



Trends in Mortality

Recent trends in US mortality in early and middle adulthood: racial/ethnic disparities in inter-cohort patterns

Emma Zang,^{1*} Hui Zheng,² Yang Claire Yang³ and Kenneth C Land⁴

¹Sanford School of Public Policy, Duke University, Durham, NC, USA, ²Department of Sociology, Ohio State University, Columbus, OH, USA, ³Department of Sociology, Lineberger Cancer Center, and Carolina Population Center, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA and ⁴Department of Sociology and Social Science Research Institute, Duke University, Durham, NC, USA

*Corresponding author. Sanford School of Public Policy, Duke University, Durham, NC 27701, USA. E-mail: xiaolu.zang@duke.edu

Editorial decision 15 October 2018; Accepted 27 October 2018

Abstract

Background: A striking increase in the all-cause mortality of US middle-aged non-Hispanic Whites in the past two decades has been documented by previous studies. The inter-cohort patterns in US mortality, as well as their racial/ethnic disparities, are still unclear.

Methods: Using official mortality data, we study US annual mortality rates for ages 25–54 from 1990 to 2016 by gender and race/ethnicity. We conduct an age-period-cohort analysis to disentangle the period and cohort forces driving the absolute changes in mortality across cohorts. Nine leading causes of death are also explored to explain the inter-cohort mortality patterns and their racial/ethnic disparities.

Results: We find cohort-specific elevated mortality trends for gender- and race/ethnicity-specific populations. For non-Hispanic Blacks and Hispanics, Baby Boomers have increased mortality trends compared with other cohorts. For non-Hispanic White females, it is late-Gen Xers and early-Gen Yers for whom the mortality trends are higher than other cohorts. For non-Hispanic White males, the elevated mortality pattern is found for Baby Boomers, late-Gen Xers, and early-Gen Yers. The mortality pattern among Baby Boomers is at least partially driven by mortality related to drug poisoning, suicide, external causes, chronic obstructive pulmonary disease and HIV/AIDS for all race and gender groups affected. The elevated mortality patterns among late-Gen Xers and early-Gen Yers are at least partially driven by mortality related to drug poisonings and alcohol-related diseases for non-Hispanic Whites. Differential patterns of drug poisoning-related mortality play an important role in the racial/ethnic disparities in these mortality patterns.

Conclusions: We find substantial racial/ethnic disparities in inter-cohort mortality patterns. Our findings also point to the unique challenges faced by younger generations.

Key words: Adult mortality, racial/ethnic disparities, gender disparities, cohort analysis

Key Messages

- For non-Hispanic Blacks and Hispanics, Baby Boomers have increased mortality trends compared with other cohorts.
- For non-Hispanic White females, late-Gen Xers and early-Gen Yers have increased mortality trends compared with other cohorts.
- For non-Hispanic White males, Baby Boomers, late-Gen Xers and early-Gen Yers have increased mortality trends.
- The elevated mortality pattern among Baby Boomers is at least partially driven by mortality related to drug poisoning, suicide, external causes, chronic obstructive pulmonary disease and HIV/AIDS for all race and sex groups.
- Differential patterns of drug poisoning-related mortality play an important role in the racial/ethnic disparities in these mortality patterns.

Introduction

Recent documentation of a striking increase in the all-cause mortality rates of middle-aged non-Hispanic Whites in the past two decades^{1,2} has stimulated extensive discussion.^{3–9} Cumulative disadvantages triggered by progressively worsening labour market opportunities at the time of entry among non-Hispanic White cohorts have been proposed to explain this mortality trend.^{1,2} However, most existing research and media reports have limited their attention to non-Hispanic Whites and have not focused on racial/ethnic differences more broadly. In addition, although there is some evidence showing that the increasing mortality trend is not confined to the Baby Boomers,^{1,2} the inter-cohort patterns in the mortality increase are still unclear.

Building on the foregoing research, this paper analyses the inter-cohort patterns in US mortality in early and middle adulthood from 1990 to 2016, by gender and race/ethnicity. Our focus on early and middle adulthood is driven by the existing evidence that mortality under age 50 accounts for much of the fact that US life expectancy lags behind that of other industrialized countries.¹⁰ To link our findings to historical context in the USA, we group all the single birth-year cohorts into five generations: early-Baby Boomers (born 1946–55), late-Baby Boomers (born 1956–64), early-Generation Xers (born 1965–72), late-Generation Xers (born 1973–80) and early-Generation Yers (born 1981–91). We first display the inter-cohort patterns in the absolute changes of age-specific mortality by gender for non-Hispanic Whites, non-Hispanic Blacks and Hispanics. Considering that these patterns may be driven by age compositions and period fluctuations rather than reflecting the real cohort patterns, inter-cohort patterns for all-cause mortality rates are also examined independent of period and age effects. For a closer look at interactions between cohort and age effects on mortality trends, we break down our analysis by 10-year age groups from early to middle adulthood: 25–34, 35–44 and 45–54. Finally, to provide some preliminary

explanations on potential causes for our identified cohorts with elevated mortality trends, we examine the inter-cohort patterns of a variety of major causes of death, including drug poisonings, suicides, alcohol-related diseases, heart disease & stroke, HIV/AIDS, diabetes mellitus, external causes, chronic obstructive pulmonary disease (COPD) and cancer. Results from our analyses show substantial racial/ethnic differences in inter-cohort mortality patterns. Besides the already much discussed Baby Boomers, late-Gen Xers and early-Gen Yers are also identified with increasing mortality trends, to which adequate attention has not been paid previously.

Methods

We study US annual mortality rates for ages 25–54 from 1990 to 2016. Data on death counts come from the Center for Disease Control and Prevention (CDC) Mortality Multiple Cause Files. For a reference population at risk, we use US population data from the National Cancer Institute Surveillance, Epidemiology and End Results Registry (SEER) for the respective years. We focus our analysis on three race/ethnicity groups: non-Hispanic Whites, non-Hispanic Blacks and Hispanics. Because Hispanic/non-Hispanic origin can only be distinguished in the SEER population data after the year 1989, we study the years 1990–2016. Since our analyses focus on mortality patterns in early and middle adulthood, we limit the data to ages 25–54. To examine what drives increasing mortality among our identified cohorts, 5-year cause-specific mortality of drug poisonings, suicides, alcohol-related diseases, heart disease and stroke, HIV/AIDS, diabetes mellitus, external causes, COPD and cancer also is examined. Details on coding causes of death are documented in the supporting information (SI Coding Cause of Death, available as [Supplementary data](#) at *IJE* online).

