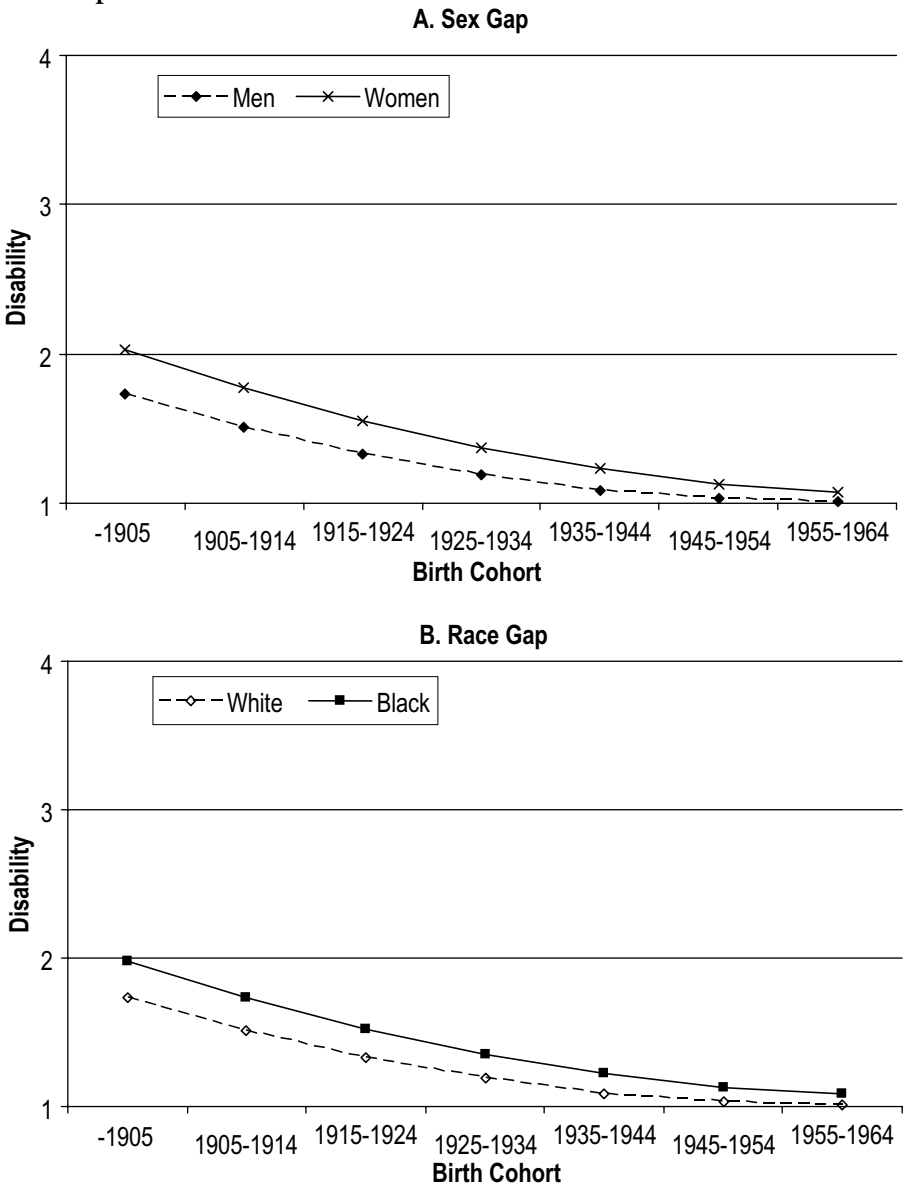


Figure 3: Predicted Mean Levels of Self-Rated Health by Birth Cohort: Race Gap

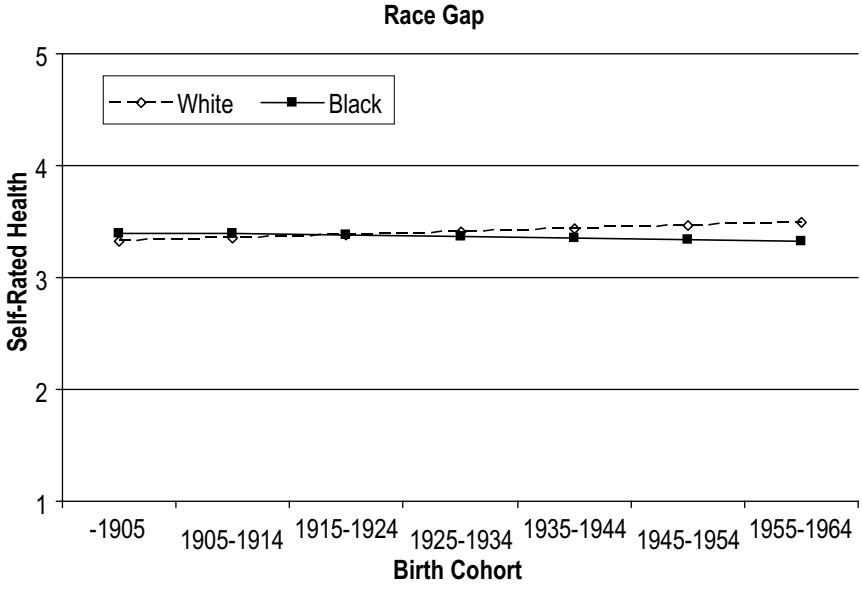


Figure 4: Predicted Age Growth Trajectories of CES-D Score by Birth Cohort: Sex Gap

