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Mortality deceleration is not informative of unobserved heterogeneity in open groups

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Abstract

Studies of mortality deceleration sometimes use the order of deceleration between two groups—such as birth cohorts or subpopulations defined by race or sex—to draw conclusions about population heterogeneity. These studies often draw on the fact that between two closed groups, *ceteris paribus*, the higher-mortality group will decelerate at a younger age. This paper gives a first consideration to the order of deceleration between open groups. I construct a model with a one-way flow from 'healthy' to 'sick' status and, crucially, assume that a subset of the cohort has elevated risk for both sickness and death. Using simulations designed to resemble, in the aggregate, cohorts in the Human Mortality Database, I show that mortality deceleration order in such a cohort is essentially unpredictable because it depends on the interaction between a large number of parameters, some of which are unobserved in empirical data. These results suggest that it may be challenging to extend the study of mortality deceleration to include open groups whose memberships are selected for frailty or robustness, while still drawing meaningful conclusions about population heterogeneity.

1 Introduction

Cohort mortality often decelerates: the rate of mortality increase over age slows down at old ages, producing a plateau in some cohort mortality hazards. This deceleration is often attributed, in whole or in part, to *mortality selection*: the change in cohort composition over age as 'frailer' members die, leaving an increasingly robust, low-mortality cohort. Most empirical research on mortality deceleration focuses on decelerations in closed cohorts or closed groups defined by race or sex. Selection dynamics in such closed heterogeneous groups are well explored in the theoretical literature (e.g. Beard 1959, 1971; Kannisto 1992; Vaupel et al. 1979; Thatcher et al. 1998; Vaupel and Yashin 1985), and empirical research profitably draws on these theoretical results.

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Chief among the theoretical predictions that guide empirical work on mortality deceleration is that if mortality deceleration results from mortality selection, then higher-mortality groups, *ceteris paribus*, should decelerate at younger ages than lower-mortality groups. This stems from the fact that higher-mortality groups are subject to more intense selective pressure (Vaupel and Yashin 1985; Vaupel et al. 1979; Kannisto 1992). Such predictions about the order of groups' mortality deceleration allow empirical comparisons of deceleration timing to be used in one of two ways.

First, such predictions allow selection-based explanations of deceleration to be tested, an important undertaking because a large body of biodemographic work mounts a case for decelerating mortality in individuals, not only populations (e.g. Carey et al. 1992, Curtsinger et al. 1992, Drapeau et al. 2000 (but see Steinsaltz 2005), Mueller et al. 2011, Rauser et al. 2005, Steinsaltz and Wachter 2006, Vaupel and Carey 1993; and see reviews in Vaupel 1997, Wachter and Finch 1997). For example, Horiuchi and Wilmoth (1998) find that the age of deceleration onset has risen over time as the level of cohort mortality has fallen (see also Engelman et al. 2010), and take this cross-cohort comparison as evidence in favour of the selection explanation, since it is consistent with what that explanation would predict. Similarly, Lynch and Brown (2001) find that within the United States, higher-mortality male cohorts decelerate at younger ages than lower-mortality women, and consider this evidence for selection.

Second, such predictions allow analysts to *assume* that differences in deceleration timing arise from differential selection, and therefore use empirical data to derive information about latent heterogeneity. For example, Lynch et al. (2003) find later deceleration among black Americans compared to white Americans in spite of the black cohorts' greater mortality, and conclude that black cohorts born 1972–1990 had greater frailty from birth than white cohorts: selection theory predicts that if the groups had the same frailty distribution at birth, then the black cohorts should decelerate earlier, not later.[1] In sum, the generalisation that, all else being equal, higher-mortality groups should decelerate at younger ages is a linchpin connecting mortality selection theory to the empirical testing of its assumptions.

This paper explores the selection dynamics of mortality deceleration between *open* groups, i.e. groups that can be entered as well as exited, such as sick vs healthy, poor vs non-poor, or countries that exchange members. Specifically, I ask whether it remains true for open groups that higher-mortality groups should decelerate before lower-mortality groups.

This question is motivated in part by a growing interest among demographers in expanding the study of mortality to include health and disability statuses (e.g. Robine et al. 2003, Zeng et al. 2006). In part, this reflects the increasing availability

[1] As Lynch et al. note, this conclusion rests on two further assumptions as well: that frailty has the same mortality consequences in each group, and that the groups age at the same rate. (These conditions are formalised below, when I present the models.)

of longitudinal data with rich health covariates. It also reflects the concerns of ageing societies that, in the hope enabling interventions which would support increased quality of life at old ages, may conceptualise mortality not merely as an event, but as a process that may—or may not—include varying dimensions of morbidity before culminating in death. An early tradition of mortality research, centered around Manton, Stallard, Woodbury, and Yashin (e.g. Manton et al. 1994, 1995; Woodbury and Manton 1983), did integrate health transitions with individual heterogeneity in a variety of models, but largely has not been echoed by more recent demographic research.[2] Perhaps because such efforts have not gained wider traction, it remains unknown what generalisations about phenomena such as mortality deceleration might apply in those contexts. As mortality research continues to incorporate health and morbidity, an open question is what kinds of knowledge about mortality, rooted in comparisons of relatively closed groups such as cohorts and countries, can be extended to open groups such as health statuses that individuals move between.

I will address this question on the most favourable ground for mortality selection theory, in order to dramatise the difficulties that exist, even on that ground, in applying selection theories about mortality deceleration to open groups. A large body of biodemographic research (cited above) challenges the idea that deceleration results solely from selection; I will assume that it does. Previous work (Wrigley-Field 2014) also shows that some widespread generalisations about mortality deceleration need not hold even in the closed-group context; the generalisation considered here—that higher-mortality groups decelerate first—does. I also use a very simple model in which frailty is binary and fixed in individuals (following the classic early work building demographic intuitions about closed groups, Vaupel and Yashin 1985), and there is only a one-way flow from sickness to health. In short, the analysis in this paper is designed to be favourable to finding straightforward predictions about mortality deceleration in an open-group context. It would be desirable for the predictions about deceleration order among open groups to be similarly straightforward as the closed-group generalisation that higher-mortality groups decelerate at younger ages. This paper demonstrates that they are not.

