

Revisiting compositional change to understand mortality inequalities

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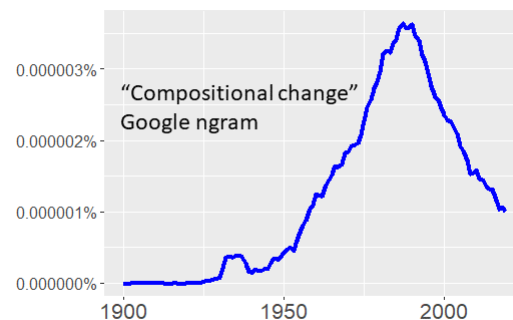
Abstract

Mortality inequalities are socially patterned and increasing in many populations. Typically, social mortality gradients are measured by gaps in survival between predefined groups across one socioeconomic status (SES) dimension, or as a gradient across a population ranked by social position. In interpreting trends, the implicit assumption in both approaches is that the distribution of mortality-relevant personal characteristics that underlie social positions is static. We know this is not the case. Populations change. The distribution of personal characteristics in (sub)populations rises and falls over age, and over birth cohort. We argue that demographers have paid too little attention to how compositional change in the interaction, and intersection, of these characteristics relates to changing mortality inequalities.

Introduction

Populations are dynamic. New people are born and migrate in while others die or migrate out. Cohorts differ in their exposure to varied disease environments and medical progress, while social norms regarding health behaviours and family life evolve. Not only do these changes happen across generations, but within birth cohorts, the composition of a population observed at older ages varies in important personal characteristics compared to when it is observed at younger ages. Compositional changes from mortality-selective sociodemographic characteristics differ in strength over time, vary across and within socioeconomic groups, and muddle our interpretations of trends in period inequalities.

These tenets are seldom fully explored, despite compositional change long being a cornerstone of demographic research. Evelyn Kitagawa pioneered her elegant decomposition method in 1955, which for the first time, allowed researchers to explicitly monitor how a change in who made up the population impacted demographic phenomenon (Kitagawa, 1955). Applied to mortality, this approach splits a change in death rates into contributions from ‘direct changes’ (e.g. from medical or public health advances) and ‘indirect and compositional changes’ (e.g. when a lower-mortality group becomes a higher share of the population). In economic circles, this decomposition approach is known as the Oaxaca-Blinder method. But, as shown by the inset figure to the right, the direct study of compositional change has fallen drastically out of fashion since the 1990s.



In this paper, we argue that compositional change needs a re-examination and bold new theoretical thrusts. We demonstrate how compositional change operating within-cohorts over age, and between-cohorts at the same age, complicate our interpretation of period mortality inequalities. We argue that as the population sciences have become more focussed on individual-level research and questions of causality, they have lost their core strength in describing how basic inequalities are shaped by changing population dynamics. We end with a plea to reorient research around a rich quantification of how changing sociodemographic characteristics are interacting and driving trends in health inequalities.

Compositional change *within* cohorts

As cohorts age, they become increasingly composed of the most advantaged subpopulations. Figure 1 shows the proportion of tertiary-educated men and women in Finland and the United States across various native-born cohorts as they aged. Since ages below age 40 are excluded and university-completion after this age is relatively rare (NCES 2024), within-cohort increases in the proportion of high-educated individuals depicts the social gradient in premature mortality.¹

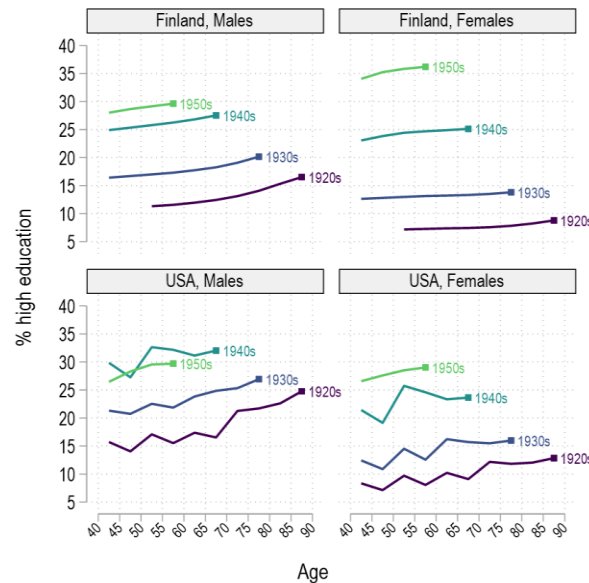


Figure 1. Percent of the native-born population at each age with a tertiary level of education by birth cohort

