Healthy life expectancy, mortality, and age prevalence of morbidity

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March 3, 2017

Abstract

In calculating period healthy life expectancy, the use of age-specific morbidity prevalence patterns assumes that age captures the important time-variation in the given health condition, i.e. that the disabling process is related to how long an individual has lived. However, many common disabling processes are better measured by a time-to-death pattern. At advanced ages the conflation of an increasing chronological-age mortality pattern and a time-to-death morbidity pattern produces an apparent morbidity pattern that also increases with advancing age. Differences in period healthy life expectancy over time or between populations cannot easily be partitioned into morbidity and mortality components because the period morbidity pattern may depend on an unknown future time-to-death process not captured by period mortality. We illustrate these concepts formally and empirically, using morbidity data from the U.S. Health and Retirement Study. While holding the time-to-death morbidity pattern fixed, we show that mortality reduction alone reduces the total life years with disability. We estimate the magnitude of this bias for different disabling processes. This has implications for any between- or within-population comparisons of period healthy life expectancy conditioned on different age patterns of mortality.

1 Introduction

Healthy life expectancy (HLE) is among the most widely used metrics of population health. It combines information on mortality and morbidity to summarize the expected years of life lived in good health, however measured. If healthy life expectancy increases faster than life expectancy, morbidity is compressed into a smaller proportion of life. HLE can increase because of changes mortality, morbidity, or both.

In calculating HLE, the use of age-specific morbidity prevalence data implicitly assumes that a chronological age pattern best characterizes variation in the health characteristic over the lifespan. However, Riffe, T., Chung, Spijker, and MacInnes (Riffe, T. et al.) show that many common disabling processes are better measured by a pattern over time-to-death (TTD) or by both age and TTD. Most morbidity patterns increase with age, and can claim empirical regularity in this regard. However, we explain how the observed shape of a morbidity age-curve can change due to changes in mortality even with no underlying change in morbidity. At advanced ages the conflation of an increasing chronological-age mortality pattern and a TTD morbidity pattern produces an apparent morbidity pattern that also increases with advancing age.

In the cohort perspective the true and apparent morbidity patterns imply the same HLE. Problems arise in the period perspective. Differences in period HLE over time or between two populations cannot easily be partitioned into morbidity and mortality components, because the period morbidity component will depend on some unknown future TTD process. For the same reason, comparisons of disability prevalence rates by age are not recommended between populations with different underlying age schedules of mortality.

In this paper we illustrate these concepts formally and empirically, using morbidity data from the U.S. Health and Retirement Study (HRS 2013). While assuming a fixed TTD morbidity function, we show that mortality reduction alone can reduce the total life years with disability (DLY). We estimate the magnitude of potential biases for different disabling processes, given different levels of mortality extracted from the Human Mortality Database (HMD 2015). We first explain how the age pattern of morbidity may partly be a function of mortality using both formulas and a schematic illustration.

2 Morbidity as a function of time-to-death

Imagine a bad health condition, G, with prevalence that varies as a function of time-to-death, y, and not as a function of chronological age, a. Since the TTD prevalence distribution in older ages is something close to exponential, there will still be an apparent age function, $g^\star(a)$. In this case $g^\star(a)$ is a heterogeneous aggregate based on both mortality and the underlying TTD process:

$$g^{\star}(a) = \frac{\int_0^{\omega} g(y)N(a,y)\ \mathrm{d}y}{N(a)} \tag{1}$$

$$= \frac{\int_0^{\omega} g(y)N(a)\mu(a+y)\frac{\ell(a+y)}{\ell(a)}\ \mathrm{d}y}{N(a)} \tag{2}$$

$$= \int_0^{\omega} g(y)f(y|a)\ \mathrm{d}y \qquad , \tag{3}$$

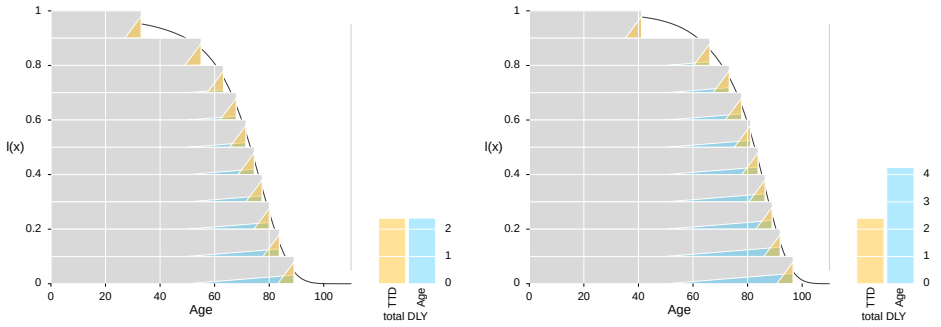
where $N(a)$ is the population aged a, $\ell(a)$ is lifetable survivorship, and $\mu(a)$ is the force of mortality. $f(y|a)$ is the conditional remaining-years distribution, which gives the probability of dying in y years given survival to age a. The expression (3) says that the proportion of those in age a that has condition G does not depend on population structure at all, but only on future mortality rates and the TTD pattern of G, $g(y)$.