In what follows, I begin by defining mortality deceleration and then by more precisely presenting the closed-group and open-group selection models. I then intuitively motivate a hypothesis: that the order of deceleration is close to unpredictable in the dynamic (open group) context because it is very sensitive to the relative sizes of the subgroups, including latent subgroups. To evaluate this hypothesis, I present a series of simulations designed to resemble, in the aggregate, cohorts in the Human Mortality Database.

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[2] More generally, there is a small but important literature on heterogeneity in dynamic settings (particularly Mohtashemi and Levins 2002 and Rogers 1992) that, while concerned with different issues than mortality deceleration, can broadly be considered inspiration for this paper.

2 Defining deceleration

This paper defines a deceleration point as a point when the second derivative of unlogged mortality becomes negative (or in other words, when the mortality slope begins to decline). This is the empirical measure used in the article (Lynch et al. 2003; see also Lynch and Brown 2001) that, by explicitly stating the conditions (reviewed below) for higher-mortality closed groups to decelerate first, helps to inspire the current analysis.

A more commonly-used measure is the Life-table Ageing Rate (LAR), the slope of logged mortality (introduced by Horiuchi and Coale 1990, though the term is introduced later; see also Horiuchi 1997, Horiuchi and Wilmoth 1997). By taking the derivatives of logged mortality, the LAR focuses attention on the increase in mortality relative to the overall level of mortality. This relative measure has the advantage of capturing an intuitive idea of what mortality deceleration means (Horiuchi and Coale 1990), but the disadvantage that selection dynamics are conflated with the level of mortality (Vaupel and Zhang 2010). For this paper, which is concerned with understanding the relationship between selection dynamics and deceleration order, this disadvantage is paramount, and so measures that take the derivatives of mortality directly are preferable to the LAR.[3]

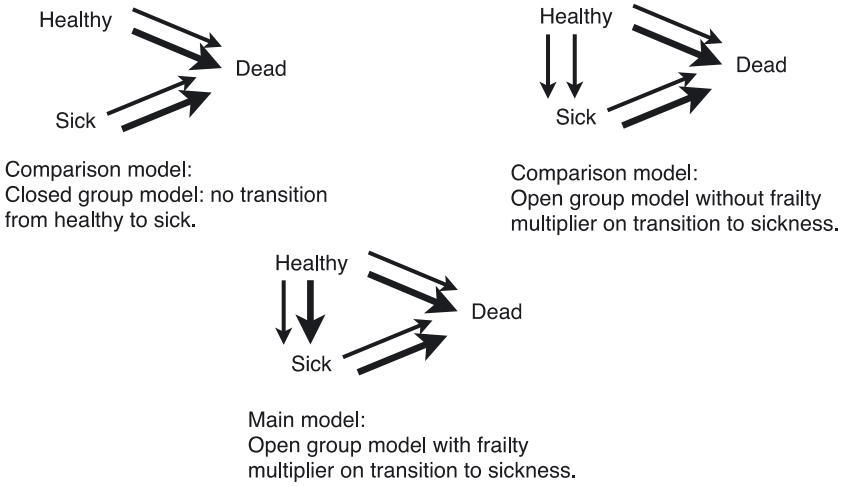
3 The closed-group and open-group selection models

Previous work on deceleration uses closed-group models in which higher-mortality groups decelerate first. The main purpose of this paper is to examine a health selection model in which some subset of the population has elevated risks both for becoming sick and for mortality conditional on sickness. This new model incorporates two changes, compared to the closed-group model: the addition of a one-way flow from health to sickness, and the fact that the frail are at heightened risk for the new flow (as well as mortality). To isolate the effects of each change, I also consider an alternative open-group model in which the healthy can become sick, but—unlike the main model—the robust and the frail are equally likely to undergo transition from health to sickness. These three models are summarised in Figure 1, and are given formally below.

Substantively, the closed-group model can be thought of as a model in which sickness/health is assigned at birth rather than acquired, and the two open-group models are distinguished by whether the risk factors for sickness and for mortality (conditional on sickness) overlap. The main model, the open-group model with

[3] There is also an alternative measure using the mortality derivatives: Rau et al. (2009) proposes marking deceleration as the point when the *third* derivative of mortality becomes negative. I avoid that measure in this paper because it admits the possibility of multiple decelerations even in the closed-group case with binary frailty (Wrigley-Field 2014), which would complicate this investigation with little analytical payoff for present purposes.

Figure 1:
Graphical representation of the three models used in this paper



elevated sickness risk for the frail, posits that there are *some* shared risks for sickness and death operating at all ages. For example, smoking, living in poverty or being born low-birthweight might each increase the risk of heart disease, and also increase the risk of death regardless of whether one has heart disease. The comparison model, in which the frail and robust have equal transition rates to sickness, posits that there are *no* shared risks for sickness and for mortality conditional on sickness. This might be a reasonable approximation for a population in which most deaths are attributable to accidents and the sick are no more likely to engage in risky behaviour, but for most purposes, the main model seems a better approximation.

As we will see, just transitioning to an open group creates problems for the predictability of deceleration order, but the major problems come with shared risks for sickness and death.

3.1 Closed-group model

Consider two closed groups, say, men and women. Imagine that both groups are heterogeneous, composed of two kinds of people—the 'frail' and the 'robust'—with frailty fixed at birth. (In a typical empirical context, such frailty would be unobserved and posited theoretically.) All individuals have Gompertz mortality with the same slope over age. The frail have higher mortality than the robust, by the same proportion for men as for women, and men have higher mortality than women. Aggregate mortality in each group depends on the mortality of frail and robust individuals, and on the proportion of each group (men or women) that is frail at each

age. The individual-level mortality is given in Equation (1):

$$\begin{aligned} \mu_{robust\ women}(a) &= \alpha e^{\beta a} \\ \mu_{frail\ women}(a) &= f\alpha e^{\beta a} \\ \mu_{robust\ men}(a) &= m\alpha e^{\beta a} \\ \mu_{frail\ men}(a) &= fm\alpha e^{\beta a} \end{aligned} \tag{1}$$

where β is the log-slope of mortality for all individuals, α is the intercept of mortality for robust women, f is the frailty multiplier on mortality, and m is the male multiplier on mortality. If the proportion frail at any age is denoted $\pi(a)$, then the mortality over age for women and men is given by Equation (2):

$$\begin{aligned} \bar{\mu}_{women}(a) &= (1 - \pi_{women}(a))\alpha e^{\beta a} + \pi_{women}(a) f\alpha e^{\beta a} \\ \bar{\mu}_{men}(a) &= (1 - \pi_{men}(a))m\alpha e^{\beta a} + \pi_{men}(a) mf\alpha e^{\beta a}, \quad \text{for } f, m > 1 \end{aligned} \tag{2}$$