In the US, the percent of high educated men increased from around 15 to 25 percent for the 1920s birth cohorts as they aged from ages 40-44 to 85-89. For women of these same cohorts, the percent doubled from around 7 to 14 percent. While birth cohorts born after the 1920s are more educated, the slopes of the lines are similar across cohorts. By comparison, the slope is generally flatter for women and for Finnish cohorts, arising from a combination of smaller educational inequalities in mortality and a lower overall level of premature mortality in those populations compared to cohorts of US men.

¹ Emigration could also impact these curves if it is associated with education. Data on the characteristics of emigrants is generally sparse. However, an OECD report estimated that the weighted total emigration rate was the same for the highly educated group as the rate among all education groups (3.8 %) among OECD citizens leaving to other OECD countries in 2005/6 (<https://doi.org/10.1787/9789264177949-table64-en>).

Such social mortality-selective processes go beyond education. As they aged, the AHEAD cohort of the U.S. HRS study experienced increases in median wealth from around \$90,000 to \$130,000 between survey waves due to the higher mortality of the poorer cohort members (Zajacova & Burgard, 2013).

When discussing the narrowing of mortality differences over age between USA and other high income countries, explanations often center around different age-specific health behaviours, and differences in access to and performance of health care systems (Ho & Preston, 2010; Palloni & Yonker, 2016). Although selection vs scarring concepts have also featured prominently in these debates, these are generally viewed in light of differences in early epidemiological environments and with respect to the robustness of individuals exposed to disease at younger ages. Rarely is compositional change due to differences in social gradients, be it by education or other social markers of disadvantage, invoked as an explanation.

Mortality selection also operates *within* SES-groups. Older low-educated individuals are likely to have experienced far more advantaged life courses than those of their same birth cohort who died at younger ages, giving the impression that education was a weaker determinant of mortality than it would have appeared if measured at a younger age. Note that these changes do not require cumulative disadvantage theories (Dupre, 2007; Ross & Wu, 1996) to hold, which posit that accumulated risks over the life course increasingly disadvantage low-SES individuals with age. So long as these groups contain a heterogeneous set of characteristics that impact mortality, the (sub)population-level risk set will change over age.

[Future figure about here will depict a nice example of how within-group social characteristics change over age]

Mortality selection might be especially problematic in studying social inequalities based on fixed older ages. In Finland, the proportion surviving to age 65 and 75 has roughly doubled since 1971 and showed strong occupational class differences (Diaconu, van Raalte, & Martikainen, 2022). During this period, occupational-class based inequalities in e_{65} and e_{75} widened sharply. On the other hand, the proportion of the population surviving to the modal age at death was remarkably similar across time and occupational classes. And occupational-class based inequalities in the modal age at death were stable too.

While all of this is known theoretically, there has been strikingly little descriptive work about how the distribution of sociodemographic mortality-relevant characteristics change over the life course of cohorts and contribute to mortality inequalities at any given age. A notable exception is recent work showing that changing distributions of various characteristics (e.g. SES indicators, region of residence) due to selective mortality does not explain the black-white mortality crossover at older ages for the 1909-1911 US birth cohorts (Breen, 2024).