When estimating death rates using data from both vital statistics and population census, discrepancies between numerators and denominators of death rates may arise due

to inconsistencies of Hispanic origin classifications in the two data sources.¹¹ These inconsistencies result from different reporting procedures adopted by these two data sources. When collecting census data, information on Hispanic origin is typically self-reported by a respondent, whereas a decedent's ethnicity on the death certificate is typically reported by the funeral director.¹¹ Sometimes the funeral director consults next of kin about this information, but often times he or she makes a judgement based on his or her own observations.¹¹ We distinguish Hispanic ethnicity despite this limitation, for two reasons. First, most previous studies have only focused on Black-White disparities.^{8,12–16} Few studies have separated out Hispanic ethnicity when examining racial/ethnic disparities in mortality. Not considering ethnicity may be more problematic for younger cohorts, because Hispanics comprise increasing shares of the Black and (particularly the) White populations in recent decades, and the periods under examination in our analysis are from the past three decades. Second, there is some previous evidence showing that adjusting for death certificate ethnic misclassification does not affect Hispanic-White mortality differentials.^{11,17}

To simultaneously estimate age, period and cohort effects, we estimate log-linear multiple classification regression models of mortality rates in the following form:¹⁸

$$\log(E_{ij}) = \log(P_{ij}) + \mu + \alpha_i + \beta_j + \gamma_k$$

where E_{ij} denotes the expected number of deaths in cell (i, j) that is assumed to be distributed as a Poisson variate, $\log(P_{ij})$ is the log of the exposure P_{ij} and is called the 'offset' or adjustment for the log-linear contingency table model, μ denotes the intercept or adjusted mean death rate, α_i denotes the i -th age effect, β_j denotes the j -th period effect, and γ_k denotes the k -th cohort effect.^{18,19} We applied the intrinsic estimator (IE) to obtain model effect coefficient estimates. The linear dependency between A, P and C variables in an age-by-period array of aggregate rates (Cohort = Period–Age) leads to a model identification problem when estimating linear and fixed effects of A, P and C. Among all existing methods that attempt to address this identification problem, the IE imposes fewer assumptions and yields valid model estimates.²⁰ Specifically, the IE method imposes an orthogonality identifying constraint, and estimates a corresponding estimable APC coefficient vector by the Moore-Penrose generalized inverse matrix.^{21–24} A review of its key assumptions and the sensitivity of its results to violations of these key assumptions can be found in previous publications.²⁴ For our specific analysis, the IE is the preferred solution to estimating APC variation in mortality when using National Vital Statistics System data.²²

Similar with other APC strategies, the IE estimates can be sensitive to the peculiarities of the data analysed and to the particular methodological decisions made in their application. Therefore, we conducted sensitivity/robustness analyses of the estimates we obtained from the IE. These include: (i) estimation of model selection statistics to justify our choice of the full APC accounting model over other simpler models; (ii) an assessment of the internal consistency of the IE estimates to choice of different reference categories in the model specification; (iii) an examination of the IE estimates for sensitivity to our cause-specific mortality model specifications by categorizing 5-year measures into 10-year groupings; and (iv) an evaluation of the IE estimates of the A, P and C effect coefficients for robustness as compared with estimates from an alternative method of APC model specification and estimation—the hierarchical age-period-cohort (HAPC) cross-classified random effects models.⁹ All of the IE results reported for the log-linear APC multiple classification model reported below held up to these sensitivity analyses. Full details of analyses are reported in the supporting information (SI Sensitivity/Robustness Analyses, available as [Supplementary data](#) at *IJE* online). We used two Stata computational modules, `ie_rate`²⁵ and `apc_ie`,²⁶ to estimate the model. Results from these two programs are consistent.

To interpret the APC-IE coefficients, note that they do not represent actual mortality rates of a certain age, period or cohort, but rather patterns of estimated effects for each temporal dimension (that is, net of the effects of the other two temporal dimensions). We obtain three sets of single-year coefficients for age, period and cohort, respectively, from our estimation. The incident rate ratio (IRR) with regard to the reference category can be calculated from the exponential of the coefficient. The reference category is the mean effect for all ages, periods or cohorts combined. A negative coefficient leads to an IRR below 1, and a positive coefficient gives an IRR above 1. For example, an IRR = 1.5 for a certain group means that this group is 50% more likely to die than the reference category.

Results

We first present the inter-cohort patterns in the absolute changes of death rates by gender and race/ethnicity in [Figure 1](#). Interesting racial/ethnic disparities in inter-cohort mortality patterns exist. Among females, compared with other cohorts in each race/ethnicity group, non-Hispanic White late-Gen Xers and early-Gen Yers have higher death rates, whereas non-Hispanic Black and Hispanic early- and late-Baby Boomers have higher death rates. Among males, compared with other cohorts in each race/ethnicity group, early- and late-Baby Boomers have higher death rates