The proportion frail in each group, $\pi_{women}(a)$ and $\pi_{men}(a)$, can be calculated directly from the survivorships at each age for each subgroup defined by sex and frailty. The survivorships for each subgroup i at age $a + x$ are given in terms of the survivorships at age a in Equation (3):

$$S_i(a + x) = S_i(a) \cdot (1 - \mu_i(a)x) \tag{3}$$

In this closed model, mortality is the only source of change in the size of each subgroup, and hence of the frail/robust composition of the groups.

The generalisation that men will decelerate at a younger age than women rests on three *ceteris paribus* assumptions built into this model (given in Lynch et al. 2003): the groups share an individual-level log-slope of mortality over age, β; they share the same proportional inequality between frail and robust mortality, f; and, not shown in Equation (1), they must also have the same proportion frail at baseline, $\pi(0)$.

3.2 Open-group model

The open-group models considered here retain the three assumptions from Lynch et al., but the move from closed to open groups adds substantial complexity to this model. To limit this complexity while homing in on the key features of open groups relevant to mortality selection, the model considered here incorporates several constraints. The most important of them is that the open-group model considered here has only a one-way flow from health to sickness. Thus, at any age, the healthy can stay healthy, become sick, or die, whereas the sick can only stay sick or die. A second key assumption of the model is that the frail are more likely than the robust to become sick, as well as to die regardless of whether they are sick. This assumption can be conceptualised as the idea that there are some shared risks for morbidity and, conditional on morbidity, for mortality, that are fixed at the individual

level. (Results from models omitting this assumption are also presented below, for comparison.) Finally, the open-group model retains the *ceteris paribus* conditions for the higher-mortality group to decelerate first in the closed-group model: individuals, regardless of their subgroup (defined by sickness/health and frailty/robustness), share a log-slope of mortality over age, β; both groups (defined by sickness/health) have the same proportional inequality between frail and robust mortality, f; and both sickness/health groups have the same proportion frail at baseline, $\pi(0)$.[4]

Thus, the mortality functions in this open-group model, conditional on $\pi(a)$, are the exact equivalent of the mortality function in the closed-group model, with m ('morbidity') now representing the mortality multiplier associated with being sick (the variable name is retained from Equations (1) and (2) since its role in Equation (4) is identical). These open-group mortality functions are given in Equation (4):

$$\begin{aligned}\bar{\mu}_{healthy}(a) &= (1-\pi_h(a))\alpha_\mu e^{\beta_\mu a} + \pi_h(a) f_\mu \alpha_\mu e^{\beta_\mu a} \\ \bar{\mu}_{sick}(a) &= (1-\pi_s(a)) m\alpha_\mu e^{\beta_\mu a} + \pi_s(a) m f_\mu \alpha_\mu e^{\beta_\mu a}, \quad \text{for } f_\mu, m > 1 \end{aligned} \tag{4}$$

However, the survivorship functions, which determine the value of $\pi(a)$ for each group, and therefore weight the subgroups in the mortality functions just presented, are more complex in the open-group model than the closed-group model—especially for the sick. Both survivorship functions depend on the rate at which the healthy become sick, given in Equation (5):

$$\bar{\omega}(a) = (1-\pi_h(a))\alpha_\omega e^{\beta_\omega a} + \pi_h(a) f_\omega \alpha_\omega e^{\beta_\omega a}, \quad \text{for } f_\omega > 1 \tag{5}$$

Thus, the total rate of becoming sick is an aggregate rate for the frail and robust healthy, with the frail healthy at greater risk in proportion to the frailty multiplier on sickness, f_ω (which need not equal the frailty multiplier on mortality, f_μ). In addition to the key analytical assumptions outlined above, Equation (4) makes one additional assumption for tractability. The rate of becoming sick among the frail and the robust has an equal log-slope over age, β_ω. This log-slope may be different from the log-slope of mortality over age, β_μ, but neither can vary between the frail and the robust. The choice of parametric form for the subgroup rate of becoming sick is relatively arbitrary, but is assumed here—like mortality—to be Gompertz to reflect with substantive plausibility the increasing morbidity risk over age. (The open-group comparison model in which the frail and robust are equally likely to become sick is identical to the model given in Equations (4) and (5) except that the frailty multiplier on sickness, f_ω, is constrained to equal, not exceed, 1.)

[4] The assumption that the healthy and sick groups have equal frailty composition at baseline is unlikely to be substantively realistic. I nevertheless make it, for two reasons. First, the assumption retains continuity with the closed-group model, which is essential since the question addressed here is whether a generalisation about the closed-group model can extend to open groups. Second, it is not clear *a priori* which group should be expected to have a larger number of frail members, if baseline is not birth—precisely because of the complexity of the differential selection of the frail in and out of the groups, as explained in this section.

The survivorship of the healthy and sick can then be given in terms of the rate of becoming sick, $\omega(a)$, given just above in Equation (4). The survivorship functions at age $a + x$ for the healthy and sick, for subgroup z (frail/robust), are given in Equation (6):

$$\begin{aligned} S_{h,z}(a+x) &= S_{h,z}(a) \cdot (1 - \mu_{h,z}(a)x)\,(1 - \omega_z(a)x) \\ S_{s,z}(a+x) &= S_{s,z}(a) \cdot (1 - \mu_{s,z}(a)x) \\ &\quad + S_{h,z}(a) \cdot \eta(0) \cdot (1 - \mu_{h,z}(a)x)\,(1 - \omega_z(a)x)\,, \\ \text{for } \eta(a) &= \frac{S_{h,z}(a)}{S_{s,z}(a)} \end{aligned} \tag{6}$$

Equation (6) shows that the survivorship of the healthy at any age is the healthy group at the previous age, minus the decrements to death and to sickness. In contrast, the survivorship of the sick is the sick group at the previous age minus the decrement to sickness, plus the portion of the *healthy* group at the previous age that survives and becomes sick. Crucially, the composition of the sick group now depends not only on the rate of flow of the healthy group into the sick group, but also on the size of the healthy group relative to the sick. This relative size, in turn, is a function of the relative sizes of the groups at baseline, $\eta(0)$, and the survivorship of the healthy group. This dependence of the selection dynamics within the sick on the relative size of the healthy will be a crucial factor in the analysis in this paper. (For ease of interpretation, while I have presented Equation (6) in terms of the ratio of the size of the healthy group to the size of the sick group, in subsequent discussion and analysis I will use the baseline per cent of the total cohort that is healthy rather than this ratio.)