A function such as $g(y)$ would have implications for the interpretation of period age patterns of morbidity, and by extension, HLE. If a function such as $g(y)$ holds, it is tautologically true that the measurement of HLE in completed cohorts (or stationary populations) will be identical whether calculated on the basis of $g^{\star}(a)$ or the underlying $g(y)$ pattern. Distortions only arise in the interpretation of period HLE under changing mortality, or with period HLE comparisons between populations with different mortality.

Since morbidity prevalence in this scenario is partly a function of mortality, the age patterns of morbidity for populations with different mortality levels or patterns cannot be compared without additional information. Under these circumstances, it is also deceptively tricky to partition period HLE differences into underlying morbidity and mortality components, because the morbidity component is (arithmetically) a function of an uncertain future mortality pattern that accompanies the apparent age pattern of morbidity. Although cohort HLE (a gold standard) is theoretically unbiased (Imai and Soneji 2007)[1], and therefore comparable, this quantity cannot be faithfully decomposed into morbidity and mortality components based on age patterns of morbidity and mortality alone if the underlying morbidity pattern is a function of time-to-death.

A toy example serves to illustrate these concepts. Figure 1 provides a schematic overview of two stationary populations. The underlying survival pattern of these two populations is based on period survival curves from Japanese males in 1970 (a) and 2010 (b) (HMD), but the reader may imagine these as two hypothetical populations. Population (a) has a life expectancy of 69.3, while population (b) has a life expectancy of 79.5, slightly more than 10 years higher. For demonstration, we partition each survival

[1]We confirm that this remains so even if morbidity prevalence in strongly patterned by time-to-death.



(a) Higher mortality setting (b) Lower mortality setting

Figure 1: Schematic survival curves from higher (1a) and lower (1b) mortality populations. Each population is subjected to the same chronological age (blue) and time-to-death (yellow) morbidity prevalence patterns. The total prevalence of each sums to disability life years (DLY), drawn on the right of each survival curve. In 1a the age and time-to-death prevalences imply the same DLY. In 1b the time-to-death DLY is identical to 1a, but the age DLY is two years higher, due entirely to improved longevity.

curve into 10 lifespan quantiles, represented with horizontal bars. Our simple TTD prevalence, $g(y)$, is drawn with identical yellow triangles at the end of each lifespan bar. Onset begins 5 years before death and culminates with 80% prevalence. The chronolgical prevalence function is drawn with blue triangles, with onset at age 50 reaching a maximum prevalence of 50% at hypothetical age 111.5. Both prevalence functions are identical for populations (a) and (b).

The resulting disability life years (DLY) is shown with barplots next to each stationary population. In population (a) the age and TTD prevalence functions yield the same DLY (and HLE). In population (b) the time-to-death DLY is identical to population (a), but the age DLY is nearly twice as high. For the age prevalence function, it is correct to conclude that increased longevity leads to increases in prevalence, but for the TTD prevalence function there is no morbidity-mortality trade-off. Instead, improved longevity leads to increased proportions of life lived disability-free, albeit with no change in the absolute concentration of morbidity in the final years of life. Analyses based on the standard Sullivan method (Sullivan 1971) are only capable of predicting increased DLY when projecting from the mortality of (a) to (b). This is so for both kinds of morbidity because the TTD prevalence pattern is erased when the same condition is measured over age. The same Sullivan method can also only conclude that the morbidity of the TTD process is more compressed in (b) than in (a), even though its essential character is unchanged. Prevalence functions are in fact more nuanced than those presented here, but our example provides a useful heuristic to

understand a previously-undescribed source of bias in common applications of the Sullivan method.

3 How changing mortality affects morbidity prevalence

The age pattern of morbidity prevalence observed in the cross-section (in older ages) depends on the extent to which prevalence is principally described by age versus TTD and on the underlying mortality level. If the prevalence is principally a function of TTD, the specifics of its shape are also important. In Figure 2 we show the age-translations of different TTD prevalence patterns when interacted with fictitious stationary populations under different mortality levels.

