IPC 2025 - Long Abstract

Multistate distributions and morbidity compression: Advancing the debate on ageing and health

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1 Introduction

During the last decades, survival prospects for humanity have improved dramatically around the globe. Despite occasional setbacks, the levels of life expectancy have increased in a sustained fashion in most countries since the turn of the 20th century (Oeppen and Vaupel, 2002; Riley, 2005). Although this can be regarded as an important accomplishment, the study of longevity dynamics has generally implicitly assumed that "more" is necessarily "better". But years of life can be spent in "good" or in "less-than-good" health. While the normative desirability of the former is almost universal, it is not clear how desirable the latter is. Given the trade-offs between quantity and quality, and the socioeconomic and personal costs associated with morbidity, assessments of populations' longevity performance should be revisited taking into consideration the fact that non-negligible portions of people's lives can be spent in varying degrees of compromised health. In this context, it is fundamental to explore how years lived in good and in less-than-good health contribute to the composition of individuals' length of life.

Countless studies have investigated the influence of morbidity on average longevity. For example, a lot is known about what fraction of life expectancy (LE) is spent healthy through 'Healthy life expectancy' (HLE) or other conceptually-related indicators (Jagger et al., 2020). In contrast, virtually nothing is known on the number of years individuals accumulate in good and less-than-good health at varying ages at death, or how the time leading up to death is distributed across different health states.

The exploration of these issues has direct bearing with the longstanding 'compression vs expansion of morbidity' debate, which investigates whether morbidity is retreating to older ages at a faster or slower pace than mortality (Fries, 1980; Grunenberg, 1977; Manton, 1982). Traditionally, the standard competing hypotheses of this debate have been tested through the comparison of population health indicators like LE and HLE (details in Section 3.2.1). However, the lack of suitable data and methods has prevented going beyond this approach, failing to take into

account the contribution of healthy and unhealthy years to individuals' lifespans across all possible ages at death.

The aim of this paper is to revisit the aforementioned 'compression vs expansion of morbidity' debate taking advantage of the analytical tools and multistate modelling techniques recently illustrated by Riffe et al. (2024). Such methods allow deriving individual-level multistate distributions estimating the number of years individuals have accumulated in good and in less-than-good health throughout their lives. Building on this approach, we propose the 'healthy year curves', a new tool designed to assess the evolution of morbidity and to identify the age groups primarily driving any observed change. After providing the necessary formal definitions, we present an empirical application of the healthy year curves in the second part of the paper, using Danish registry data and showing results for women and men separately for 2008 and 2018.

2 Background

2.1 Compression vs expansion of morbidity debate

The unprecedented success in delaying the ages at which individuals die have led many scientists to speculate whether improved survival prospects would be accompanied by concomitant morbidity declines. In this regard, three main hypotheses have been proposed. The so-called 'compression of morbidity' hypothesis suggests that, with increases in longevity, the onset of morbidity is gradually compressed towards the last years of life, thus reducing the number of years individuals are expected to live in less-than-good health (Fries, 1980). At the opposite extreme, the 'expansion of morbidity' hypothesis suggests that, in post-epidemiological transition countries, further gains in longevity would be achieved through the survival of people living in morbid states – thus resulting in more disease in the population (Grunenberg, 1977). Between these two extremes, the 'dynamic equilibrium' hypothesis proposes that, with increasing survivorship, severe disability decreases but mild and moderate disability increase (Manton, 1982).

In the following section, we will first give a brief overview of the framework we will operate in, drawing upon newly introduced multistate methods. Next, we will outline the approach typically employed to test the hypotheses discussed above. Finally, we will present the tool we propose to enhance our understanding of morbidity dynamics, contributing to an ongoing debate with significant implications for the sustainability of welfare and healthcare systems.

3 Methods

3.1 Bivariate distributions

In our models, we assume that years of life can be spent either in 'good' or in 'less-than-good' health states. The precise definitions of what it means to be in

one state or the other are context-specific, and in the empirical section of the paper we will show an illustration. With the multistate life table techniques described in Riffe et al. (2024) it is possible to generate bivariate random variables $L = (H, U)$ measuring the cumulated number of years each individual has lived in 'good' health (H) and in 'less-than-good' health (U) at the time of death. The joint density function associated with L will be denoted as $f(h, u)$ (that is, $f(h, u)$ can be interpreted as the relative likelihood that a randomly chosen individual has accumulated h years in good health and u years in less-than-good health at time of death). Assuming that individuals' lifespans are bounded between 0 and ω (the maximal possible age at death), by definition one has that

$$\int_0^{\omega} \int_0^{\omega-h} f\left(h, u\right) dudh = \int_0^{\omega} \int_0^{\omega-u} f\left(h, u\right) dhdu = 1.$$