In summary, Equations (5) and (6) make it clear that in spite of the constraints imposed on the open-group model used here, the mortality of the healthy and—especially—the sick, aggregated over frailty, depends on many more parameters than did the aggregate mortality of closed-group men and women. In place of that closed-group model's five parameters—the intercept (baseline mortality) for robust women, α; the frailty, f, and male, m, mortality multipliers; the log-slope of mortality for all subgroups, β; and the baseline per cent frail for both groups, $\pi(0)$—there are nine parameters in the open-group model. These nine parameters are: the five parameters analogous to the closed-group model—the intercept (baseline mortality) for the robust healthy, α_μ; the frailty, f_μ, and sick, m, mortality multipliers; the log-slope of mortality for all subgroups, β_μ; and the baseline per cent frail for both groups, $\pi(0)$—as well as three additional sickness parameters—the intercept (baseline rate) of becoming sick for the robust, α_ω; the log-slope of becoming sick for the frail and robust, β_ω; and the frailty multiplier on becoming sick, f_ω—and an additional baseline cohort composition parameter: the baseline size of the healthy group relative to the sick, $\eta(0)$. In short, the addition of a single unidimensional path from healthy to sick adds substantial complexity to the aggregate mortality of each group.

3.3 Intuition and hypotheses

In the closed-group model, the higher-mortality men should always decelerate at younger ages than the lower-mortality women when the two otherwise share all mortality parameters. In the open-group model, the situation is more complex. To understand the model, we can ask two questions: how do the mortalities of the healthy and sick, in the open-group model, compare to women and men in the closed-group model? And, of paramount importance to this paper, how do the mortalities of the healthy and sick compare to one another?

For the healthy, the open-group model is a straightforward extension of the closed-group model, with mortality simply extended to two separate decrements, loss to death and loss to sickness. Thus, the healthy in the open-group model certainly are more selected than are the women in the closed-group model with identical mortality parameters. But are they more or less selected than the open-group sick? Although the sick have higher mortality than the healthy, the two decrements from the healthy group might—or might not—give it a higher total decrement rate than the sick group has to mortality alone.[5] Since both death and sickness occur selectively, more often to the frail, this might in principle lead the healthy group to be more quickly selected than the sick group, and thus to decelerate earlier—the opposite of the pattern in the closed-group model, but arising for the same reason as the pattern in that model.

For the sick, however, the increment from the healthy group creates substantial complications. To see why intuitively, think of the sick group at any age as divided along a further dimension: the newly sick and the long-standing sick (a stylised distinction serving as a heuristic for the real, continuously-varying duration of sickness). The long-standing sick may have been disproportionately frail early on, since the frail tend to become sick quickly. But subject to the elevated mortality of sickness, that long-standing sick subgroup may have been whittled down to a smaller, largely robust set of survivors.

The newly sick, on the other hand, have not yet faced the intense selective pressures of sickness for very long. Therefore, one possibility is that, as the frail continue to become sick disproportionately, the continued flow from health into sickness produces a *frailty replenishment*, as the frail continuously become sick, die, and are replaced in the sick group. Such frailty replenishment, by offsetting the increased mortality selection of the sick, could also lead the sick to decelerate at older ages than the healthy, reversing the closed-group deceleration order.

However, the opposite order of deceleration is also possible. The newly sick, rather than replenishing the frailty of the total sick, can also tilt the composition of the total sick farther towards robustness. The easiest way to see this is to imagine

[5] Moreover, the total decrement rate for the healthy and for the sick might cross over age if one has a larger intercept and the other a larger slope.

that the healthy group simply runs out of frail members.[6] Then the flow of people from healthy to sick will compound the effects of mortality selection among the sick, potentially precipitating a mortality deceleration. One possible outcome is that if this fall in frailty composition among the sick occurs quickly enough, it might precipitate a mortality deceleration even if the healthy group did not decelerate while it lost its frail members. In that case, the sick might decelerate while the healthy do not. Alternatively, if we now imagine that the healthy have not fully lost their frail members, but have only lost enough to abruptly lower the per cent frail among the sick, then we can imagine that the sick decelerate while the healthy will decelerate later, as they lose still more frail. In that case, the open-group model produces the same deceleration order as the closed-group model, rather than reversing it.

In summary, moving to an open-group model (with a one-way flow from healthy to sick and an elevated transition rate among the frail) should lead the healthy to decelerate earlier than the analogously low-mortality women do in the closed-group model with identical mortality parameters. The sick, on the other hand, might decelerate either earlier or later, in this open-group model, than the comparably high-mortality men in the closed-group model with identical mortality parameters but no increment. Most importantly for our purposes, mortality deceleration among the sick can occur either before or after mortality deceleration among the healthy in the open-group model.

Two additional observations can be made before turning to simulated data. First, the degree to which the increment of the newly sick changes the composition of the total sick depends in part on the size of the healthy group relative to the sick. The larger the healthy group, the more it will alter the sick—towards frailty or towards robustness. If the healthy group is very large, then proportionally small decrements from the healthy group may alter its composition little while changing the composition of the sick group substantially.

Second, it seems plausible that while the healthy group should mimic other binary-frailty closed groups in having at most one absolute deceleration, the sick group may decelerate more than once. This possibility arises from the interaction of two flows changing the sick group composition—the decrement to mortality and the increment from the healthy—and the fact that the direction of the effect of the latter increment may change as the composition of both groups changes.

To explore these possibilities, I simulate the model under a wide range of parameter values.