The first column contains three different types of disability, all of which are experienced by half of the population at the time of death, but which differ in the timing of onset prior to death and in the steepness of the curve with the approach to death. The first type of disability is virtually nonexistent 5 years prior to death, but then increases very rapidly as death approaches. The middle variant of disability is rare 15 years before death, but increases to about 20 percent of the population 5 years before death and rises sharply thereafter. The bottom figure depicts a disabling process that although still strictly determined by time-to-death, is common and accumulates very slowly starting from about 50 years before death.

The second column translates the time-to-death disability prevalence curves into the "apparent" chronological age prevalence of disability for different mortality levels depicted by the death density curves above. These mortality levels roughly correspond to USA males in 2002 $e_{60} = 20.0$ years, Canadian females in 2004 $e_{60} = 25.0$ years, and projected Japanese females a decade or so from now $e_{60} = 30.0$ years (latest observed level in 2012 was $e_{60} = 28.3$ years (HMD 2015)). In all cases increasing remaining life expectancy results in decreasing age-specific disability prevalence by chronological age. With steeply increasing disability prior to death (first row), the differences in disability prevalence are largest above age 80, where the bulk of mortality occurs, while with more gently increasing disability (second and third rows) the greatest differences in disability age-prevalence curves appear at younger ages.

These differences induced by mortality alone are not trivial. In the middle variant, which closely resembles the TTD prevalence of disability in bathing, a 10-year increase in e_{60} results in a 50 percent drop in disability prevalence at age 80 from around 20 to 10 percent. Meanwhile, the age at which a quarter of the population were considered disabled in this scenario differed by about 5 years with a 5-year improvement in e_{60} from 20 to 25.

Of course the disability prevalence is rarely a function of thanatological

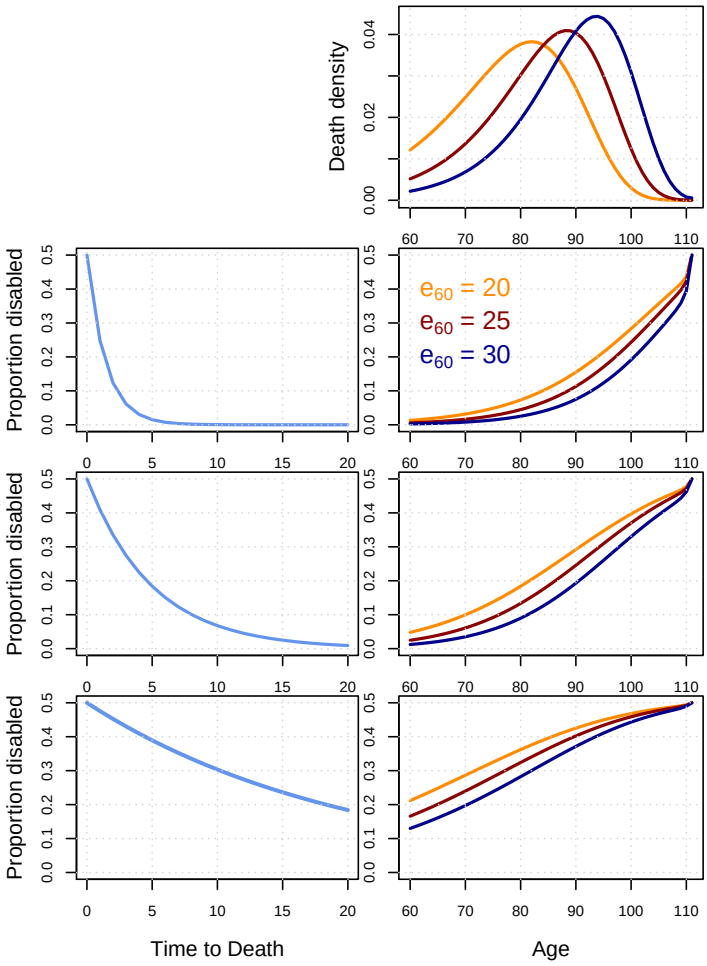


Figure 2: The age pattern of morbidity prevalence in ages 60+ derived from interacting different TTD prevalence patterns (left column) with fictitious stationary populations subject to the color-coded death distributinos depicted in the top right figure.