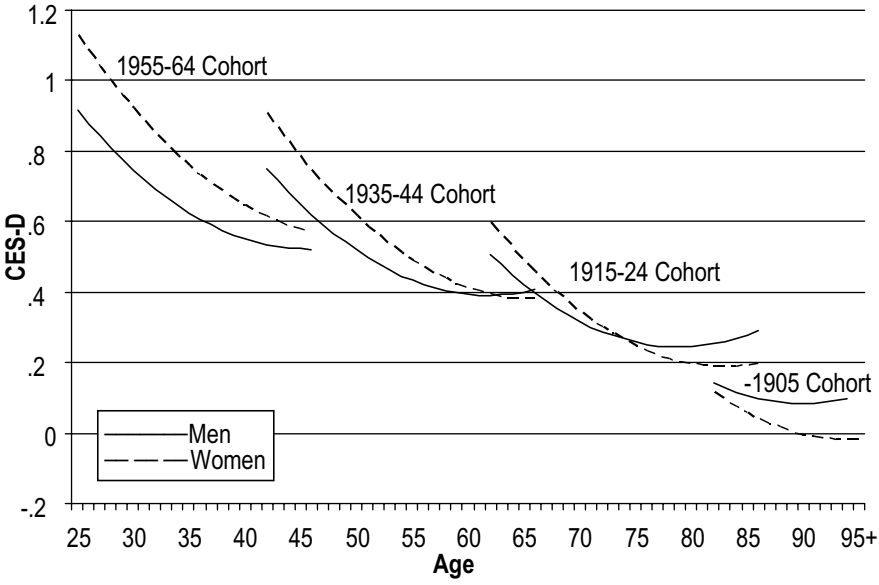
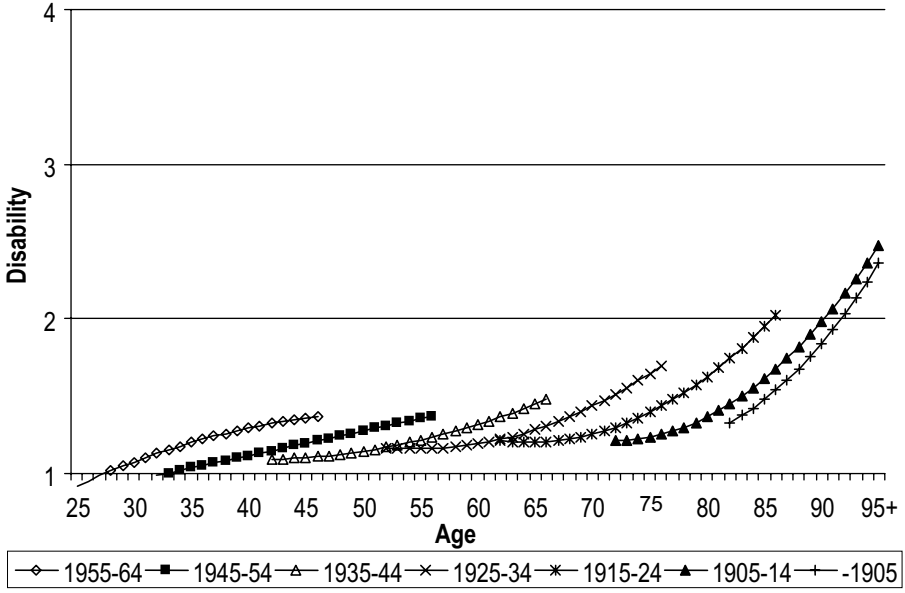
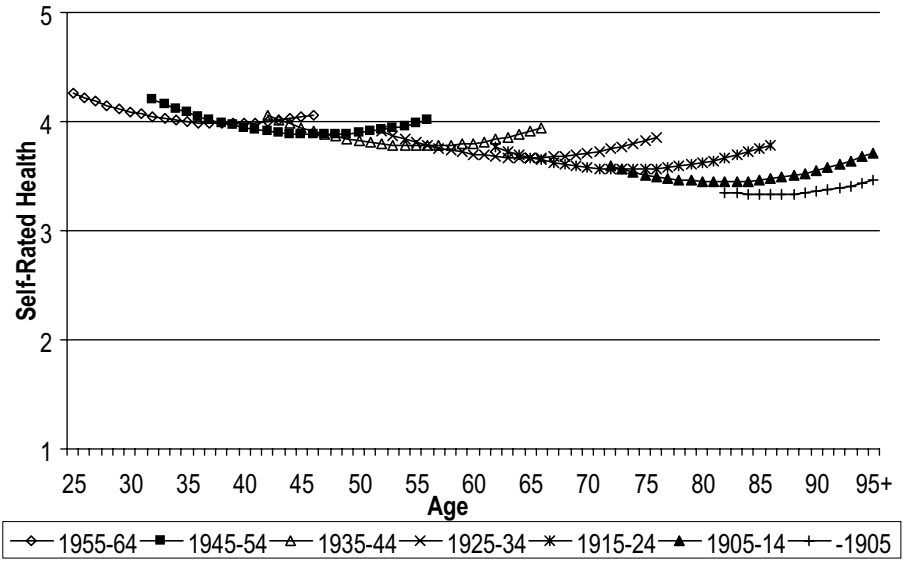


Figure 5: Predicted Age Growth Trajectories of Disability and Self-Rated Health by Birth Cohort





and diabetes. More recent cohorts also report slightly larger declines in general health with age than earlier cohorts.

Discussion and Conclusion

This longitudinal study addresses some long-standing questions in the stratification of aging from the perspective of cohort analysis. We evaluate the proposition that considering the process of cohort change is important for the theory, measurement and analysis of social inequalities in health over the life course. We find substantial evidence that supports this proposition based on a systematic investigation of the distinct role of aging and cohort in the relationships between sex/race and physical and mental health. We find that changes in disparities in depressive symptoms, disability and self-assessments of health across the life course are largely cohort-related phenomena. We also identify major mechanisms by which cohort differences in these health outcomes are realized. The findings prompt more careful and thorough examinations of various aging-related hypotheses in a cohort-specific context.

Our first question concerns how cohort effects condition overall health trajectories over the life course. We find strong support for the *inter-cohort change hypothesis* that there exist substantial inter-cohort variations in aging trajectories of health. Our findings suggest that inter-cohort change in health may be due to social historical changes exogenous to developmental/age changes in physical and mental states. This finding highlights the necessity of examining cohort effects for a proper attribution of aging effects in life course research.