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[6] If the total decrement of the healthy group is greater than the mortality decrement in the sick group, this can happen while the sick group still has frail members—and hence can still decelerate—despite the equal proportion frail in the groups at baseline.

4 Simulation procedure

The purpose of the simulations is to compare the age at deceleration among sick and healthy groups who age according to the model just described. Each cohort is constructed as a set of two multistate life tables—one representing the frail subgroup, and one the robust—which never exchange members. The age-specific mortality of the sick and healthy groups is then constructed as the average mortality of their respective frail and robust members, weighted by the size of each subgroup. In constructing the life tables, I use standard formulas which assume constant mortality within each age interval (Preston et al. 2001: 46–47). Since this assumption is inconsistent with the model assumption of Gompertz mortality within each subgroup, I use age intervals of only one-tenth of one year, ranging over 100 yearly ages (producing 1001 age observations). All frail members in all cohorts are extinct by the end of the age interval, ensuring that all decelerations are observed.

The onset of mortality deceleration is defined as the point when the second derivative of mortality becomes negative. The first derivative of mortality is estimated as the two-sided average difference in mortality, and likewise, the second derivative is estimated as the two-sided average difference in the first difference.[7] Since this measure requires comparison points on each side of each age, the second derivative cannot be estimated for the first two and last two age units, leaving 997 age observations with estimated second derivatives of mortality for each group.

Since the model may be sensitive to the values of many parameters, it is desirable to evaluate specifically those groups whose mortality seems realistic for human populations. Yet many of the parameters of interest are putatively unobserved in empirical data, and so it is difficult to directly evaluate whether they are reasonable or realistic. The strategy I adopt, then, is to consider primarily those sets of groups that generate *aggregate* cohort mortality that resembles aggregate mortality of real cohorts in the Human Mortality Database (HMD).[8] To do this, I first allow the parameters to covary freely over a fairly wide range of values, producing 2,603,664 total simulated cohorts, each containing groups defined by frailty and robustness, sickness and

[7] In other words, the slope of mortality at age a is estimated as half of the difference between mortality at age $a + 1$ and mortality at age $a - 1$. I use the two-sided difference because I consider it to be the best measure for equivalent purposes in empirical research, since one-sided differences may exaggerate distortions arising from tempo effects (by simultaneously inflating and depressing mortality at adjacent ages, when more or fewer people than expected die during a given interval), while two-sided differences will smooth over such distortions. However, since the model here is deterministic (in the sense that there is no stochastic variation that might produce such tempo distortions), the choice between two-sided and one-sided difference estimates of the derivatives is arbitrary in this context.

[8] Human Mortality Database. University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at http://www.mortality.org or http://www.humanmortality.de (data downloaded on 18 August 2011). I use all cohort (vs period) data included in the HMD.

health.[9] I then estimate a Gompertz model on the full cohort produced by each set of groups, and an identical model on each cohort in the HMD.[10] Finally, I construct a parallelogram around the estimated Gompertz intercept and slope of the real data (a shape that, consistent with Strehler and Mildvan's (1960; see also Finkelstein 2012; Zheng et al. 2011) observation, fits well the distribution of these data), and keep only the 46,684 simulated cohorts whose aggregate slopes and intercepts fall within the parallelogram of the HMD slopes and intercepts. Of those simulated cohorts, 11,639 have a deceleration among both the healthy and the sick, allowing comparison of the age at each.[11] These 11,639 comprise the main sample in the paper because, following the standard practice in empirical research on deceleration, the main object of interest is the direction and magnitude of the difference between the age at sick and at healthy deceleration. Since this measure fails to draw information from cohorts in which only one, or neither, group decelerates, this outcome should not be considered a comprehensive look at deceleration dynamics in an open-group model, but rather an example of what empirical researchers would conclude if they applied standard

[9] Specifically, I run the models on all combinations of these parameter ranges: $\alpha_\mu = [0.0031, 0.0101]$ in units of 0.001, as well as 0.00001, 0.00005, 0.0001, 0.0005, 0.0101, and 0.0201; $\beta_\mu = [0.05, 0.125]$ in units of 0.015; $f_\mu = 2,\ 3$; $m = 2,\ 3$; $\alpha_\omega = \{.00001, 0.00005, 0.0001, 0.0005, 0.0101, 0.0201\}$; $\beta_\omega = [0.05, 0.125]$ in units of 0.015; $f_\omega = 2, 3$; $\pi(0)$ for both groups $= [0.3, 0.9]$ in units of 0.1; and the baseline proportion healthy $= [0.3, 0.9]$ in units of 0.1.

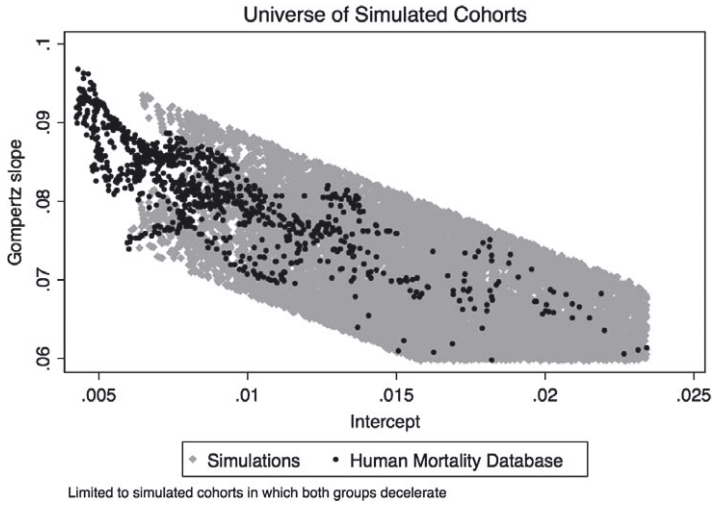
These parameter values were obtained by selecting an initial set that previous research with a closed-group model (Wrigley-Field 2014) suggested might yield realistic aggregate values and then iteratively augmented (i.e. extended through trial-and-error) to find more cohorts that fell close to the HMD data. No parameter choices were made with regard to their likely deceleration outcomes.

The range of values of the baseline proportion frail at each group is chosen to exclude only groups whose baseline frailty is too low to make any deceleration likely. Previous research (Wrigley-Field 2014) shows that, in the closed-group version of this model with baseline frailty set to 75 per cent, absolute decelerations tend to occur when the frail make up between about one-third and two-thirds of the cohort.