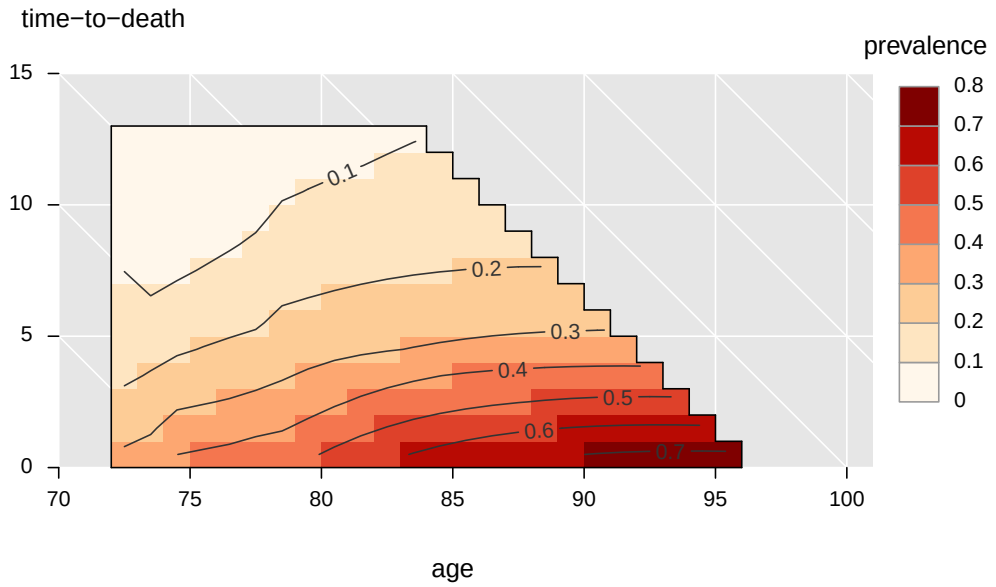


Figure 3: Proportion of males from the 1915-1919 cohort with at least one (of five) instrumental activity disabilities, by time-to-death and age.

age alone. For example, Figure 3 shows US male IADL-disability prevalence broken down by age and TTD. The horizontally running gradients indicate that time-to-death is a more important time axis to predict deterioration in IADL than time since birth. Thus our assumption of fixed morbidity prevalence on the time-to-death axis is not unrealistic, at least for some characteristics. This is the case for many, but not all, indicators of health and disability (Riffe, T., Chung, Spijker, and MacInnes Riffe, T. et al.). In reality, the prevalence of disability varies on both the age and time-to-death axes. The degree to which prevalence is better measured by age or time-to-death for different disabling conditions remains an open research question.

4 Estimating bounds for the impact of mortality differences on estimates of disabled life years

The Sullivan method is the most commonly used method to partition life expectancy into estimates of the total life years lived in a state of good health (HLY) or disability (DLY). Its popularity owes to its minimal data requirements. Only current age-specific disability prevalence rates are needed in addition to a life table or age-specific mortality rates. Specifcally, the number of person-years with disability ${}_n\pi_x \cdot {}_nL_x$ in age-group x to $x+n$ are the product of the person-years lived from the life table ${}_nL_x$ and the proportion

disabled ${}_n\pi_x$. The total DLY is the sum of ${}_n\pi_x \cdot {}_n L_x$ over all age groups (Sullivan 1971).

It has long been recognized that the Sullivan method does not produce a pure synthetic measure of health expectancy, since it combines flow data of current mortality incidence with stock data of morbidity prevalence. While the mortality flow responds immediately to period change, for instance from medical innovations, the morbidity stock is slower to change because it reflects past cohort experiences with disability incidence and recovery (Mathers and Robine 1997, Barendregt et al. 1994). Moreover, as was illustrated in Figure 2, the prevalence at any age may not only depend (mathematically)) on past transitions into and out of disability but also on future deaths.

Nevertheless, comparing populations on the basis of life years lived in a state of good health (HLE) or disability (DLY) is standard practice in population health. The difference in either metric, either a within-population difference over two time periods or a between-population difference in the same time period, is often decomposed into mortality and morbidity components on the basis of differences in survivorship and morbidity age-prevalence respectively (Nusselder and Looman 2004, Andreev et al. 2002). According to Nusselder and Looman (2004), the corresponding mortality and disability effects at each age group for a within-population decomposition are:

$$ {}_nMOR_x = \left[\frac{{}_n\pi_{x(t)} +_n \pi_{x(t+y)}}{2}\right] \cdot \Delta_n L_x \tag{4} $$

$$ {}_nDIS_x = \left[\frac{{}_nL_{x(t)} +_n L_{x(t+y)}}{2}\right] \cdot \Delta_n \pi_x \quad , \tag{5} $$

where Δ refers to the change in the variable from time t to $t+y$, ${}_n\pi_x$ is prevalence in the discrete age group $[x, x+n)$, and L_x is lifetable exposure. Discrete prevalence ${}_n\pi_x$ is essentially our $g^\star(a)$ from equation (3). The sum of the two components over all ages is equal to the total change in DLY. These formulas are essentially an application of standard Kitagawa (1955) decomposition.