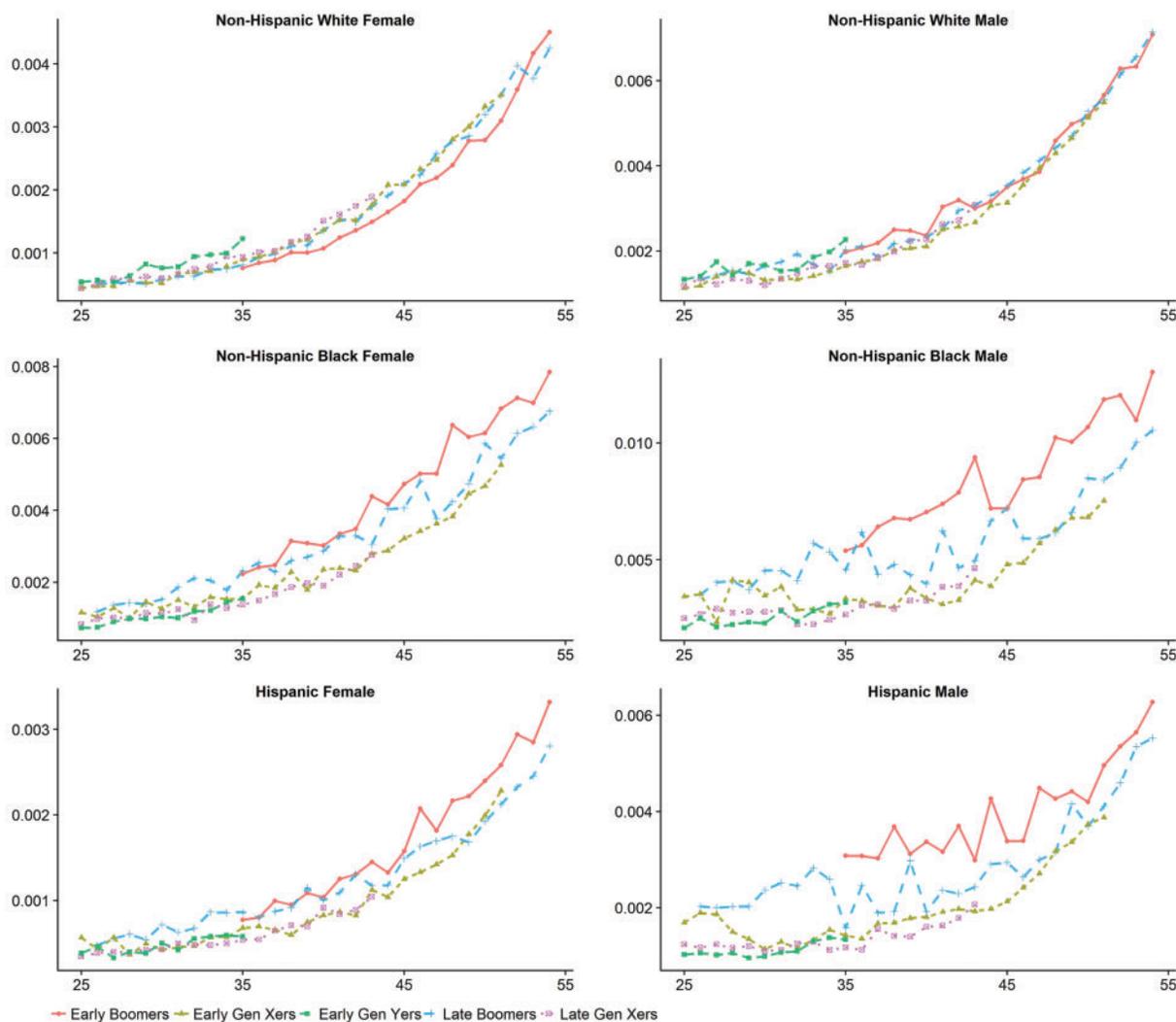


Figure 1. Inter-cohort patterns of age-specific death rates by gender and race/ethnicity for ages 25–54.

across all races, but the higher death rate for early-Gen Yers is also very noticeable among non-Hispanic Whites.

These inter-cohort mortality patterns can be driven by both period forces and cohort forces. Period effects refer to the effects of temporal social contexts on all age groups simultaneously, whereas cohort effects refer to the effects of unique early life experiences that are common to each specific birth cohort.²⁷ To disentangle these two forces, we first examine the period trends independent of age and cohort effects. Figure 2 presents graphed single-year coefficients of the estimated period effects for all-cause mortality, for each of the age groups 25–34, 35–44 and 45–54, holding the variation associated with age and cohort constant. Consistent reductions in Hispanic and non-Hispanic Black mortality rates since the early and middle 1990s are evident for all three age groups, net of the age and cohort effects. However, the pattern for non-Hispanic Whites is distinct. Net of the estimated age and cohort

effects, mortality rates in early and middle adulthood (ages 25–34 and 35–44 panels) have increased since the 1990s among non-Hispanic White females. By contrast, the period patterns in early and middle adulthood for non-Hispanic White males show declines in the mid-1990s and then show increases since the late 1990s, followed by relatively stable trends since the early 2000s. For ages 45–54, the estimated period effect coefficients for non-Hispanic White females show increases in the 1990s followed by a levelling off in the 2000s. Patterns for non-Hispanic White males show slight declines in the mid-1990s and then slight increases since the late 1990s, followed by a levelling off since the early 2000s.

The period trends suggest that period forces have likely contributed to the observed inter-cohort mortality patterns in Figure 1. For non-Hispanic Blacks and Hispanics, the period coefficients are the highest in the early 1990s, which corresponds to the time when Baby Boomers were in their

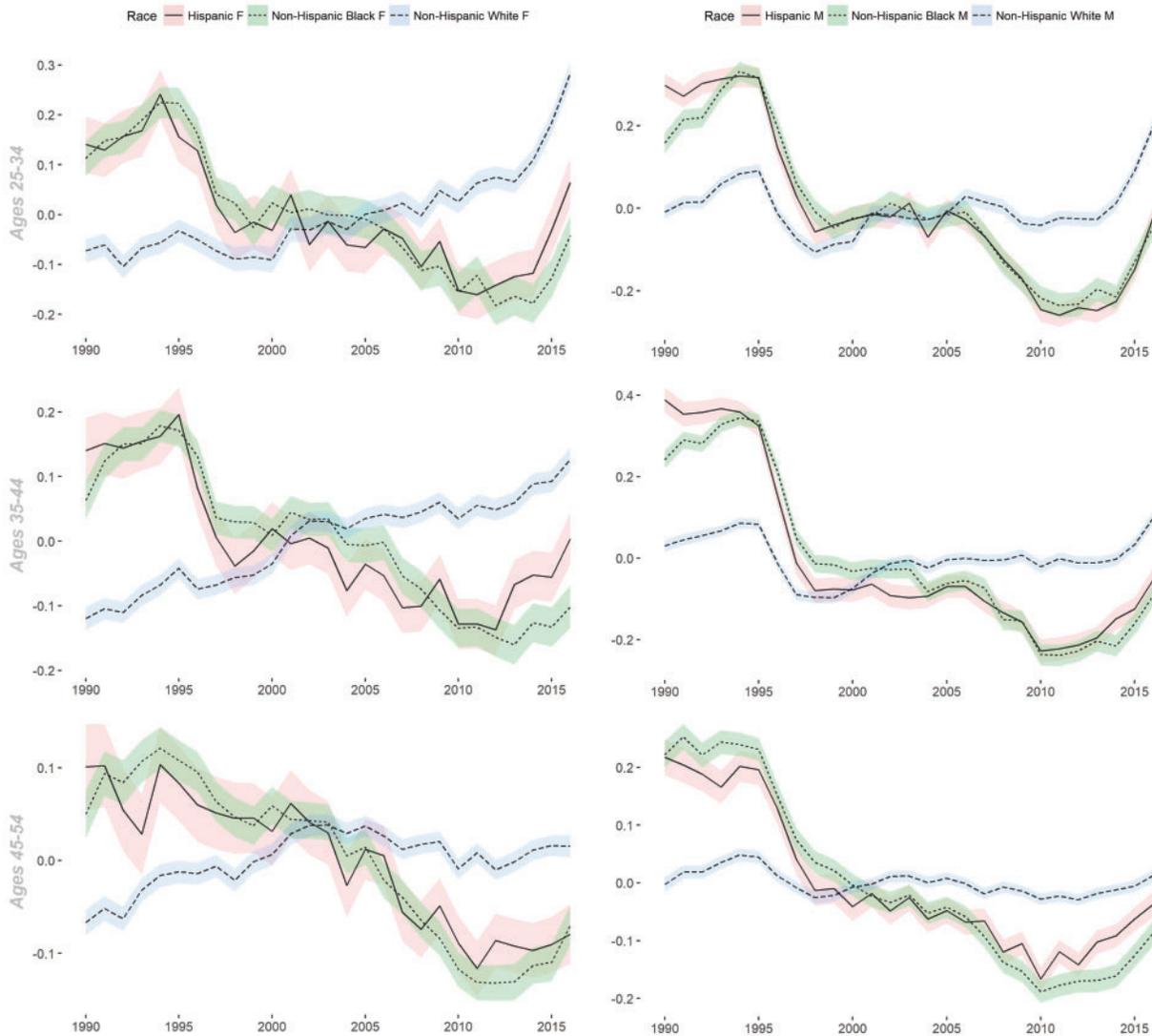


Figure 2. Estimated period effect coefficients for all-cause mortality by gender and race/ethnicity for age groups 25–34, 35–44 and 45–54, with 95% confidence intervals.