[10] For the purposes of this comparison, I consider each model life table to range over the ages 50 to 150. This choice does not affect the life table calculation, since the life tables are calculated with the baseline age rescaled to zero, but it does affect the interpretation of the parameters. The model therefore assumes that each disaggregated subgroup has Gompertz mortality from mid-life onward, but makes no assumption about earlier life. Likewise, the model assumes that a relatively high percentage of each group is frail at age 50. This high per cent frail at age 50 does not impose an assumption of unduly high frailty at birth because, on the model given here with lifelong Gompertz mortality at the individual level, mortality selection overwhelmingly occurs at higher-mortality older ages. (The reasonableness of this assumption is discussed in Wrigley-Field (2014).) When I estimate the aggregated Gompertz models in the model cohorts and the HMD cohorts, I restrict the age range to 50–100 to match the availability of real data.

[11] Of the 46,484 cohorts, 26,570 (57%) show no deceleration in either group; 4,887 (10%) show deceleration only among the sick; 3,588 (8%) show deceleration only among the healthy; and so the 11,639 with decelerations among both groups—the cohorts analysed in this paper—are 25 per cent of the total.

Figure 2:
Aggregate parameters of the universe of simulated cohorts and the Human Mortality Database cohorts



closed-group reasoning in an open-group context.[12] Figure 2 shows the aggregate Gompertz parameters of these simulated cohorts.[13]

Finally, to provide a basis for comparison, I construct the two alternative models. First, I construct a set of closed-group models based on the subset of 'realistic' open-group models. For each set of mortality parameters included in that set of open-group models, I construct a closed-group model with those mortality parameters, but with the parameters describing the flow from sickness into health set to zero (in other words, the equivalent of the model for women and men given in Equations (1) and (2)). This produces a set of 1,307 closed-group models, of which 181 show a

[12] The outcome used here also loses some information to left truncation (if we do not think of baseline age as birth): some groups already have a negative second derivative of mortality at the baseline age. This is most common among sick groups: of the 11,639 cohorts considered here, 2,125 of the sick groups, but only 10 of the healthy groups, have already decelerated at the baseline age. Indeed, among cohorts in which the sick decelerate twice, the first deceleration overwhelmingly appears at baseline age. However, the *order* of deceleration is truncated only in the ten cohorts in which both groups have a negative second derivative at the baseline age.

[13] No simulations in the final set of simulated cohorts correspond to the upper left region of the HMD parallelogram, which corresponds to the most recent cohorts. This region is largely populated by simulated cohorts but, at the parameter combinations used here, those cohorts do not show decelerations among both groups.

deceleration among both groups.[14] These 181 closed-group cohorts are compared to the open-group cohorts in which the healthy and sick decelerate.

Second, I construct a set of models in which the frail and robust have equal rates of transitioning to sickness—that is, the frailty multiplier on sickness, f_ω, is set equal to 1—although the frail have higher mortality in each sickness group. I create one such model for each combination of parameters (except the frailty multiplier on sickness) in the main set of 'realistic' models, producing 24,083 comparison models with equal sickness rates. Of these, 4,373 show a deceleration among both groups, and are compared to the main models.

5 Simulation results

In all models, the main outcome of interest is the sign (i.e. deceleration order) and magnitude of the difference between the sick and healthy groups' ages at deceleration. To provide a basis for comparing the open-group model results, which are the results of main interest, I begin by examining the 181 closed-group models in which both the healthy and the sick decelerate. As expected, in all cases the higher-mortality sick decelerate at a younger age than the lower-mortality healthy. As summarised in the first row of Table 1, the difference between the age at healthy deceleration and the age at sick deceleration ranges from 7.5 to 22.1 years, averaging 12.7 years. (Table 1 also reports median differences; none of the age difference distributions are badly skewed.)

The key quantity of interest in this paper is the difference between the healthy and the sick age at deceleration in the open-group model in which the frail are more likely to become sick, as well as to die. In short, as summarised in Table 1, in this model, it is no longer the case that the sick always decelerate at younger ages than the healthy; nor is the reverse reliably the case.

An immediate complication is that of the 11,639 cohorts in the open-group model in which both groups decelerate, 1,425 cohorts (12%) show two distinct intervals of deceleration among the sick, with a return to accelerating mortality in between. To avoid any distortion in the results arising from comparing two deceleration points among the sick to one among the healthy, Table 1 reports separately the difference between healthy and sick deceleration among cohorts in which the sick decelerate only once; first sick decelerations compared to the single healthy deceleration, among cohorts in which the sick decelerate twice; and, among those cohorts, second sick decelerations compared to single healthy decelerations.

[14] The number of closed-group models—1,307—is substantially smaller than the number of HMD-compatible open-group models—46,684—because the latter include many sets of cohorts that share mortality parameters but vary in sickness parameters. Each such set of open-group models corresponds to a single closed-group model.

Table 1:
Age at deceleration in healthy vs sick groups

Model	Sick decel.	Difference in deceleration age in healthy vs sick groups (in years)				Proportion of cohorts in which sick group decelerates before healthy group
		Min	Max	Mean	Median	
Closed-group model (n = 181)	Single (n = 181)	7.5	22.1	12.7	11.6	1
Main model:						
Open-group model with	Single (n = 10,214)	−36.8	22.6	−4.6	−4.4	0.27
frailty multiplier on	First (n = 1,425)	−14.8	33.9	11.4	10.6	0.99
sickness >1 (n = 11,639)	Second (n = 1,425)	−39.3	3.8	−10.6	−8.4	0.02
Open-group model	Single (n = 4,357)	−4.5	52.8	10.9	9.5	0.97
without frailty multiplier	First (n = 14)	8.5	12.4	11.4	11.7	1
on sickness >1 (n = 4,373)	Second (n = 14)	−6.4	−3.1	−5.1	−5.1	0

Note: All results in Table 1 are for simulated cohorts in which both healthy and sick groups decelerate. In all cases, the healthy group decelerates only a single time, so all sick decelerations (single, first, or second) are compared to the single healthy deceleration in the cohort.