Although it is true that these two components arise from changes in survivorship and morbidity prevalence respectively, difficulties arise in the interpretation of the components as pure mortality and disability effects. If the time-to-death profile of disability prevalence does not change over different mortality regimes, inducing mortality decline alone will result in declines in the age-pattern of prevalence component, as we demonstrated. This method of decomposition will therefore still attribute a portion of the change in DLY to morbidity, and we consider this to be a source of bias.

To get a sense of the upper magnitude of this bias, we test how the disability component of this decomposition method would change when a fixed time-to-death prevalence of morbidity is applied to different mortality regimes using empirical data. Morbidity data come from the US Health and

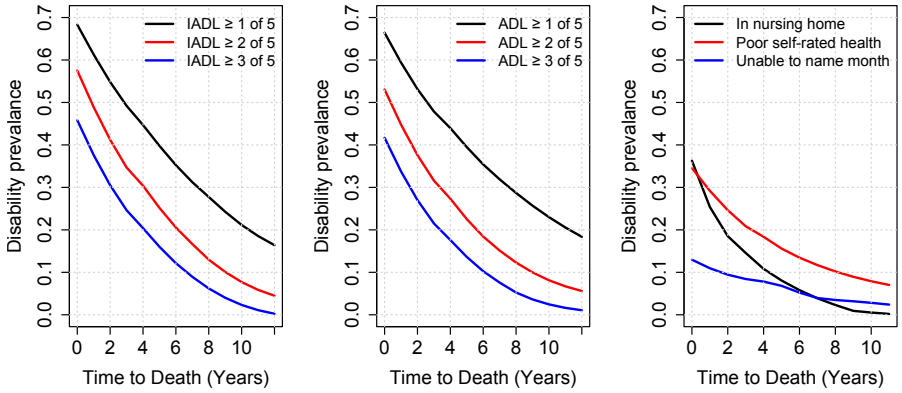


Figure 4: The disability prevalence by time to death for various disability types, based on American HRS data (females).

Retirement Study while mortality and exposure data come from the Human Mortality Database (HMD 2015).

Specifically, we consider the age prevalence of difficulties in carrying out at least 1, 2, or 3 (out of 5) functional Activities of Daily Living (ADL), difficulties in carrying out at least 1, 2, or 3 (out of 5) instrumental Activities of Daily Living (IADL), living in a nursing home, having poor self-rated health, and being unable to name the month of the year. We estimate these time-to-death prevalences for quinquennial cohorts from 1905 to 1930, separately for males and females. We calculate the time-to-death $g(y)$ profiles of disability prevalence for all extinct lifespans above age 65 for each cohort. Estimation is done via a loess smooth over age, time-to-death, and birth cohorts. We then average the time-to-death profiles of disability over lifespans and over the 6 cohorts. The time-to-death prevalence of morbidity for each disability type is shown for females in Figure 4.

We calculate the apparent period chronological age prevalence of morbidity, $g^\star(a)$, for all medium to large populations of the Human Mortality Database, had they experienced the US time-to-death profile of morbidity. Eastern European countries are excluded from this exercise due to widely varying age patterns of mortality, particularly in the years surrounding political transition. To calculate the age-pattern of morbidity, we assume the survival pattern of each lifetable to be a stationary population and apply a discretization of equation (3).

We then make pair-wise comparisons of DLY between each population in the same year for the years 1980, 1990, and 2000. For within-country comparisons, we compare each population in 10-year jumps, for all years starting from 1950. Altogether this leads to 187 within-population comparisons and

1785 between-population comparisons for each sex. Finally we decompose the change or difference in DLY between the population pairs into mortality and morbidity components using the Nusselder and Looman (2004) method described above and in Equations (4) and (5). The true value of the change in DLY and the true value of the disability component are both zero by design. Thus, the estimated disability component from this decomposition gives a gauge of bias.

