When does disadvantage produce younger mortality deceleration, and when older? Comparing populations defined by sex, race, poverty and health status using U.S. Medicare data

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Overview

Mortality selection explanations for mortality deceleration at old ages -- theories that explain deceleration as a consequence of changes in population composition as the frailest members of the population die off -- generate competing predictions about which subgroups of a population will decelerate in mortality at earlier ages.

On the one hand, a common prediction of mortality selection theories is that disadvantaged subpopulations, subject to higher selective pressure, will decelerate earlier than their mortality-advantaged counterparts. This prediction has been made theoretically many times (e.g., Vaupel, Manton, and Stallard 1979; Vaupel and Yashin 1985). Empirical analyses have also confirmed this prediction in several contexts: for example, in earlier deceleration in cause-specific mortality for more higher-mortality causes of death, and in older age at deceleration over time as mid-life mortality falls (Horiuchi & Wilmoth 1998).

On the other hand, mortality selection theories predict that populations with a greater proportion of frail members will decelerate at later ages, if deceleration is a result of frail members being exhausted from the population. Lynch et al (2003) formulate this prediction and confirm it empirically, finding that African-Americans decelerate at older ages than white Americans, and that this age has been declining over time.

We expand this debate in two main ways. First, we add to the dimensions of disadvantage considered empirically, by examining deceleration among subpopulations defined not only by race and sex, but also by poverty status and by physician-ascertained health status. Particularly since health status is a credible measurement of at least one important dimension of frailty, this allows us to more directly consider whether disadvantaged subpopulations with a greater proportion of frail members decelerate at later ages, or whether their correspondingly higher mortality leads them to decelerate earlier.

Second, on a theoretical level, we add consideration of an additional factor influencing a subpopulation's timing of deceleration: the degree of inequality experienced at older ages in particular. We argue that any trait that tends to be held consistently by individuals over time, and that raises mortality consistently over the life course, will create two contradictory effects on aggregate mortality rates at the oldest ages: on the one hand, because it raises mortality at the individual level, it will tend to raise mortality in the aggregate; on the other hand, because it has raised mortality at the individual level in the past, it will tend to be associated with lower aggregate mortality at old ages since those who survived despite it may be robust along other dimensions of heterogeneity. The extent of the mortality increase suffered at the individual level in old ages, we argue, as well as the degree of correlation between who is most disadvantaged at oldest ages and earlier in life, can attenuate or accentuate the effects of mortality selection in slowing mortality increase among the disadvantage.

Data

This study uses individual-level data on a cohort of 28.7 million elderly Medicare beneficiaries, followed longitudinally from 1993 to 2002. The data represent 96 percent of all Americans aged 66 and above who were alive on January 1, 1993.[1]

The Medicare Vital Status file, mostly drawn from the Social Security Administration's Master Beneficiary Record (MBR) file, provides daily death-date follow up (Elwert 2008). Recent evaluations suggest that Medicare death reporting provides the closest match with the National Death Index among national mortality databases, surpassing even the SSA Death Master File (Sohn et al. 2006). Kestenbaum (1992) argues the advantages of (older) Medicare data over Census and death certificate data.

The Medicare Provider and Analysis Review file provides in-patient hospitalization records for 1992, from which we extract detailed health information to control for differences in baseline morbidity. We summarize that information into Charlson Comorbidity Scores, widely used in medical research. Work by Yashin et al (2007a, 2007b) on the Cumulative Index suggests that mordibity measures summarizing the number of distinct health detriments, as in our Charlson scores, may be excellent measures of aging and health deterioration processes.

Since individuals typically enter Medicare at age 65, we restrict the analysis to individuals who were older than 66 at baseline in order to guarantee the availability of one year of health background controls for the entire sample. We also restrict our sample to those less than 97 years old at baseline (106 years old at the end of follow-up) to avoid possible residual problems with age reporting among the oldest old.

The Medicare Denominator file provides additional individual-level demographic information from Social Security records, including age, sex, area of residence, and a poverty indicator at baseline.

Records from all Medicare files were matched using unique individual level identifiers. The record linkage rate was 100 percent.

Methods

Age-specific death rates are estimated using flexible Poisson models. The unique size of our database allows our age measures to be unusually precise: we use three-month age units, which may estimate hazards more accurately than yearly rates at the oldest ages, when hazards may change quite quickly (Gavrilov & Gavrilova 2009). We use a lowess smoother with a narrow bandwidth (.2) to smooth three-month rates across seasonal variation. This dataset also allows us to estimate rates nearly non-parametrically; we allow the effects of poverty and sickness to vary freely over the agespan for each race and sex. Most important is allowing the effect of age itself to vary freely (separately for each race and sex). This is significant because parametric decisions can make a significant difference to the estimation of age at deceleration (Rau et al 2009).

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Following Rau et. al. (2009), we estimate deceleration by finding the age at which the second derivative is at its maximum.

Preliminary Results

Our analysis contributes several striking findings. First, we find that race by itself is not a consistent marker of deceleration timing: among women, black mortality decelerates somewhat later, and among men, black mortality decelerates somewhat earlier than white mortality. On the whole, race-specific differences in deceleration appear to be substantively small.

By contrast, we find, second, that morbidity and especially poverty trace large differences in mortality deceleration: the non-sick, non-poor decelerate substantially *earlier* than the other subpopulations defined by poverty and sickness status, as shown in Figure 1:

Figure 1. Age when second derivative of mortality is at its maximum					
		White Women	Black Women	White Men	Black Men
Non-poor	**Non-sick**	**91.7**	**89.7**	**90.7**	**87.2**
Poor	Non-sick	96.3	96.3	96.3	96.6
Non-poor	Moderately Sick	93.3	95.9	96.8	96.8
Poor	Moderately Sick	96.1	96.3	96.8	96.8
Non-poor	Very Sick	93.1	96.8	92.4	93.1
Poor	Very Sick	95.6	96.8	96.3	95.4

Among the sick and the poor, there is relatively little variation in age of maximum acceleration. But among the non-sick non-poor, it is the populations with the highest mortality through most of the agespan who decelerate first -- with black men at the early end and white women at the late end. In other words, the largest observed difference in deceleration timing between blacks and whites is observed among the most advantaged rather than the most disadvantaged members of both races – and the difference furthermore indicates *earlier* deceleration among advantaged blacks than among advantaged whites.

In sum, these findings suggest that whether disadvantaged groups decelerate earlier or later than their advantaged counterparts is not necessarily empirically consistent across dimensions of disadvantage.