Among those 10,214 simulated cohorts in which the sick decelerate a single time, as shown in the second line of Table 1, the difference between the age at healthy deceleration and the age at sick deceleration ranges from −36.8 to 22.6 (averaging −4.6). In other words, on average, the healthy decelerate at a *younger* age than the sick, with the sick decelerating first in only 27 per cent of cohorts. This is the opposite of what we would expect from a naive extension of the closed-group generalisation.

The third and fourth lines of Table 1 compare healthy and sick ages at deceleration when the sick decelerate twice. Among these cohorts, comparing the *first* sick deceleration to the single healthy deceleration, we find that the difference in ages ranges from −14.8 to 33.9 (averaging 11.4). Thus, here, too, the groups may decelerate in either order, although the first sick deceleration occurs before the single healthy deceleration in 99 per cent of cohorts. Comparing the *second* sick deceleration to the single healthy deceleration, we find that the difference between healthy age and sick age at deceleration ranges from −39.3 to 3.8 (averaging −10.6, or median −8.4; this age difference has the most skewed distribution). The positive maximum difference shows that in the open-group model, it is possible for the sick group to decelerate twice before the healthy group has decelerated. But 98 per cent of second decelerations among the sick do occur after the healthy have decelerated. In short, when the sick decelerate twice, their decelerations nearly always fall on either side of the healthy group's deceleration—but it is possible, instead, for both sick decelerations to occur before or after the healthy deceleration.

5.1 An open-group model with equal sickness rates

For comparison to the main model used in this paper, consider the open-group model that drops the assumption that the frail become sick at a higher rate than the robust. The last three lines of Table 1 summarise the deceleration order among the 4,373 cohorts with equal sickness rates between the frail and robust in which both the healthy and sick groups decelerate. When the sick decelerate only once, 97 per cent of the time, this deceleration occurs before the healthy deceleration, by 8.5 years on average. When the sick decelerate twice, in *all* cohorts considered here, the sick decelerate for the first time before the healthy, and for the second time afterward. These cohorts are rare: in the model with equal frail and robust transitions to sickness, only 14 cohorts (0.3%) have two sick decelerations.

In summary, it is not necessary for the frail to transition between groups at a higher rate than the robust to have mortality deceleration patterns in an open-group model that are different from those of the equivalent closed-group model in two respects: the healthy may decelerate at a younger age than the sick, and the sick may decelerate twice. But when the frail and the robust transition between groups at an equal rate, such patterns are very rare in the cohorts modelled here.

5.2 How predictable is open-group deceleration order?

The results presented so far demonstrate that the open-group model considered here does not conclusively predict the order of deceleration between groups, as the closed-group model does. We might also wonder to what extent the order of deceleration fluctuates with small changes in parameter values. If such fluctuations are rare, then the deceleration order might still be useful in an open-group context, as long as parameter values can be estimated (from data) or assumed (in the case of latent parameters) with reasonable precision. But if the deceleration order fluctuates wildly with variation in parameter values, it is unlikely to be useful as a test of an assumed selection model.

As a first step towards exploring the sensitivity of deceleration order to parameter values in the open-group model, I focus on the relationship between the baseline per cent healthy and the order of deceleration between the healthy and the sick. I choose the baseline per cent healthy as the parameter of interest because, unlike parameters that are specific to frail or robust subgroups, it is observable in empirical data—hence a potentially useful basis for empirical predictions of deceleration order in an open-group context. Moreover, as explained in the 'Intuition and Hypotheses' section above, I expect the baseline per cent healthy to play an important role in amplifying the selection dynamics associated with the movement from health to sickness.

To analyse the relationship between baseline per cent healthy and deceleration order, I construct 7,961 sets of at least two cohorts that share all parameters except their baseline per cent healthy and examine the deceleration order within those sets.

(When the sick group decelerates twice, I use the first sick deceleration.) In 11 per cent of these sets, the order of deceleration switches, with the healthy decelerating first at some values of baseline per cent healthy, and the sick decelerating first at other values.[15]

Such deceleration order switching occurs at all measured values of the baseline per cent healthy, but it tends to occur at very high values—86 per cent on average. When the order of deceleration does switch within a set of cohorts sharing all parameters except the baseline per cent healthy, in 94 per cent of cases, the cohorts with the lower baseline per cent healthy have the sick group decelerating at a younger age, while those with the higher baseline per cent healthy have the healthy group decelerate first. To the extent that delayed mortality deceleration among the sick reflects frailty replenishment from the healthy, it is sensible that a larger healthy group would yield a later age of mortality deceleration among the sick.

In one set of cohorts, the order of deceleration switches twice: the healthy decelerate before the sick when the baseline proportion healthy is 0.8, and the sick decelerate first when it is larger or smaller.[16] To further explore the contribution of the baseline per cent healthy for this set of cohorts, I model cohorts with this set of parameter values at baseline healthy values ranging fully—from 0.5 to 99.5 per cent—in units of half of one per cent. Figure 3 shows the age at sick deceleration for these cohorts over the baseline per cent healthy. The horizontal line at age 86.8 represents the age at healthy deceleration (constant across the cohorts, since mortality deceleration among the healthy is unaffected by the size of the healthy group). At values of the baseline per cent healthy below 72.5 per cent and above 90 per cent, the sick decelerate before the healthy. At values of the baseline per cent healthy between 72.5 per cent and 90 per cent, the healthy decelerate first. Such sensitivity of deceleration order to the baseline per cent healthy requires fairly precise consideration of the latter to interpret the former.[17]