We compare the association between the change in the disability component (Equation 5) and the increase or difference in remaining life expectancy at age 60 for each population pair in Figure 5. By doing so we aim to provide a rough empirically-based estimate of the upper bound of the change in the disability component that is attributable to the different underlying mortality levels of any two populations being compared. Thus if female e_{60} increases in a country by 5 years, up to about 1 year of the reduction in DLY that is attributed to the disability component could be solely arising from the decrease in mortality, in the case where disability is measured as having difficulty in at least three ADLs. Departure from this upper bound in real life depends on the degree to which the disability prevalence changes on a time-to-death versus chronological age axis, the extent to which the US average time-to-death prevalence is representative, and the departure from the stationary population assumption.

Overall, the relationship between the change in disability component and the increase in e_{60} is strikingly linear although the slopes differ for males and females and by disability type. To some extent this is because the final level of disability prevalence (i.e. in the final year of life) differs by disability type. In Appendix Figure 6 we standardize for the maximum disability prevalence to give a clearer comparison between disability types. Even after standardization, the change in the disability component is greater with larger e_{60} differences for disability types with more gradually changing prevalence levels by time-to-death and for females who likewise have more gradually changing prevalence levels by time-to-death. This is because steep time-to-death profiles of disability concentrate disability prevalence near the death distribution, whereas gradually changing (but still entirely time-to-death) disability profiles spread out disability prevalence over a wider range of ages before each age at death.

If projecting morbidity prevalence, one can also refer to these results as a heuristic on the bias inherent in assuming a fixed age-pattern of morbidity.

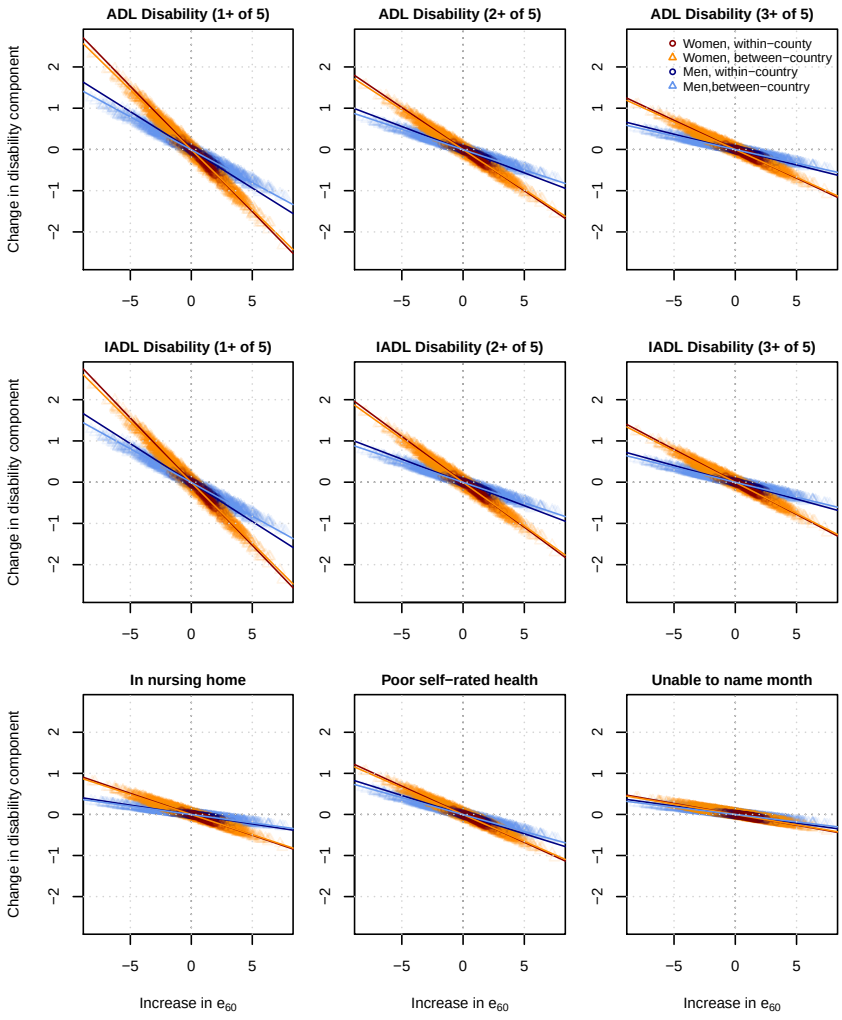


Figure 5: Results of the hypothetical decomposition exercise. The size of the morbidity component using a standard decompositon method is plotted against the difference in remaining life expectancy at age 60 (e_{60}) in each pair of populations. Linear trend lines are also provided for each sex and decomposition type.