One reason for neglecting this type of research might be because classic theories of population heterogeneity are based largely on innate unobserved personal characteristics, fixed at birth, which determine the frailty distribution of the population (Vaupel, Manton, & Stallard, 1979). As the frail die younger on average, the population will become dominated by less frail individuals at older ages. The concept of *dynamic heterogeneity*² gets closer theoretically to selective mortality of individuals with less favourable characteristics acquired after birth (Caswell, 2009; Tuljapurkar et al., 2009). Essentially it argues that individual life courses are full of forking paths which move them from higher- to lower-risk (or vice versa) mortality subgroups, which can be modeled in multistate settings. However, like the classic lifetable, the outcome of interest is generally state expectancies. Because multistate models are based on Markovian processes, where each transition does not depend on the sequence of state transitions or duration spent in any state, the direct connection to cohort compositional change over age is not explicit.

Compositional change *between* cohorts

Not only are socioeconomic inequalities in mortality impacted by changing group membership across age within a cohort, but also across cohorts any given age. This occurs as a result of changes *before* the groups are defined (e.g. changing sorting mechanisms) as well as by changing characteristics of the cohort as it ages.

² This is the term coined by Tuljapurkar, Steiner, and Orzack (2009) to distinguish it from ‘fixed heterogeneity’ e.g. the classic models of Vaupel and colleagues (1979). Somewhat confusingly, at around the same time, Caswell (2009) introduced the term ‘individual stochasticity’ to describe the same thing as dynamic heterogeneity. The Caswell thinking is that life histories are modeled as a Markov process, with individuals grouped into subpopulations based on observed characteristics. In such a world, everyone starting in the same population stage (for example age 30, unmarried) is subject to identical rates of transitioning to other stages (for example age 31, married). The outcome of this process is stochastic.

Changing selection patterns into sociodemographic groups between cohorts has received comparably more attention in health inequalities research, especially relating to educational expansion (Bound, Geronimus, Rodriguez, & Waidmann, 2015; Dowd & Hamoudi, 2014; Hendi, 2015). As the low educated group shrinks in size, the group is theorized to become increasingly composed of individuals with serious health conditions or from highly disadvantaged family backgrounds. Such adversities both moderate and mediate the relationship between education and mortality, as the adversities increase mortality risk directly, but also decrease the likelihood of completing basic levels of education (Dowd & Hamoudi, 2014; Jackisch & van Raalte, in prep). As a result, mortality inequalities between the now smaller lowest educational group and all other educational groups increases (Hendi, 2024; Mackenbach, 2019).

Things become even more complex when we try to interpret period (calendar year) changes in mortality inequalities since the size of population groups change over age but the weight of these ages in aggregate measures like life expectancies do not. Only a few studies have approached this question, generally for educational expansion, by recreating educational groups based on fixed age-specific educational percentiles (Bound, Geronimus, Rodriguez, & Waidmann, 2015; Dowd & Hamoudi, 2014; Hendi, 2015). This is not so easily done without some degree of random reallocation between groups. In some years, over half of the population might attain the same level of education. In doing so, these studies tend to find that inequalities are reduced, but do not disappear.

Recent work highlights a strikingly consistent pattern of education-group mortality rate ratios with respect to group size (Hendi, Elo, & Martikainen, 2021; Mackenbach, 2019). Importantly it is asymmetric, meaning that smaller low-educated groups are more disadvantaged than smaller high-educated groups are advantaged. Hendi describes this regularity as ‘convex inequality’ that closely follows a negative logarithmic relationship (Hendi, 2024).

This finding impacts our usual ways of measuring period inequalities in mortality. Generally, we compare populations using age-standardized methods such as life expectancies or age-standardized death rates. But if the size of the socially defined subpopulation relates to its mortality, then we should expect to see a relationship between the size of the subgroup at a given age and its contribution to the mortality disadvantage.

Indeed, this appears to be the case. [Figure 2](#) shows the association between how an age contributes to life expectancy gaps between high and low educated in a given period, and the share of

that age-group that is low educated among Finnish men over a 35-year period. The share of low educated men is decreasing over time, as the colours change from dark blue to red moving from left to right within each age group facet. Within each age group, as the share of low educated decreases, the contribution of that age group to the gap in life expectancy between low and high educated increases.

