The Growing Divide: How Cohort Processes Affect Educational Differences in Black and White U.S. Mortality Risk.

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Overview

Educational differences in U.S. adult mortality risk have been widely studied in population sciences. For the most part, research has shown a strong educational gradient in U.S. adult mortality risk, but also has shown that the gradient differs for men and women and for race/ethnic groups (Cutler et al. 2010; Montez et al. 2009; Hummer and Lariscy 2010). Further, evidence increasingly suggests that educational gaps in U.S. adult mortality risk are growing with time (Cutler et al. 2010; Hummer and Lariscy 2010). Unfortunately, the majority of work on temporal changes to educational differences in mortality risk simply document period-based changes to age-specific mortality and/or life expectancy. Only minimal effort has been made to develop a comprehensive theory to explain (1) educational gaps in mortality risk, (2) differences in these gaps by race/ethnicity and gender, and (3) historical changes therein. In this paper we advance a cohort perspective to explain why the association between educational attainment and mortality risk is changing, and why we expect disparate changes in this association by sex and race/ethnicity. We draw from both the fundamental cause theory (Link and Phelan 1995) and the life course perspective (Ben-Shlomo and Kuh 2002; Montez and Hayward 2010), but couch these perspectives in a cohort framework in order to better understand trends in U.S. adult mortality. We then examine age, period, and cohort patterns of educational differences in U.S. adult mortality risk for non-Hispanic black and non-Hispanic white male and female populations during the time period 1986 through 2006. We hypothesize that (1) educational differences in U.S. adult mortality risk are growing, (2) educational differences in mortality risk are growing across cohorts, not periods, and (3) the cohort processes driving educational differences in mortality affect sex and race/ethnicity groups in the United States differently. Regarding this latter hypothesis, we specifically hypothesize (4) that educational gaps in U.S. mortality risk are growing more substantially for non-Hispanic white men and women than for non-Hispanic black men and women. To test our hypotheses we employ recently developed hierarchical age-period-cohort (HAPC) cross-classified random effects models (CCREM) to simultaneously examine age, period, and cohort patterns of educational differences in all-cause, heart disease, and non-lung cancer U.S. adult mortality risk. Analyses are stratified by sex and race/ethnicity to test whether or not educational gradients in mortality risk differ for U.S. non-Hispanic black and non-Hispanic white men and women. Results provide evidence that is overwhelmingly supportive of all four hypotheses.

Data

We use the National Health Interview Survey (NHIS) from years 1986 to 2004, linked with the individual-level cause-of-death mortality records from the 1986 to 2006 National Death Index (NDI), to analyze age-period-cohort (APC) patterns of sex-specific U.S. adult mortality risks by race/ethnicity and educational attainment between 1986 and 2006. These National Health Interview Survey-Linked Mortality Files (NHIS-LMF) provide a unique data structure that combines repeated cross-sectional waves with individual-level longitudinal status of mortality. The individual-level NHIS-LMF data were aggregated into five-year age-period-cohort blocks, and were collapsed by sex, race/ethnicity, and educational attainment (less than 12 years, high school graduate, and some college & above). These collapsed data provide sufficient cases of both cell-specific observations and cell-specific mortality outcomes to produce stable and consistent mortality estimates for nearly all possible five-year age-period-cohort combinations.

Methods

We use recently developed hierarchical age-period-cohort (HAPC) models for repeated cross-sectional survey data (Yang and Land 2006). These methods utilize a cross-classified random effects model (CCREM) to embed each respondent within both a five-year time period and birth cohort at a given five-year age group. Aggregated counts of deaths as well as aggregated person-years lived across the five-year age-period-cohort cell's time frame were used to compute five-year age-specific mortality rates for each education subsample. The HAPC-CCREM estimates fixed effects of the five-year age groups and random effects of the five-year period and five-year cohort groups, and is structured in the following way:

Level-1 within cell model: $$\ln(D_{ijk}) = \alpha_{jk} + \beta_{jk} A_i + \ln(\exp_{ijk}) + e_{ijk}$$

where D_{ijk} stands for the counts of deaths of the ith age group for $i = 1, \ldots, n_{jk}$ age groups within the jth period for $j = 1, \ldots, J$ time period and the kth cohort for $k = 1, \ldots, K$ birth cohort; A_i denotes the dummy five-year age groups $1,\ldots, n_{jk}$; α_{jk} is the intercept indicating the reference age group [65-69) who was in period j and belong to cohort k; $\ln(\exp_{ijk})$ is the natural log of the aggregated exposure time lived during the five-year age-period-cohort cell; and e_{ijk} is the random cell residual.