5 Discussion

Healthy life expectancy remains a popular tool for analyzing population health. At any given time, a snapshot of the life years lived in good or poor health is captured. This information is well-summarized in both the period and cohort perspective, regardless of whether morbidity prevalence is a function of chronological age or time until death. Difficulties arise in the interpretation of period differences in this quantity. The chronological age pattern of disability can increase or decrease solely as a function of mortality change even when the underlying morbidity function is held constant. Thus, for instance, observed widening ratios in the age profiles of disability prevalence between subgroups (Crimmins and Saito 2001) cannot be attributed to changes in the disabling process without taking into account changing mortality profiles. This observation calls into question the practice of forecasting observed age-specific rates of decline in disability (Manton et al. 2006, Khaw 1999), and especially the more common practice of holding age-patterns of disability fixed in morbidity projections. Health economists refer to a similar 'red herring' argument, namely that medical costs are more closely associated with time-to-death than with chronological age. As a result, health care cost projections based on a chronological age rather than time-to-death pattern of expenditure are artificially inflated (Zweifel et al. 1999, Geue et al. 2014).

Instead, we argue that the better way to measure changes in health or disability is from a cohort perspective (Manton and Land 2000, Manton et al. 2008, Christensen et al. 2013). Manton and Land (2000), for instance, found large differences between period and cohort estimates of active life expectancy (ALE). ALE at ages 65 and 85 was between 1.6 and 2.6 times larger in the cohort perspective than for similar period estimates, and the expected years of life disabled were smaller in the cohort perspective. Additionally, they uncovered larger differences between the cohort and period perspectives for men than women, which they attribute to differences in disability transition rates between the sexes. We hypothesize that some of these larger differences might also be attributable to larger mortality reduction among men. Further, while cohort HLE estimates are unproblematic as an index, decompositions of HLE differencs between cohorts into morbidity and mortality components are usually biased. This is because the age pattern of morbidity is itself decomposable into morbidity and mortality components, as we demonstrate.

Several studies have looked at the macro relationship between overall mortality levels and sex differences in HLE. At higher levels of life expectancy, female advantage in healthy life expectancy diminishes, or even reverses into male advantage (Van Oyen et al. 2013). Meanwhile, the larger the proportional female advantage in longevity, the larger the female excess in the proportion of life in poor health (Luy and Minagawa 2014). That

mortality levels and disability prevalence are related is perhaps not surprising. As our example illustrates, differences in underlying mortality can lead to differences in the age profile of disability. Additionally, although the association between the severity of chronic conditions and poor health was found to be similar for men and women, morbidity prevalence rates are generally higher among women, particularly for arthritis and chronic pain (Case and Paxson 2005). It would be worthwhile to investigate whether there might not only be differences in the composition of chronic conditions between the sexes, but whether the underlying morbidity process itself might differ between the sexes in its chronological versus time-to-death axis (Riffe, T., Chung, Spijker, and MacInnes Riffe, T. et al.).

In reality, not all end-of-life health conditions are exclusive functions of time-to-death, but morbidity often varies as a function of both aspects of time, and expressing morbidity prevalence as a function of both age and time-to-death, $g(a, y)$, can increase precision (Wolf et al. 2015, Riffe, T., Chung, Spijker, and MacInnes Riffe, T. et al.). There is great variety in the temporal variation of the prevalence of late-life health conditions. There is also great variety in individual trajectories with the approach to death (Lunney et al. 2003). That morbidity prevalence may for certain health conditions be a function of time to death does not imply that morbidity incidence is necessarily a function of time to death. First, an age-patterned sequence of health states wherein mortality risk increases with passing states could produce a time-to-death prevalence pattern, e.g. an accumulation of DNA damage over time resulting in increased ristks of cancer and thereby death. Second, it is also plausible that some morbidity conditions are linked to a more general process of dying, thereby linking morbidity to a process that ends with death and consequently producing a time-to-death prevalence pattern. For example, certain conditions may manifest themselves that are not (primary) causes of the impending death but consequences of nearness to death caused by some other (primary) factor. Either of these explanations does not conflict with the reality that causes must precede effects, and that therefore death cannot cause the morbidity that precedes it (Lynch 2015).

Increasingly, data on mortality incidence and recovery are available from multiple waves of survey panels such as the HRS, allowing researchers to calculate healthy life expectancy using sophisticated multistate models. Unfortunately such data is not available in all countries, or if it is, time trends are limited to the recent past. We are not arguing that a TTD approach should replace multistate models when such data is available. Our aim is rather to expose the implications of comparing healthy life expectancy from age-structured prevalence-based models with different underlying mortality regimes.