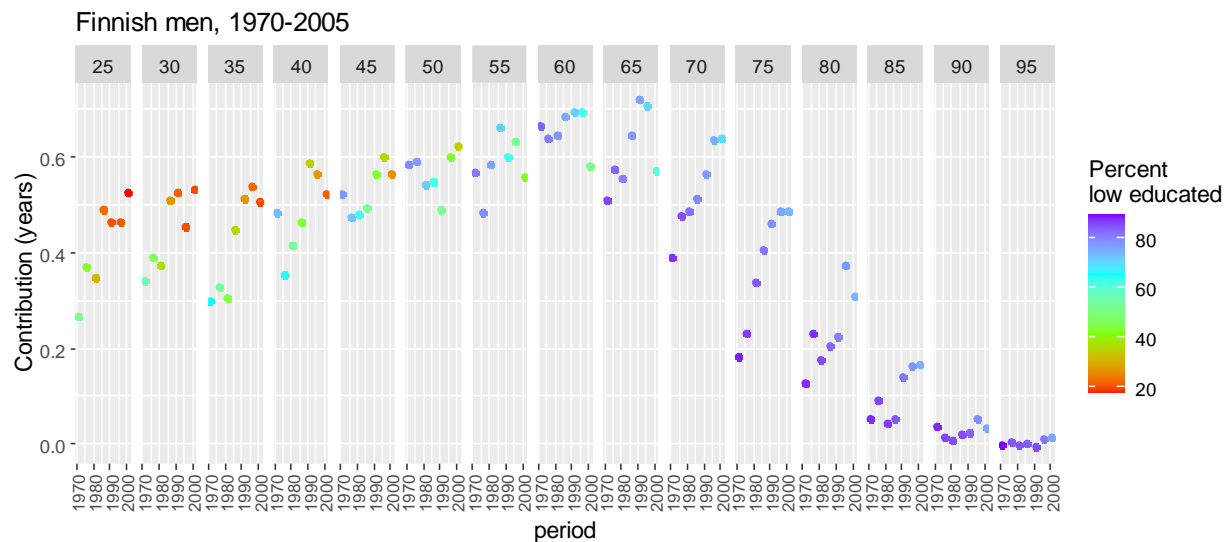


Figure 2: The relationship between the size of the lower educated group observed at each age-period (as a percent of the total) and its contribution to period life expectancy disadvantage against the higher educated.

Life tables do not take the group size into consideration, since they are built up from rates. But group sizes matter because they relate to selection. Part of why we aren't yet seeing older ages contributing to mortality inequalities may be because these group sizes continue to be dominated by the low educated. For age groups that are inflated compared to their stationary equivalent, this might seriously bias our estimates of their impact on mortality inequalities.

However, based on this preliminary analysis alone we cannot say that group size is the most important or even a very important component to the growing life expectancy gap by level of education. Social gradients in mortality are generally increasing in age, period, and cohort dimensions just as educational expansion is happening across age, period, and cohort dimensions. To further tease this relationship apart and make estimates on its size, future work between now and the IPC conference will (a) extend this to 2019; (b) compare it to other markers of SES that do not change in size – e.g. income quintiles where the cut-points are age and sex-specific; (c) check to what extent these relationships generalize beyond Finland.

Health behaviours have also changed substantially across cohorts, and are known drivers of mortality inequalities. The impact of changing smoking patterns on mortality inequalities has been thoroughly investigated (Denney, Rogers, Hummer, & Pampel, 2010; Long et al., 2021), but other health behaviours less so. For example, there is some evidence that trailing baby boomers have experienced increased mortality risk from a host of risky behaviours (especially drug abuse and HIV/AIDS) throughout their life course compared to those born before and afterwards (Acosta et al., 2020; Acosta & van Raalte, 2019).