early and middle adulthood (ages 25–54). For non-Hispanic White males, some of the highest period coefficients appear around the mid-1990s, which corresponds to the higher death rates among early-Baby Boomers at ages 35–44 in [Figure 1](#). The sharp increase of period coefficients in the mid-2010s is consistent with the higher death rates among early-Gen Yers at ages 25–34. For non-Hispanic White females, the increasing period coefficients over time are consistent with the observed higher death rates among late-Gen Xers and early Gen-Yers at ages 25–44. The increases during the late 1990s in the estimated period trends among non-Hispanic Whites in early and middle adulthood (ages 25–54) coincide with the timing of the onset of the opioid epidemic.

Next, we examine the estimated cohort patterns net of age and period effects. [Figure 3](#) shows the single-year cohort estimates by race/ethnicity and gender. A reorganization of the results for each of the 10-year age groups

25–34, 35–44 and 45–54, can be found in the supporting information ([Supplementary Figure 4](#), available as [Supplementary data](#) at *IJE* online). For non-Hispanic White males, non-Hispanic Blacks, and Hispanics, despite some small differences between age groups, we generally see an increase in estimated cohort coefficients among early-Baby Boomers (born 1946–55) and then a decrease among late-Baby Boomers (born 1956–64) and early-Gen Xers (born 1965–72). By contrast, for non-Hispanic White females, cohort coefficients continuously increase from early- to late-Boomers and decrease among early-Gen Xers (born 1965–72). Cohort coefficients then increase again among late-Gen Xers (born 1973–80) in all sex and race groups. Among the early-Gen Yers (born 1981–90), cohort coefficients decline slightly among non-Hispanic Blacks, whereas they increase slightly among non-Hispanic Whites and Hispanics.

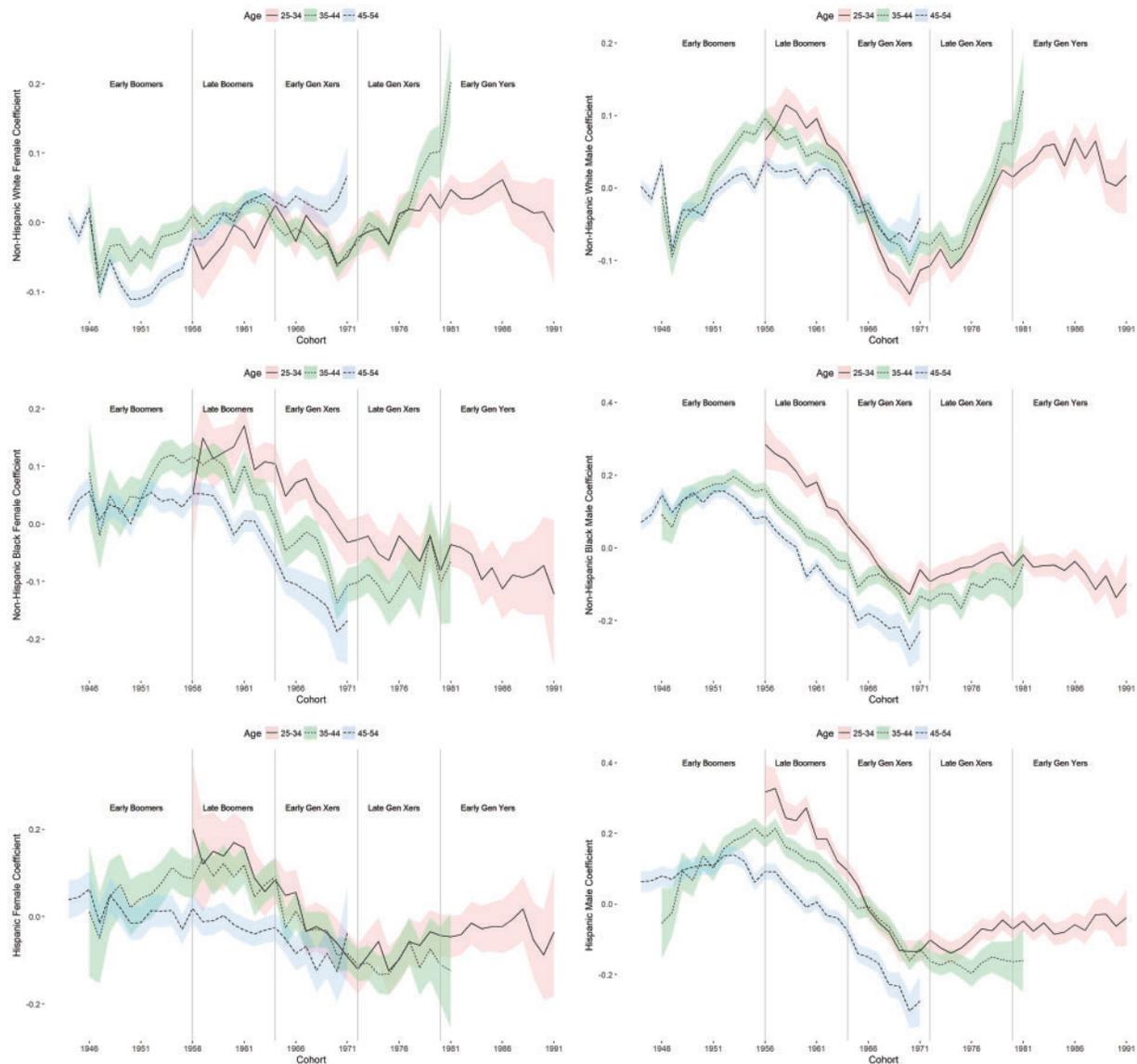


Figure 3. Estimated cohort effect coefficients for all-cause mortality by gender and race/ethnicity for age groups 25–34, 35–44 and 45–54, with 95% confidence intervals.