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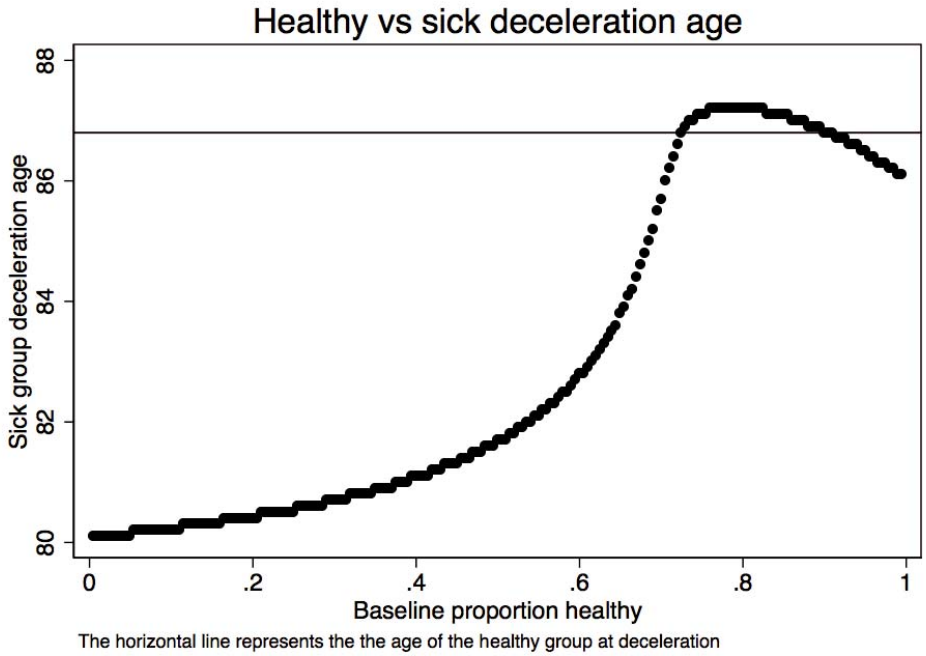
[15] Because this analysis is restricted to cohorts in the subset with similar aggregate parameters to the HMD cohorts, the sets contain varying numbers of cohorts, with a maximum of seven (corresponding to seven modelled values of the baseline per cent frail, ranging from 0.3 to 0.9). The frequency of deceleration order switching given—11 per cent—corresponds to sets containing at least two cohorts. Because the sets are modelled over only a partial range of the proportion frail, and are further limited to values generating aggregate cohorts compatible with the HMD, I believe that the measure used here of the frequency of order switching is conservative.

[16] These cohorts have aggregate Gompertz parameter values similar to mid-19th century Iceland and late-19th century France. Their subgroup parameter values are: $\alpha_\mu = 0.0031$; $\beta_\mu = 0.08$; $f_\mu = 3$; $m = 2$; $\alpha_\omega = 0.0001$; $\beta_\omega = 0.11$; $f_\omega = 2$; $\pi(0) = 0.9$.

[17] Since the sets here consider only cohorts with aggregate parameters similar to the HMD, order-switching over values of the baseline per cent healthy may appear less prevalent than it would with a more permissive universe of cohorts, and hence, larger sets of cohorts for comparison. Sets of cohorts can switch deceleration orders twice only if they contain at least three cohorts. 753 sets contain only two cohorts. Of these, 13 per cent have a single order switch.

Figure 3:
Age at mortality deceleration among the sick group as a function of the baseline per cent healthy, compared with age at deceleration among the healthy group



6 Conclusion

There is a long tradition in demography of understanding that population composition alters and changes the interpretation of aggregate population behaviour, dating back at least to Vaupel et al. (1979) and Vaupel and Yashin (1985). Most of this research has taken place in a closed-group context, and we now understand a great deal about the properties of population composition and selection in that context. There also is a smaller body of work on population dynamics that incorporates the insights of this classic body of mortality selection research (e.g. Manton et al. 1994, 1995; Woodbury and Manton 1983), and this area should expand in the future, with the explosion of longitudinal health data. But this study suggests some unforeseen difficulties in integrating open-group models with one of the most important indicators of cohort heterogeneity: mortality deceleration.

The results in this paper demonstrate that the deceleration dynamics of open groups do not lend themselves to simple generalisations. Among closed groups, the higher-mortality group always decelerates at a younger age than the lower-mortality group, all else being equal. In open groups, however, either deceleration order is

possible. Moreover, the order of deceleration can be sensitive to relatively small changes in parameter values, as in the perverse set of cohorts illustrated in Figure 3, in which the deceleration order switches back and forth as the baseline per cent healthy increases.

The lack of reliable generalisations about the order of mortality deceleration in an open-group context poses a challenge to the usefulness of measurements of deceleration order. This, in turn, renders less promising one of a relatively small number of methods for testing selection theory and drawing inferences about unobserved heterogeneity in mortality risk.

Deceleration order is informative when it violates the predictions made by selection theory. Such a violation tells analysts that one of two things is going on. One possibility is that although the deceleration arises from selection, the *ceteris paribus* conditions for the predicted deceleration order are not met, i.e. there is some other difference in heterogeneity between the groups (as Lynch et al. 2003 conclude about African-Americans and white Americans). The other possibility is that deceleration does not arise solely or primarily from mortality selection, but rather from deceleration in individuals' rate of ageing, as prominent biodemographers have argued (e.g. Mueller et al. 2011; and see reviews in Vaupel 1997, Wachter and Finch 1997). Conversely, deceleration orders that fail to violate the predictions of selection theory are often considered to lend some support to selection theory (e.g. Horiuchi and Wilmoth 1998).

Such inferences are possible only when selection theory generates a clear prediction about which group should decelerate at an earlier age than another. The results in this paper demonstrate that when we turn from comparing closed groups, such as birth cohorts, to open groups, such as health statuses, then—particularly when frailty may increase (or decrease) the risk of transitioning between groups—selection theory no longer makes such a clear prediction. This renders the order of deceleration uninformative about heterogeneity without reliable information about underlying parameter values.

This paper provides a preliminary consideration of deceleration order between open groups. An important question that the paper does not address is whether these results generalise beyond the binary frailty model used here to continuous frailty models, particularly the widely-used gamma-Gompertz model (Gampe 2010, Horiuchi and Wilmoth 1998, Missov and Finkelstein 2011, Steinsaltz and Wachter 2006, Vaupel et al. 1979). Future research should also explore the sensitivity of these results to the functional form, over age, of the risk of becoming sick. Whether or not open-group deceleration dynamics turn out to be sensitive to model choice, however, these results show that in the binary context, they are sensitive to latent parameter values. A practical implication is that if deceleration order in an open-group setting is used either to test selection theory or to derive conclusions about cohort heterogeneity, the sensitivity of the conclusions to model assumptions should be explicitly modelled. It may be the case that in specific open-group settings, such sensitivity analyses would reveal that some conclusions from deceleration order are well-supported. Based on the results presented here, such conclusions should not be assumed to be warranted in general.

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