Level-2 between cell random intercept model: $$\alpha_{jk} = \pi_0 + t_{0j} + c_{0k}$$

in which α_{jk} specifies that the fixed age effects vary from period to period and from cohort to cohort. π_0 is the expected mean at the reference age [65-69) averaged over all periods and cohorts; t_{0j} is the overall 5-year period effect averaged over all five-year birth cohorts with variance σ_{t0}; and c_{0k} is the overall 5-year cohort effect averaged over all five-year periods with variance σ_{k0}.

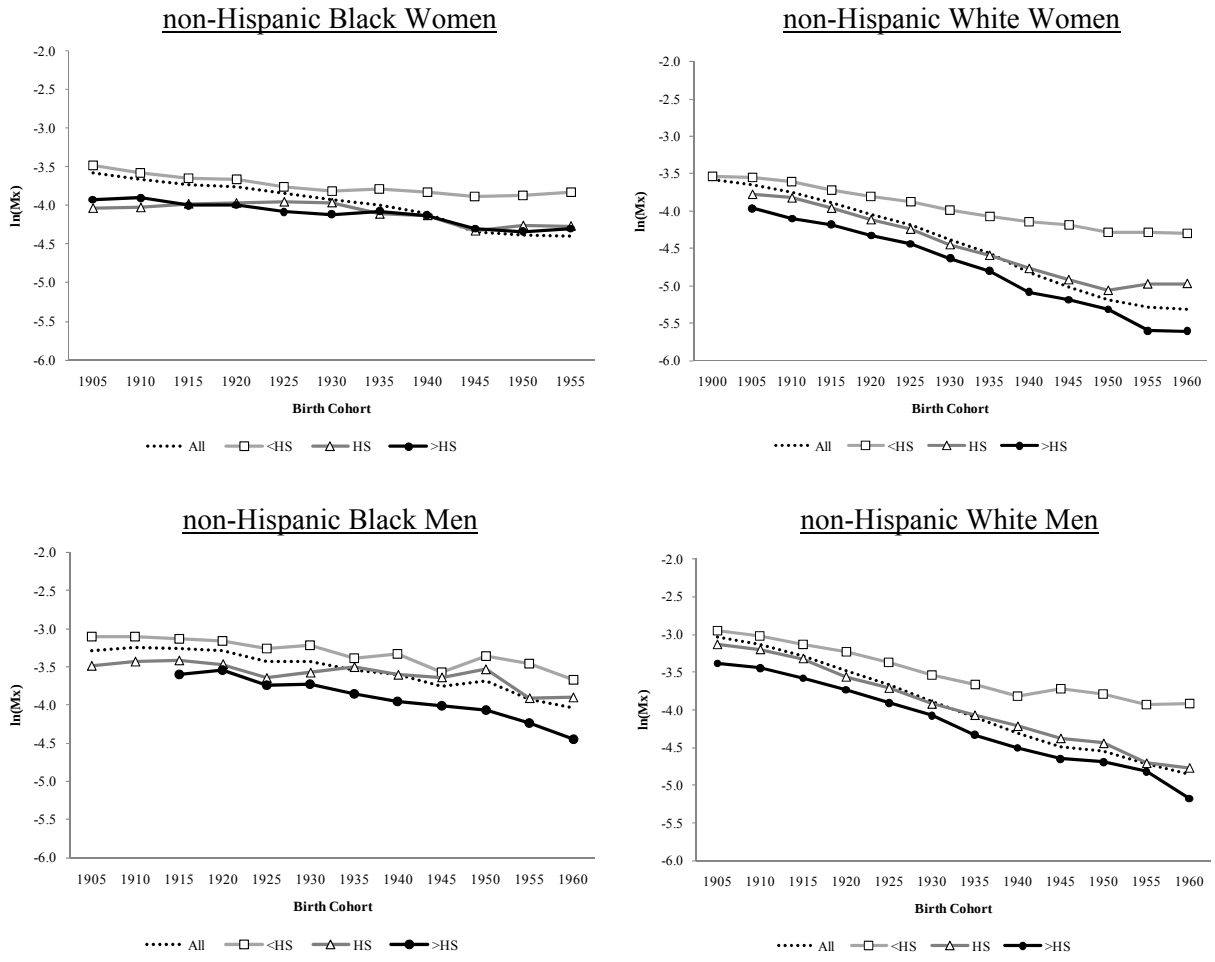
We combine the level-1 and level-2 models to estimate counts of deaths in each 5-year age-period-cohort cell using SAS PROC GLIMMIX with a log-linear Poisson family, offsetting the aggregated person-years lived across the cells to generate age-period-cohort specific mortality rates. Models are stratified by educational attainment, sex, and race/ethnicity.