Consistent with the observed patterns in Figure 1, for non-Hispanic Blacks and Hispanics, the cohort coefficients are the highest among Baby Boomers. We also observe cohort-based mortality increases among non-Hispanic Black and Hispanic late-Gen Xers. For non-Hispanic White males, the cohort coefficients are the highest among Baby Boomers and early-Gen Yers. However, the cohort coefficients have already increased sharply among late-Gen Xers. For non-Hispanic White females, the highest cohort coefficients appear among late-Gen Xers and early-Gen Yers. The cohort effect trend also increases among Baby Boomers, but the magnitude of the increase seems to be smaller than that among late-Gen Xers and early-Gen Yers.

Our cohort-based mortality results show substantial race/ethnic disparities in inter-cohort mortality patterns.

First, cohort-based mortality is the highest among Baby Boomers for non-Hispanic Blacks and Hispanics, which is mainly driven by the increase in cohort-based mortality among early-Baby Boomers. For non-Hispanic White females, by contrast, cohort-based mortality is the highest among late-Gen Xers and early-Gen Yers, which is mainly driven by the sharp increase in cohort-based mortality among late-Gen Xers. For non-Hispanic White males, cohort-based mortality is the highest among Baby Boomers, late-Gen Xers and early-Gen Yers, which is mainly driven by increase in cohort-based mortality among early-Baby Boomers and late-Gen Xers. Generally speaking, it appears that Baby Boomers, late-Gen Xers and early-Gen Yers across all race and gender groups have increased mortality patterns at least sometime in early or middle adulthood.

To explore the potential-driven factors of the mortality trends among our identified three cohorts, we investigate cohort components in cause-specific mortality trends using nine leading causes: drug poisonings, suicides and alcohol-related diseases, heart disease and stroke, HIV/AIDS, diabetes mellitus, external causes, COPD and cancer. Our choice of these nine causes is based on recent literature on their important roles in mortality levels or mortality increases.^{1,7,28–30} Due to page limits, we only present results of causes with increasing cohort effect trends among Baby Boomers, late-Gen Xers and early-Gen Yers. Their corresponding period trends are available in the supporting information (Supplementary Figure 5, available as Supplementary data at *IJE* online). Three main findings are especially noteworthy from our investigation.

First, among the nine causes of investigation, drug poisonings, suicides, COPD, external causes and HIV/AIDS-related mortality are the five leading causes for the elevated all-cause mortality trends among Baby Boomers across all gender and race/ethnicity groups. Cohort effect trends decrease or stay at a low level among Baby Boomers for the remaining four causes of death. To provide a general picture of this finding, Figure 4 shows estimates of the cohort coefficients for the five causes (drug poisonings, suicides, COPD, external causes and HIV/AIDS) by gender with all races/ethnicities combined. For female Baby Boomers, the increases of cohort coefficients for mortality related to HIV/AIDS and COPD are particularly sharp, whereas the increases are relatively minor for the other three causes. For male Baby Boomers, besides HIV/AIDS and COPD, the increase of cohort coefficients for mortality related to drug poisoning is also noticeable.

Second, among the nine causes of investigation, racial/ethnic disparities exist in the leading causes among late-

Gen Xers and early-Gen Yers. Figure 5 shows the estimated cohort coefficients of causes with increasing mortality trends among late-Gen Xers and early-Gen Yers by race/ethnicity and gender. For non-Hispanic Whites, drug poisonings and alcohol-related diseases are the leading causes. For Hispanics, drug poisonings and suicides are the leading causes. Whereas the leading causes are the same for males and females among non-Hispanic Whites and Hispanics, the leading causes for non-Hispanic Black males and females are different. For non-Hispanic Black females, diabetes-related mortality is the only one that has an increasing trend among late-Gen Xers and early-Gen Yers. For non-Hispanic Black males, the corresponding leading causes are cancer, external causes and alcohol-related diseases.

Third, race/ethnic disparities in inter-cohort mortality patterns documented in this study are at least partially driven by differential drug poisoning mortality patterns. This statement applies to both the differential cohort patterns between non-Hispanic White female and other Baby Boomers and the differential cohort patterns between non-Hispanic White and other late-Gen Xers and early-Gen Yers. Figure 6 shows estimated cohort coefficients of drug poisoning mortality by race/ethnicity and gender. Among Baby Boomers, the increase of cohort coefficients for non-Hispanic White females is much smaller compared with the other groups. Among late-Gen Xers and early-Gen Yers, the increases of cohort coefficients for non-Hispanic Whites are much larger compared with the other groups.

In sum, our cohort-based mortality results show substantial racial/ethnic disparities in inter-cohort mortality patterns. For non-Hispanic Blacks and Hispanics, Baby Boomers have elevated mortality trends compared with

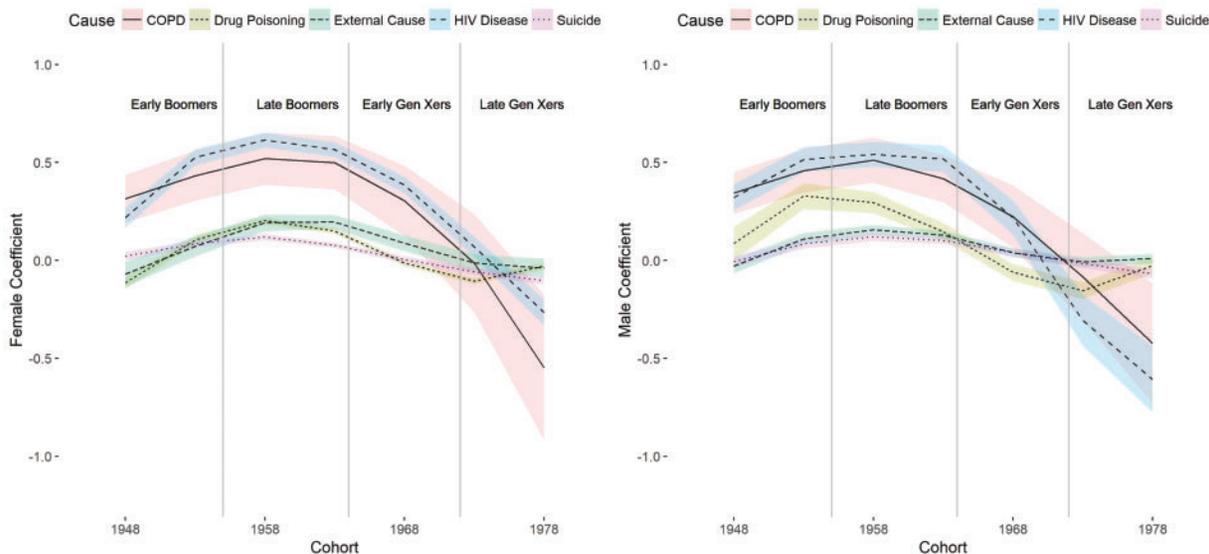


Figure 4. Estimated cohort effect coefficients for Baby Boomers by gender and cause, with 95% confidence intervals.