We also find that the directions of cohort change do not consistently favor more recent cohorts but differs by health outcomes and parameters of health trajectories. On the one hand, more recent cohorts fare increasingly better in mean levels of physical functioning and self-assessments of health (see figures 2 and 3) and experience faster declines in depression and slower increments in disability with age (see figures 4 and 5). This reflects the long-term health benefits of physiological improvements and smoking cessation across cohorts. On the other hand, more recent cohorts suffer from more depressive symptoms on average (see Figure 1) and show faster declines in perceived general health with age than earlier cohorts do (see Figure 5). As expected, this pattern partly reflects the mental health impacts of unique demographic and cultural experiences of different 20th-century cohorts with regards to marriage patterns, family structures and lifestyles affecting nutrition and body mass. The finding is also consistent with previous research that shows that earlier cohorts born in and coming of age during the Great Depression and world wars may have gained more satisfaction and positive views of themselves

having survived economic deprivations, social instability and related social hardships (Idler 1993). Later cohorts, in contrast, face more complicated problems associated with prosperity – such as labor market competition, human relations and medicalization – and may thus manifest more mental health problems (Yang 2007, 2008).

The second question concerns how cohort effects alter existing explanations of sex and race gaps in health trajectories over the life course. We find support for the *intra-cohort inequality hypothesis* only in the analysis of depression, which shows that aging narrows and levels the sex gap over the life course net of cohort effects. This finding on depression is consistent with the role theory of aging (Gove, Ortega and Style 1989); self-integration, insight and positive psychosocial traits such as satisfaction and self-esteem all grow with age and, in turn, these signs of maturity decrease depression. We do not find evidence for diverging or converging intra-cohort inequalities in disability and self-rated health net of inter-cohort differences in health trajectories. Therefore, neither the cumulative advantage nor the age-as-leveler theory explain the mechanisms generating the life course patterns of sex and race disparities in these two health outcomes. In other words, sex and race inequalities in disability and self-rated health across the life course are persistent and constant. We, therefore, reach the same conclusion as that of Ferraro and colleagues. However, our examination of cohort effects in the longitudinal growth curve analyses provides more conceptual clarity and a better empirical test of competing aging theories. The strong presence of inter-cohort differences as opposed to age differences in sex and race gaps in health suggests that we focus on cohort-related explanations, which are further addressed by the third question.

The third question assesses how cohorts differ in patterns of intra-cohort changes in sex and race inequalities in health trajectories. We find support for the *inter-cohort difference in intra-cohort inequality hypothesis* that sex and race gaps in levels of health change across cohorts. The directions of such changes differ by health outcomes. With respect to mean levels of disability, the sex and race gaps have narrowed (see Figure 2). This is expected because large overall cohort improvements in health capital directly led to better physiological capacities and greater improvements for the most recent female and black cohorts, thereby reducing their disadvantages in physical functioning relative to their male and white counterparts. However, cohort reduction in sex and race inequalities in physical health does not necessarily translate into similar changes in mental and perceived health. In fact, we find that these gaps grow larger in more recent cohorts due to larger increases in mean levels of depression for female and black cohorts and smaller increases in mean self-assessments of health for black cohorts (see figures 1 and 3). Persistent

and growing sex and race inequalities in depression and self-rated health in successive birth cohorts may indicate a lack of improvement and possible adverse changes in the social risk factors of these two health indicators for women and blacks. For example, women born after WWII experienced unanticipated increases in social stress associated with balancing work and family, marital dissolution and economic deprivation in female-headed households that decreased their perceived sense of well-being. Racial discrimination in terms of residential segregation and economic isolation also continues to affect blacks' quality of life in ways that may matter more for their mental than physical health. Future research needs to incorporate explicit measures of these factors to further explain cohort variations in sex and race gaps in health.