From this bivariate distribution, it is possible to recover the standard age-at-death distribution, where age at death X is simply defined as $H + U$. For any $x \in [0, \omega]$, let $\mathcal{D}_x := \{(h, u) \in \mathbb{R}^2 | h \geq 0, u \geq 0, h + u = x\}$ be the set of pairs of non-negative values of h and u adding up to x. The elements of $\mathcal{D}_x$ describe all possible combinations of years spent in good and in less-than-good health that add up to x (i.e., $(h = 0, u = x)$, $(h = 1, u = x - 1), \cdots, (h = x, u = 0)$). The density function of X will be denoted as $\varphi(x)$ and is defined as

$$\varphi\left(x\right) = \int_{\mathcal{D}_x} f = \int_0^x f\left(h, x - h\right) dh = \int_0^x f\left(x - u, u\right) du \tag{1}$$

for any given age at death $x \in [0, \omega]$. By construction,

$$\int_0^{\omega} \varphi\left(x\right) dx = 1$$

and therefore

$$\int_0^{\omega} \int_0^x f\left(h, x - h\right) dhdx = \int_0^{\omega} \int_0^x f\left(x - u, u\right) dudx = 1$$

Thus, the (H, U) distribution can be seen as a generalisation of the traditional age-at-death distribution that allows estimating the cumulated number of years individuals have spent in different health states at the end of their lives – rather than merely accounting for their overall length. Figure 1 shows the shape of a hypothetical joint density function $f\left(h, u\right)$ associated to $L = (H, U)$. Being L a two-dimensional random variable, the plot of $f\left(h, u\right)$ is a 2-dimensional surface embedded in a 3-dimensional space and the values of the density function of X, $\varphi\left(x\right)$, are estimated by integrating $f\left(h, u\right)$ along the $\mathcal{D}_x$ diagonals defined above. In this setting, 'Healthy life expectancy' becomes

$$\int_0^{\omega} \int_0^{\omega-h} hf\left(h, u\right) dudh = HLE = E[H], \tag{2}$$

the average number of years individuals have spent in good health throughout their lifetimes. Likewise, we can define 'Unhealthy life expectancy' (ULE) as

$$\int_0^{\omega} \int_0^{\omega-h} uf\left(h, u\right) dudh = ULE = E[U], \tag{3}$$