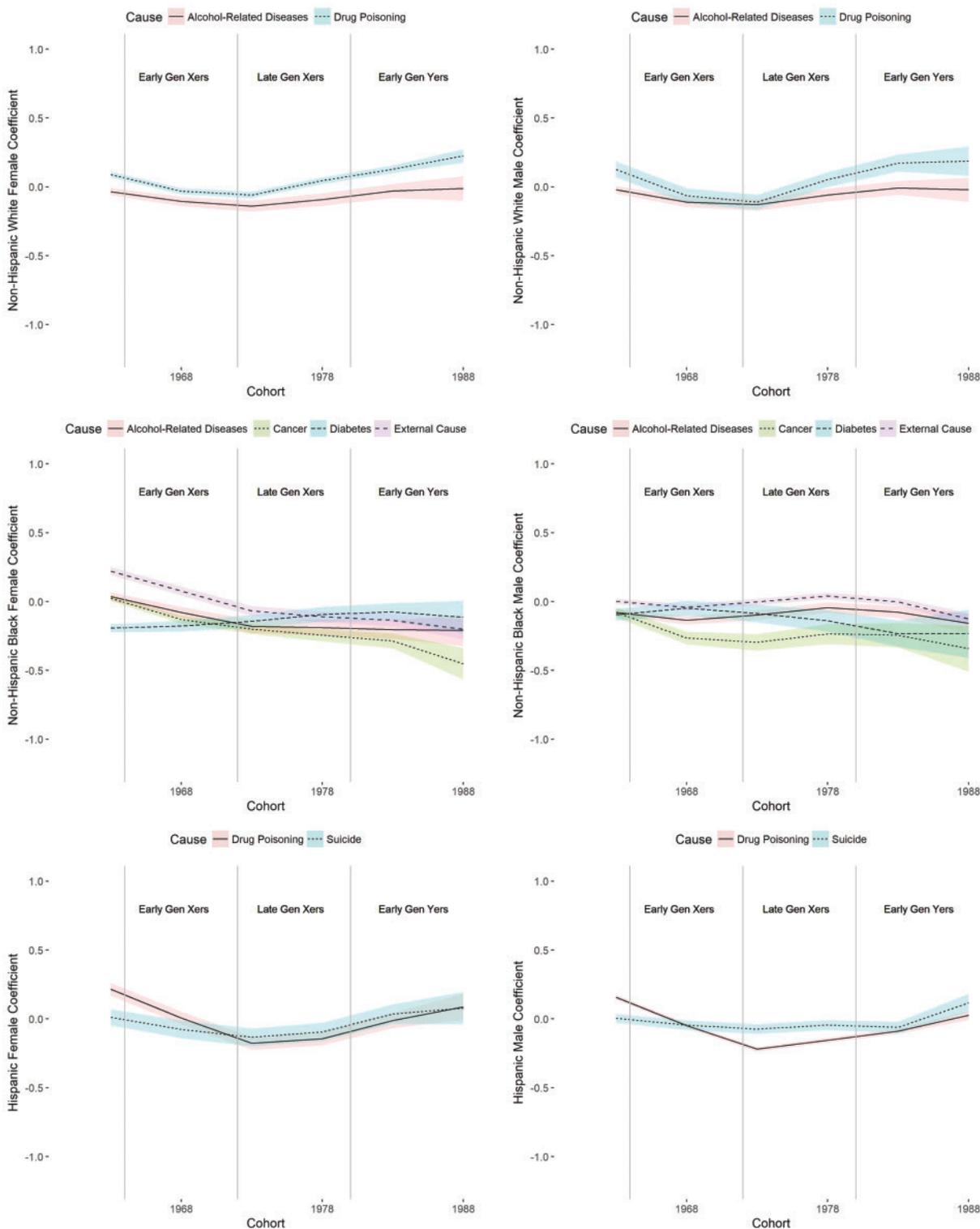


Figure 5. Estimated cohort effect coefficients for Gen Xers by race/ethnicity, gender and cause, with 95% confidence intervals.

other cohorts. For non-Hispanic White females, late-Gen Xers and early-Gen Yers have elevated mortality trends compared with other cohorts. For non-Hispanic White males, Baby Boomers, late-Gen Xers and early-Gen Yers have higher mortality trends. The increased mortality

pattern among Baby Boomers is at least partially driven by mortality related to drug poisoning, suicide, external causes, COPD and HIV/AIDS for all race and gender groups affected. The mortality patterns among late-Gen Xers and early-Gen Yers are at least partially driven by

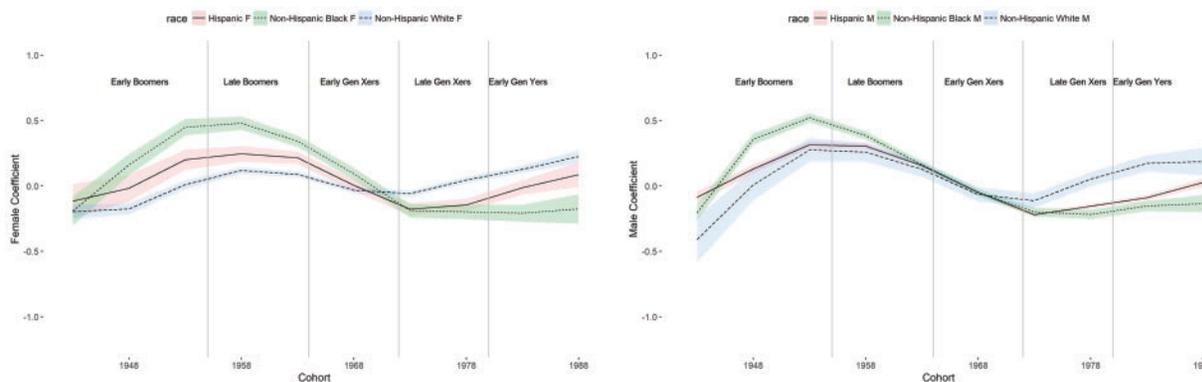


Figure 6. Estimated cohort effect coefficients for drug poisoning mortality by race/ethnicity and gender, with 95% confidence intervals.

mortality related to drug poisonings and alcohol-related diseases for non-Hispanic Whites. Differential patterns of drug poisoning-related mortality play an important role in the racial /ethnic disparities in these mortality patterns.