We also find evidence for inter-cohort differences in the intra-cohort sex gaps in growth rates of depression. This means that the salience of sex stratification in depression not only varies across the life course but also varies across social groups defined by cohort membership. In fact, the patterns of sex gap changes with age are strongly modified by and dependent on birth cohort. We see less convergence in the male-female gap in depression with age in more recent cohorts, suggesting a weaker age-as-leveler process in these cohorts (see Figure 4). The reversal in the sex difference in depression trajectories in the earliest cohort born before 1905 may be related to war combat experiences and traumas that elevated depression in male veterans in those cohorts. Future studies should formally examine this speculation with more specific measures of cohorts' life histories. Because we do not find any significant changes in the intra-cohort sex or race gaps in disability or self-rated health with age but find substantial inter-cohort variations in sex and race gaps in mean levels of these outcomes, we conclude that any changes with age observed in previous studies are actually due to inter-cohort differences in disability or self-rated health levels. Thus, there is no stronger cumulative advantage process or age-as-leveler effect at work in later or earlier cohorts when the confounding effects of aging and cohort are distinguished and other social factors controlled.

We do not completely understand the mechanisms contributing to cohort variations in life course patterns of health disparities. The analysis shows that adjusting for socioeconomic status, marital status, physiological conditions, and smoking reduces cohort effects and sex and race gaps in cohort means and age trajectories. Thus, a large part of the cohort variations under discussion can be attributed to cohort changes and differential changes in these factors by sex and race across cohorts. The residual cohort effects, however, remain significant. Therefore, other mechanisms may be at work. First, changes in cohort and age effects may be related to changes with age or cohort in the impact of correlates on

health. For example, the convergence in the sex gap in depression with age may be a result of social resources and status having less impact on mental health as one matures and becomes more immune to stressors. The larger sex and race gaps in health in more recent cohorts may be due to a greater impact of social risk factors on health in these cohorts. We assessed this possibility by including in the models a series of interaction terms of age and each control variable and of cohort and each control variable. No significant interaction effects were found, so it is unlikely that a differential impact of these factors with age or cohort explains life course or cohort patterns. Second, selective survival may decrease heterogeneity in health in older ages. But the results on attrition effects suggest that selective survival does not act as the dominant force that drives life course changes.

Third, this study is by no means exhaustive in the search for potential explanatory factors. A myriad of mental and physical health correlates such as biomarkers of physiological capacity, social relations, social roles, stress and other environmental factors are not included in the analysis but may be additional sources of cohort variation; these should be examined in future research. Findings of cohort differences in several self-reported health outcomes based on one sample of moderate size are also insufficient for generalization and must be further tested in additional studies using other nationally representative surveys and objective health measures.

In conclusion, cohort membership contextualizes individuals' health change with age and also conditions sex and race inequalities in health. Cohort effects also seem to be essential for moderating sex inequality in age trajectories for certain health indicators such as depression. The significance of cohort change in shaping social inequalities in health over the life course implies the relevance of both biological forces and historical context, and it suggests emerging new patterns for future cohorts entering adulthood and old age as a result of their new life circumstances.

Notes

1. Ages above 95 were recoded as 95 to avoid erratic estimates in the growth curve models due to extremely small sample sizes for these ages.

2. We experimented with different cohort groupings such as five-year intervals and unequal intervals corresponding to the unequal intervals between each survey and found largely similar results. The current operationalization, however, is superior in terms of parsimony.

3. A small number of respondents of other races (3 percent of total sample) were excluded in the final analyses because regression results show they do not differ significantly from blacks in health measures included in this study.

4. Regression analyses were conducted with and without weights. The results are largely similar. We report results from regressions with weights here since not all factors affecting the sampling weight are controlled in regression models.

5. Alternative distributional assumptions were tested using Generalized Hierarchical Linear Models such as hierarchical binomial logit (e.g., disabled vs. no disability) and ordinal logit models (e.g., self-rated health). Results are similar. HLM with normal link is presented here for ease of interpretation across all health outcomes.

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