Beyond changing education and health behaviours, the impact of other major compositional shifts in sociodemographic characteristics on mortality inequalities is less well understood. Social change is strongly associated with cohort replacement (Ryder, 1965). This includes changing social norms relating to divorce, cohabitation, multi-partner fertility, fertility timing etc., all of which have led to increased family diversity (Ajzen & Klobas, 2013; Liefbroer & Billari, 2010; Perelli-Harris et al., 2012). At the individual level, there are strong associations between many of these characteristics and mortality. But scaling these individual-level associations up to the population level is rarely done. Such shifts are complex to model, as family patterns change dynamically across age. This makes populations observed at the same age incomparable across time along multiple social dimensions. As a result, it remains unclear how diverse social and family characteristics relate to aggregate mortality trends, as well as to mortality inequalities along one or more socioeconomic dimensions. To the extent that these social compositional changes differ across countries in their timing and intensity, they also challenge our interpretations of why social mortality gradients are steeper in some countries and time periods compared to others.

Why compositional change needs a re-examination

There are reasons to think that compositional change is an increasingly important component of mortality inequalities, both across individuals, and between groups defined by SES. [Figure 3](#) shows aggregate trends in life expectancy and life-span variation, measured here by the standard deviation in age at death, for Finnish females since 1971 (van Raalte, Sasson, & Martikainen, 2018). All educational groups experienced increasing average lifespans, with a small increase in the gap between groups over time (left panel). An equally important phenomenon is that ages at death are becoming more spread out among the low educated, and more similar among the high educated (right panel). This implies that the

within-group heterogeneity is changing, sometimes sharply. Mortality theories do not predict this development.

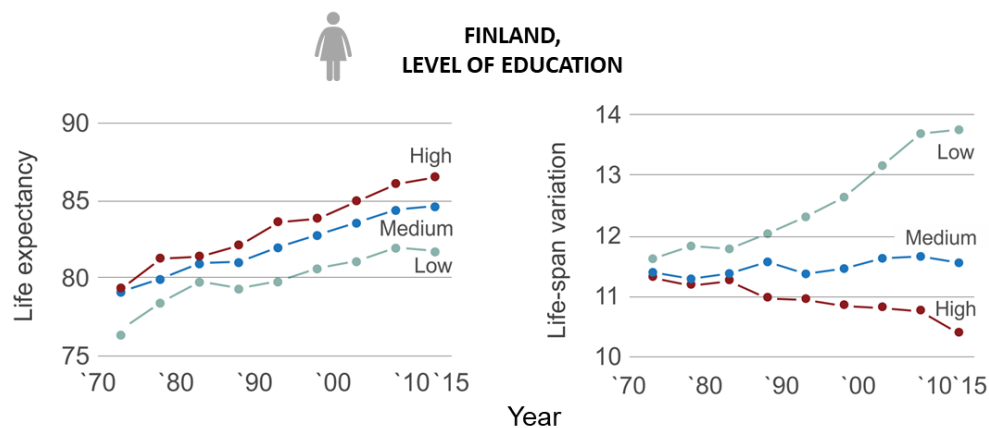


Figure 3: Trends in life expectancy and life-span variation measured by the standard deviation, both conditional upon survival to age 30. Figure adapted from van Raalte et al. (2018).

Changing selection patterns into SES-groups could explain why lifespan variation is diverging sharply across educational groups, as educational expansion has drastically reduced the size of low educated individuals. However, life-span variation is diverging for other SES markers such as income quintile and occupational class, though less dramatically (van Raalte et al., 2018). Thus, to explain growing lifespan variation among low-income groups with stable population sizes requires further explanations. It could be that the theorized causal relationships between SES and mortality (e.g. Doyal & Pennell, 1979; Link & Phelan, 1995; Lopez, Collishaw, & Piha, 1994; Marmot, 2005; Wilkinson, 1997) are changing. But it could also be that once adult SES positions are established, the same low-SES cohort will have different social characteristics at older ages because of selective mortality (intra-cohort compositional change)—and that older ages increasingly drive inequalities in a context of general mortality declines. Or it could be that low-SES populations at the same age have a different distribution of health-protective personal characteristics across generations (inter-cohort compositional change) given changing social norms, for instance, regarding family formation or risk-taking behaviour. Any of these compositional-change driven explanations impact mortality inequality over the age, period, and cohort dimensions. And none of them have been thoroughly tested.