Discussion

Our findings indicate that drug poisonings, external causes, suicides, COPD and HIV/AIDS have contributed to the increase in cohort-based mortality trends among Baby Boomers across all race/ethnicity and gender groups. The important roles of HIV/AIDS, suicides and drug poisonings in the elevated mortality pattern among Whites have been emphasized by previous studies.^{1,7} However, the important roles of external causes and COPD in this mortality trend among Baby Boomers across all race groups have not been much discussed in existing literature.

One of the most important new findings in our analyses is that we identified late-Gen X and early-Gen Y as generations with elevated mortality patterns in addition to the already much discussed Baby Boomer generation, particularly for non-Hispanic Whites. Late-Gen Xers and early-Gen Yers, born in 1973–91, were aged 25–43 during the period covered in our sample. Different from Baby Boomers who were hit by the Great Recession in midlife, late-Gen Xers and early-Gen Yers were hit in early adulthood. According to a poll done by the Pew Research Center, compared with the generations that preceded them, Gen Xers showed the most dramatic decrease in financial satisfaction and confidence in retirement savings.³¹ Gen Xers were also hit the hardest by the housing crisis of the late 2000s, with the highest percentage of mortgages greater than the current market values of the property.³¹ Because of the Great Recession and its aftermath in the 2008–16 years, young adults (late-Gen Xers and early-Gen Yers) had a particularly hard time finding jobs.³¹ These all point to the unique challenges faced by young adult late-Gen Xers and early-Gen Yers.

However, these economic disadvantages alone cannot explain the racial/ethnic differences in inter-cohort mortality patterns. The underlying causes for increasing cohort-based mortality rates seem to be very different across races/ethnicities. For non-Hispanic Whites and to some extent Hispanics, drug poisoning mortality contributed significantly to the increasing mortality pattern among these generations. But for non-Hispanic Blacks, drug poisoning mortality does not seem to play a role among these generations. Instead, cancer, external causes and alcohol-related diseases are relevant causes for non-Hispanic Black males, whereas diabetes is a relevant cause for non-Hispanic Black females. The reasons for these race/ethnic differences are still unclear, but they are very unlikely to be driven only by differential cumulative disadvantages triggered by progressively worsening labour market opportunities at the time of entry, as proposed by previous studies.^{1,2} Here we discuss three alternative possibilities.

First, the differences may reflect differential exposures to opioid prescriptions between non-Hispanic Blacks and other races. There are two potential reasons for these differential exposures across races. First, non-Hispanic Blacks may have disadvantages in access to health care compared with other race groups.³² The limitation in access to health care may lead to relatively limited access to prescription drugs among non-Hispanic Blacks. Second, due to racial bias in pain perception among medical practitioners, non-Hispanic Black patients with pain may be treated differently from non-Hispanic Whites.^{33,34} For example, a non-Hispanic White patient may be more likely to obtain prescription drugs than a non-Hispanic Black patient during a pain-related health care visit.^{34,35}

Second, it may reflect racial disparities in the prevalence of obesity in the USA. Obesity is considered one of the major forces driving up US mortality.^{28,30} Existing evidence shows that Blacks, particularly Black females, have higher risks of obesity compared with other races in the USA.^{36–38} This is also reflected in our finding that diabetes is one

leading cause for the elevated mortality among non-Hispanic Black female late-Gen Xers.

Third, it may reflect racial disparities in the prevalence of smoking in the USA. Smoking is also one of the major forces driving up US mortality.²⁸ It has been found to contribute to higher risks of diabetes, cancer and accidental death.^{39,40} Smoking is often a way to get relaxed among low-income individuals with stress,⁴¹ and has a particularly high prevalence among Blacks. Existing evidence shows that smoking has contributed significantly to the Black-White life expectancy gap among old-age males.⁴² It is likely that smoking has contributed to the increasing mortality pattern caused by cancer, external causes and diabetes among non-Hispanic Black younger generations.

Looking forward, late-Gen Xers and early-Gen Yers may continue to face big economic challenges. The conditions behind the mortality patterns for these cohorts, such as stress, pain and obesity, may exert a heavier burden of other related conditions (e.g. disability, depression), which will put additional costs on US Social Security and Medicare systems during the dependent years of these two generations. Social scientists and policy makers are aware of the financial burden for the younger generation, but the elevated mortality rate among them has largely been ignored, which will further cloud the economic and health care prospects. This trend merits additional attention and effective policy responses.

Although this paper studies US mortality trends, the results also provide important implications for other countries that are having issues of drug poisoning, obesity and smoking. Substantial increases in drug poisoning, smoking and obesity prevalence and mortality have been found in many countries throughout the world over the past decade.^{43–47} As a country with relatively high levels of drug poisoning, obesity and smoking-related mortality, research findings for the US may contribute to an understanding of how socioeconomic conditions affect mortality patterns related to these causes in other societal contexts.

Supplementary Data

Supplementary data are available at *IJE* online.

Acknowledgements

We thank Jarron M Saint Onge, Jessica Y Ho and Scott M Lynch for helpful comments and discussions. Versions of this article were presented at the Population Association of America Annual Meeting, Chicago, IL, 26–29 April 2017, and at the American Sociological Association Annual Meeting, Montreal, QC, 12–15 August 2017.

Conflict of interest: None declared.