To model prevalence as a function of time-to-death requires no surreal understanding of how things work, but is rather a modelling choice (Wolf et al. 2015). When modeling for descriptive or exploratory purposes (as

we have done to produce e.g. Figure 3), and possibly for projective purposes, one can safely use time-to-death as a predictive variable. However, using time-to-death in models intended for causal interpretation is more hazardous; it is argued that time-to-death may function as a proxy for unobserved variables such as biomarkers for impending mortality (Wolf et al. 2015). However, others argue that including this variable as a proxy in models will introduce omitted variable bias (Lynch 2015). Regardless, even in the descriptive case, the reader may understand the time-to-death morbidity prevalence pattern $g(y)$ as generally hypothetical, since the current evidence base is limited in scope. Much more empirical work is needed in order to determine whether modeling morbidity prevalence as a function of time-to-death is more widely applicable to other health conditions, younger ages, more recent birth cohorts, and other populations in different stages of epidemiological transition. Further, the more nuanced function $g(a, y)$ changes over time and it is not fixed as in our examples. Nevertheless, the distortions demonstrated are likely to arise in everyday practice when comparing health trends over age between populations and over time, since many health conditions appear to show strong time-to-death components. Trends in mortality may offset or amplify changes in morbidity. Therefore, in order to separate effects, more careful measurements are required than is typically the case.

6 Appendix

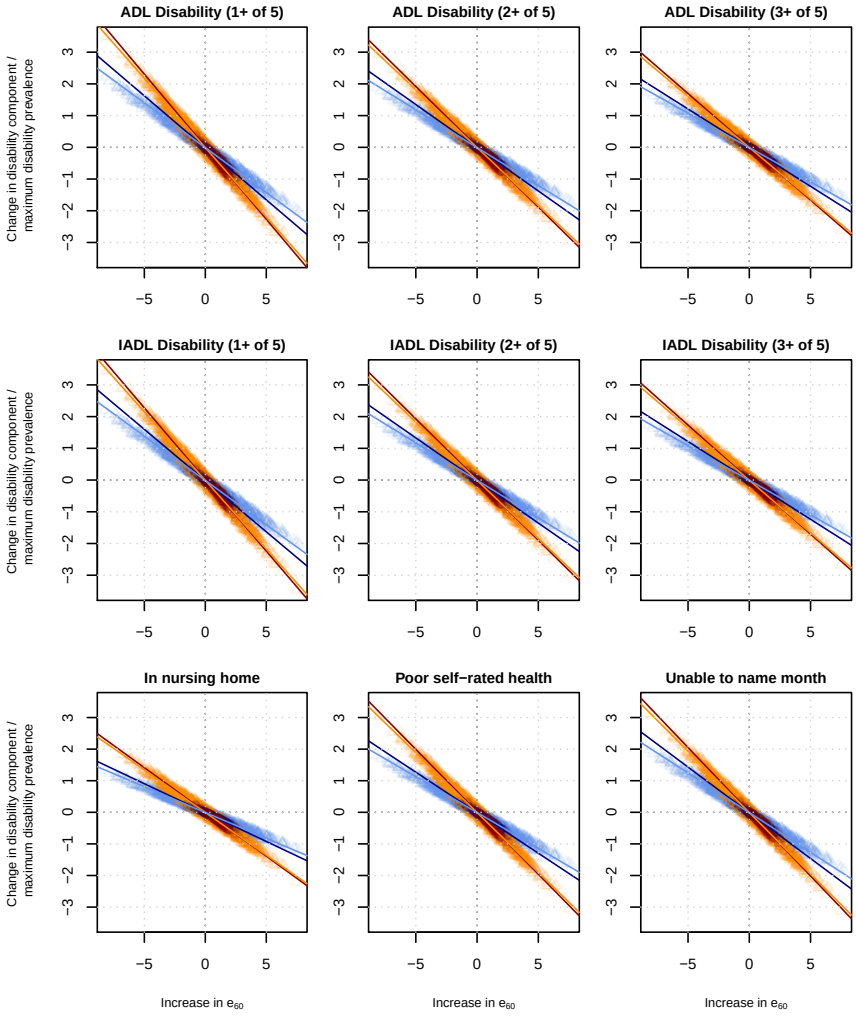


Figure 6: Results of the hypothetical decomposition exercise standardized to the maximum disability prevalence for each disability type. The size of the morbidity component using a standard decompositon method is plotted against the difference in remaining life expectancy at age 60 (e_{60}) in each pair of populations. Linear trend lines are also provided for each sex and decomposition type.

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