What remains clearer, from descriptive research, is that differences in the magnitude of mortality inequalities across populations are largely driven by survival differences in the most disadvantaged groups. Life expectancies of the highest educated are remarkably similar across European

countries (van Raalte et al., 2011) and U.S. states (Montez et al., 2019). But the sets of characteristics that makes the low educated groups different across these populations, and presumably over time, to generate this disadvantage has received comparably little attention.

Discussion

A general lack of progress in tackling mortality inequalities, despite decades of research into the social determinants of health, has led to pleas for new narratives (Lundberg, 2020). As we and others have argued, major breakthroughs in understanding population-health inequalities require a systematic understanding of compositional differences and compositional change (Montez & Bisesti, 2024; van Raalte, 2021). In our view, this has not been forthcoming because of a major shift in the social and biomedical sciences away from general descriptions of aggregate trends and toward using causal inference to explain the processes. By and large, this shift has been an important and necessary development. But it has also come with costs. Changes to the composition of a population are less frequently an outcome of interest, but more regarded as confounders that should be controlled away to get causal estimates, for example by propensity-score matching, inverse probability weighting, or using family fixed-effects study designs. Such methodologies are critical to understanding and ranking the determinants of mortality at the *individual* level. But at the *population* level, aggregate trends can only be understood with a parallel investigation of how the confounders themselves are changing in size and importance, and how they interact with one another.

The time is ripe to make genuinely ground-breaking theoretical and empirical advances. The demographic data landscape has changed enormously over the past few decades (Kashyap, 2021). We are no longer limited by tabulated cross-sectional data on a handful of variables. We can exploit unprecedentedly detailed information on individuals' life courses available within population registers and long-running panel surveys. Increasing computational power and parallel advances in powerful statistical and machine-learning approaches have opened up possibilities to exploit the predictive power of these newer data sources and generate new hypotheses. While both micro-level regression approaches and macro-level decomposition techniques have already examined some of the more obvious aspects of compositional change (for instance educational expansion, (Dowd & Hamoudi, 2014; Hendi, 2015) neither has fully explored the rich web of interactions between social variables.

Typically risk factors for mortality are modelled individually or within a few selected domains, such as looking at the impact of health behaviours or economic factors on mortality. Only a few studies have looked simultaneously at multiple characteristics and attempted to rank their importance. These have either been limited in number (Mackenbach et al., 2019; Stringhini et al., 2017) or based on heavily-modeled findings pooled across populations in complex ways (Murray et al., 2020). A notable recent exception is recent research that investigated the predictive value of 57 risk factors for mortality contained in the HRS study of Americans aged 50+ (Puterman et al., 2020). Smoking was the most important predictor, although adult SES characteristics, history of divorce or unemployment, and low life satisfaction also ranked highly. Childhood adversity, on the other hand, did not come out as a particularly strong predictor—although this adversity was conceptualized by self-reported childhood SES-circumstances, a much narrower and potentially less traumatic concept than what is usually considered (McLaughlin, Sheridan, Humphreys, Belsky, & Ellis, 2021). With long-running register data and panel studies, we could greatly expand the personal characteristics examined to include time-varying family characteristics including living arrangements, childhood adversities, diagnosed diseases, migration experience, full working and income histories, incarceration or institutionalization, etc.

We need to realign mortality inequalities research. We need to move the field beyond updates of mortality inequalities by education, income, and occupational class after each census round, beyond recycling debates of social selection vs. social causation, and beyond a heavy emphasis on explaining inequalities on the basis of downstream health behaviours and causes of death. Mortality theories need to consider social characteristics in dynamic ways. For population health can only be understood by understanding our populations. And understanding how they change.

Future outlook

This manuscript is still very much work in progress, and somewhat hastily put together. We will experiment with better examples to illustrate our arguments and bring in a richer literature to strengthen our points.

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