References

- Case A, Deaton A. Rising morbidity and mortality in midlife among white non-Hispanic Americans in the 21st century. *Proc Natl Acad Sci U S A* 2015;**112**:15078–83.
- Case A, Deaton A. Mortality and morbidity in the 21st century. *Brookings Pap Econ Act*, 2017;2017:397–476.
- Snyder SE. Urban and rural divergence in mortality trends: a comment on Case and Deaton. *Proc Natl Acad Sci U S A* 2016;**113**:E815.
- Gelman A, Auerbach J. Age-aggregation bias in mortality trends. *Proc Natl Acad Sci U S A* 2016;**113**:E816.
- Case A, Deaton A. Reply to Schmid, Snyder, and Gelman and Auerbach: correlates of the increase in white non-Hispanic mid-life mortality in the 21st century. *Proc Natl Acad Sci U S A* 2016;**113**:E818–19.
- Schmid CH. Increased mortality for white middle-aged Americans not fully explained by causes suggested.
- Masters RK, Tilstra AM, Simon DH. Explaining recent mortality trends among younger and middle-aged White Americans. *Int J Epidemiol* 2018;**47**:81–8.
- Masters RK, Tilstra AM, Simon DH. Mortality from suicide, chronic liver disease, and drug poisonings among middle-aged US white men and women, 1980–2013. *Biodemography Soc Biol* 2017;**63**:31–37.
- Meara E, Skinner J. Losing ground at midlife in America. *Proc Natl Acad Sci U S A* 2015;**112**:15006–67.
- Ho JY. Mortality under age 50 accounts for much of the fact that US life expectancy lags that of other high-income countries. *Health Aff (Millwood)* 2013;**32**:459–67.
- Arias E, Schauman WS, Eschbach K, Sorlie PD, Backlund E. The validity of race and Hispanic origin reporting on death certificates in the United States. *Vital Health Stat* 2008;(148):1–23.
- Sloan FA, Ayyagari P, Salm M, Grossman D. The longevity gap between black and white men in the United States at the beginning and end of the 20th century. *Am J Public Health* 2010;**100**:357–63.
- Levine RS, Foster JE, Fullilove RE *et al*. Black-white inequalities in mortality and life expectancy, 1933–1999: implications for healthy people 2010. *Public Health Rep* 2001;**116**:474–83.
- Masters RK, Hummer RA, Powers DA. Educational differences in US adult mortality. *Am Sociol Rev* 2012;**77**:548–72.
- Elo IT, Drenvestedt GL. Cause-specific contributions to black-white differences in male mortality from 1960 to 1995. *Demogr Res* 2004;**2**:255–76.
- Satcher D, Fryer GE, McCann J, Troutman A, Woolf SH, Rust G. What if we were equal? A comparison of the black-white mortality gap in 1960 and 2000. *Health Aff (Millwood)* 2005;**24**:459–64.
- Arias E. *United States Life Tables, 2010*. Hyattsville, MD: National Center for Health Statistics, 2014.
- Yang Y, Fu WJ, Land KC. A methodological comparison of age-period-cohort models: the intrinsic estimator and conventional generalized linear models. *Sociol Methodol* 2004;**34**:75–110.
- Yang Y, Schulhofer-Wohl S, Fu WJ, Land KC. The intrinsic estimator for age-period-cohort analysis: what it is and how to use it. *Am J Sociol* 2008;**113**:1697–736.
- Yang Y, Land KC. *Age-Period-Cohort Analysis: New Models, Methods, and Empirical Applications*. Boca Raton, FL: Chapman & Hall/CRC, 2013.

21. Kupper LL, Janis JM, Karmous A, Greenberg BG. Statistical age-period-cohort analysis: a review and critique. *J Chronic Dis* 1985;38:811–30.
22. Masters RK, Hummer RA, Powers DA, Beck A, Lin S-F, Finch BK. Long-term trends in adult mortality for US blacks and whites: an examination of period-and cohort-based changes. *Demography* 2014;51:2047–73.
23. Masters RK, Powers DA, Hummer RA, Beck A, Lin S-F, Finch BK. Fitting age-period-cohort models using the Intrinsic Estimator: assumptions and misapplications. *Demography* 2016;53:1253–59.
24. Land KC, Fu Q, Guo X, Jeon SY, Reither EN, Zang E. Playing with the rules and making misleading statements: a response to Luo, Hodges, Winship, and Powers. *Am J Sociol* 2016;122:962–73.
25. Powers DA. IE_RATE: Stata module to conduct age, period, and cohort (APC) analysis of tabular rate data using the intrinsic estimator. Statistical Software Components S457445, Boston College Department of Economics, 2014.
26. Schulhofer-Wohl S, Yang Y. APC: Stata module for estimating age-period-cohort effects. Statistical Software Components S456754, Boston College Department of Economics, 2006.
27. Yang Y. Trends in US adult chronic disease mortality, 1960–1999: age, period, and cohort variations. *Demography* 2008;45:387–416.
28. Preston SH, Stokes A, Mehta NK, Cao B. Projecting the effect of changes in smoking and obesity on future life expectancy in the United States. *Demography* 2014;51:27–49.
29. Ma J, Ward EM, Siegel RL, Jemal A. Temporal trends in mortality in the United States, 1969–2013. *JAMA* 2015;314:1731–39.
30. Preston SH, Vierboom YC, Stokes A. The role of obesity in exceptionally slow US mortality improvement. *Proc Natl Acad Sci* 2018;115:957–61.
31. Kohut A, Taylor P, Keeter S, Doherty C, Dimock M, Parker K. *The Generation Gap and the 2012 Election*. Washington, DC: Pew Research Center, 2011.
32. U.S. Department of Health & Human Services. *Disparities in Healthcare Quality Among Racial and Ethnic Groups - Selected Findings From the 2011 National Healthcare Quality and Disparities Reports*. 2011. <https://archive.ahrq.gov/research/findings/nhqrdr/nhqrdr11/minority.html> (11 October 2018, date last accessed).
33. Trawalter S, Hoffman KM, Waytz A. Racial bias in perceptions of others' pain. *PLoS One* 2012;7:e48546.
34. Hoffman KM, Trawalter S, Axt JR, Oliver MN. Racial bias in pain assessment and treatment recommendations, and false beliefs about biological differences between blacks and whites. *Proc Natl Acad Sci US A* 2016;113:4296–301.
35. Pletcher MJ, Kertesz SG, Kohn MA, Gonzales R. Trends in opioid prescribing by race/ethnicity for patients seeking care in US emergency departments. *JAMA* 2008;299:70–78.
36. Wang Y, Beydoun MA, Liang L, Caballero B, Kumanyika SK. Will all Americans become overweight or obese? Estimating the progression and cost of the US obesity epidemic. *Obesity (Silver Spring)* 2008;16:2323–30.
37. Wang Y, Beydoun MA. The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiol Rev* 2007;29:6–28.
38. Wang L, Southerland J, Wang K *et al*. Ethnic differences in risk factors for obesity among adults in California, the United States. *J Obes* 2017;2017:1.
39. Leistikow BN, Martin DC, Jacobs J, Rocke DM, Noderer K. Smoking as a risk factor for accident death: a meta-analysis of cohort studies. *Accid Anal Prev* 2000;32:397–405.
40. US Department of Health Human Services. *The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General*. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2014.
41. Lutfey K, Freese J. Toward some fundamentals of fundamental causality: Socioeconomic status and health in the routine clinic visit for diabetes. *Am J Sociol* 2005;110:1326–72.
42. Ho JY, Elo IT. The contribution of smoking to black-white differences in US mortality. *Demography* 2013;50:545–68.
43. Martins SS, Sampson L, Cerdá M, Galea S. Worldwide prevalence and trends in unintentional drug overdose: a systematic review of the literature. *Am J Public Health* 2015;105:e29–49.
44. Wang Y, Lobstein T. Worldwide trends in childhood overweight and obesity. *Int J Pediatr Obes* 2006;1:11–25.
45. Ng M, Fleming T, Robinson M *et al*. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 2014;384:766–81.
46. Jha P, Ranson MK, Nguyen SN, Yach D. Estimates of global and regional smoking prevalence in 1995, by age and sex. *Am J Public Health* 2002;92:1002–06.
47. Ng M, Freeman MK, Fleming TD *et al*. Smoking prevalence and cigarette consumption in 187 countries, 1980–2012. *JAMA* 2014;311:183–92.