Preliminary Results

Due to space limitations, only the random cohort effects for the all-cause mortality analyses are presently discussed. To be sure, estimated fixed age effects and estimated random period effects are both consistent with past research (results not shown) (Yang 2008). Also due to space limitations, tables of estimated fixed and random effects are not shown. Results are presented in graphical form in Figure 1 and Figure 2.

Figure 1 shows cohort trends in educational differences in fitted M_xs for the U.S. non-Hispanic black and non-Hispanic white male and female adult populations. Figure 2 displays the same results, but contrasts race/ethnic differences in the cohort trends within respective education levels. Evidence within these results provides support for all four of our hypotheses. First, educational differences in M_xs grew across the time period 1986-2006 for all four population groups. Second, period effects were found to be both statistically insignificant and substantively small, while cohort effects were found to be substantively large and statistically significant. For example, the all-education HAPC-CCREM analysis for U.S. non-Hispanic white female mortality risk produced a cohort covariance parameter of .409 (t-value of 2.33), whereas the respective period covariance parameter was estimated to be .061 (t-value of 1.23). Thus, the vast majority of the reduction in U.S. adult mortality risk between 1986 and 2006 was due to cohort processes. Third, both the *size* of the educational gradient in U.S. adult mortality risk and the *cohort changes* to the gradient differ by sex and race/ethnic group. Regarding size, we see the smallest educational gradient in mortality in the non-Hispanic black female population, and the largest educational gradient in the non-Hispanic white female population. Regarding temporal change, all four subpopulations' education gaps in mortality risk grew across cohorts. However, the degree to which these gaps widened differ by race/ethnicity.

Figure 1: Educational Differences in All-cause U.S. Adult Mortality Risk, 1986-2007, Across Cohorts, by Sex and Race/Ethnic Group.



Consistent with our fourth hypothesis, the smallest change occurred in the non-Hispanic black female population, whereas the largest change occurred in both the male and female non-Hispanic white population. Indeed, for the non-Hispanic black female population we see both the smallest gradient and the smallest temporal change to the gradient. Conversely, for both the non-Hispanic white male and female populations, we see the largest gradients as well as the greatest temporal changes to these gradients. That the relationship between educational attainment and mortality changed in different ways implies that the subpopulations experienced very different socio-historical conditions across their life courses. Consistent with the fundamental cause theory of health, the male and female non-Hispanic white populations garner greater health returns from education than the male and female non-Hispanic black populations. These disparate returns are especially apparent in Figure 2. At all education levels, the non-Hispanic white population experienced greater reductions in mortality risk across cohorts. However, the greatest differences in cohort reductions in M_x between the U.S. non-Hispanic black female and non-Hispanic white female populations occurred amongst those with an education greater than high school. Similarly, black-white differences in the cohort reductions in M_x for men with a less than high school education are much smaller than the respective differences amongst men with a high school or greater than high school education. In short, racial inequality in U.S. educational returns is fueling racial and educational inequality in mortality risk across cohorts.

Figure 2: Sex and Race/Ethnicity Group Differences in Cohort Changes to Education Effects on U.S. Adult Mortality Risk, 1986-2007.