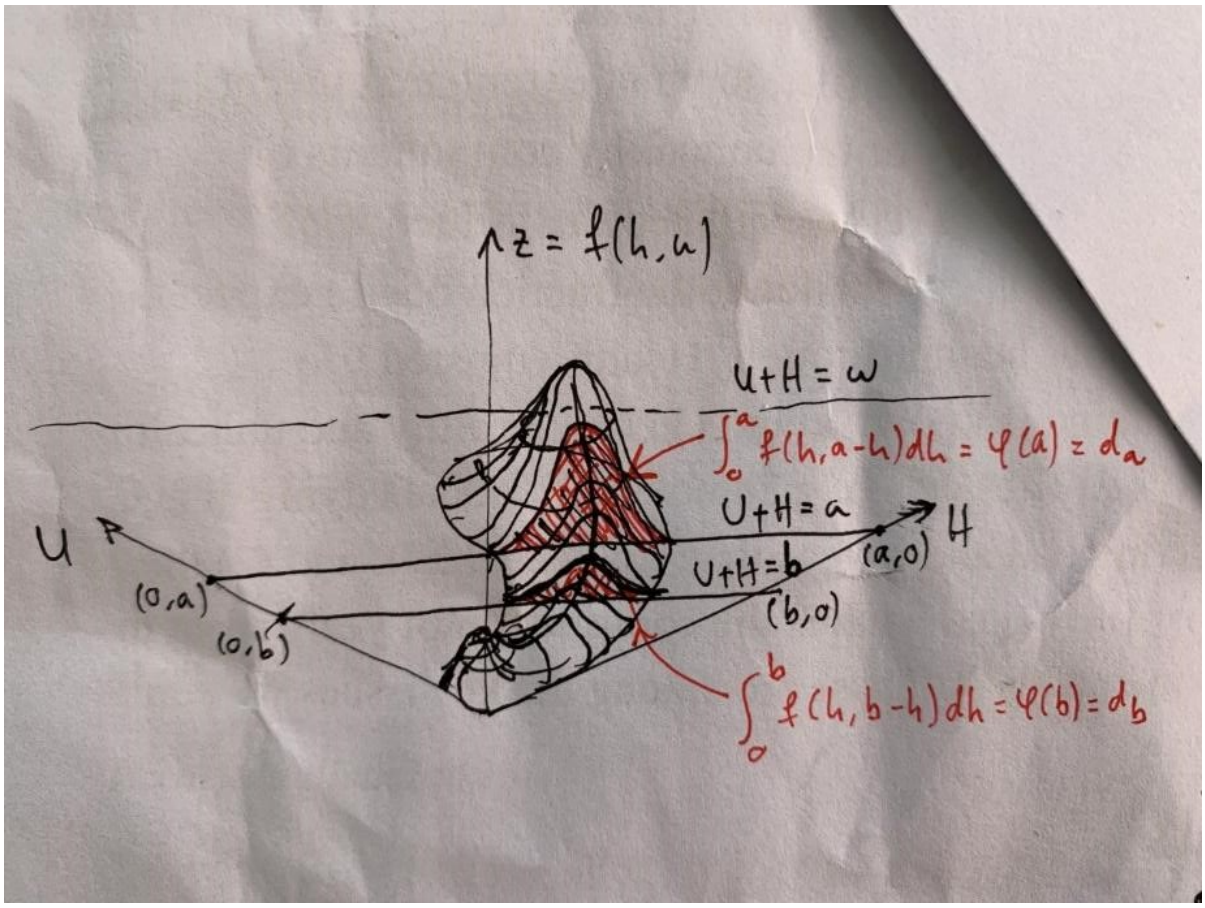

Figure 1. Illustration of a hypothetical joint density function $f(h,u)$, together with a representation of a couple of values of the age at death distribution $\varphi(x)$.

the average number of years individuals have lived in less-than-good health in their lifetimes. Putting together (2) and (3),

$$\begin{aligned} HLE + ULE &= \int_0^{\omega} \int_0^{\omega-h} hf\,(h,u)\,dudh + \int_0^{\omega} \int_0^{\omega-h} uf\,(h,u)\,dudh \\ &= \int_0^{\omega} \int_0^{\omega-h} (h+u) f\,(h,u)\,dudh \\ &= \int_0^{\omega} \int_h^{\omega} xf\,(h,x-h)\,dxdh = LE \end{aligned} \tag{4}$$

which corresponds to the traditional life expectancy.

More compactly, in random variable notation

$$HLE + ULE = E[H] + E[U] = E[H+U] = E[X] = LE \tag{5}$$

because of the linearity of the expected value.

3.2 Revisiting the compression vs expansion of morbidity debate

In this subsection, we first show the approach that has been traditionally used to test competing hypotheses in the compression vs expansion of morbidity debate, and then proceed to present the novel approach we propose here based on the analytical setting introduced in the previous subsection.

3.2.1 The classical approach

Since their inception, 'compression' or 'expansion of morbidity' hypotheses have been typically tested by comparing the values of LE vis-à-vis those of HLE (Robine et al., 2020). Usually, increases (resp. decreases) in the ratio HLE/LE over time lend support to the compression (resp. expansion) of morbidity hypothesis. Figure 2 illustrates how this classical approach translates in the analytical setting we adopt in this paper. We assume that (1) at a given point in time, say t_1, the values of HLE and ULE equal H_1 and U_1, respectively; and (2) the values of LE are expected to increase over time. Under those assumptions, Figure 2 shows the combinations of HLE and ULE that must be observed in time $t_2 > t_1$ for the 'expansion' or 'compression of morbidity' to occur. The line separating the opposite conclusions of 'expansion' vs 'compression' is the one satisfying the restriction $\frac{HLE}{ULE} = \frac{H_1}{U_1}$. As an illustration, consider the values (at time t_1) of $H_1 = 60$ and $U_1 = 10$, so $LE_1 = 70$. If at time t_2 one has that $LE_2 = 80$, $H_2 = 69$ and $U_2 = 11$, then the classical approach would conclude that a compression of morbidity has occurred, because the fraction HLE/LE has increased from 0.857 to 0.862. Alternatively, if at time t_2 one had that $\tilde{LE}_2 = 80$, $\tilde{H}_2 = 65$ and $\tilde{U}_2 = 15$, then it would conclude that an expansion of morbidity has occurred, because the fraction HLE/LE declines to 0.812.

In the classical approach, the only piece of information that is needed to reach a conclusion is the change over time in the relative size of the population-based indicators HLE and LE. This ignores the shape of the (H, U) distribution (i.e., whether individuals dying at different ages spend more or less years in good or in less-than-good health), an issue we take up in the following subsection.

3.2.2 Healthy year curves

The method we are going to illustrate takes advantage of the fact that, having information about the joint distribution of H and U, we can estimate the number of years individuals have accumulated in good health at all possible ages at death x – thus offering a richer and more nuanced picture that goes beyond exclusively relying on the average-based indicators LE and HLE. In order to proceed, we need to introduce some formal definitions.

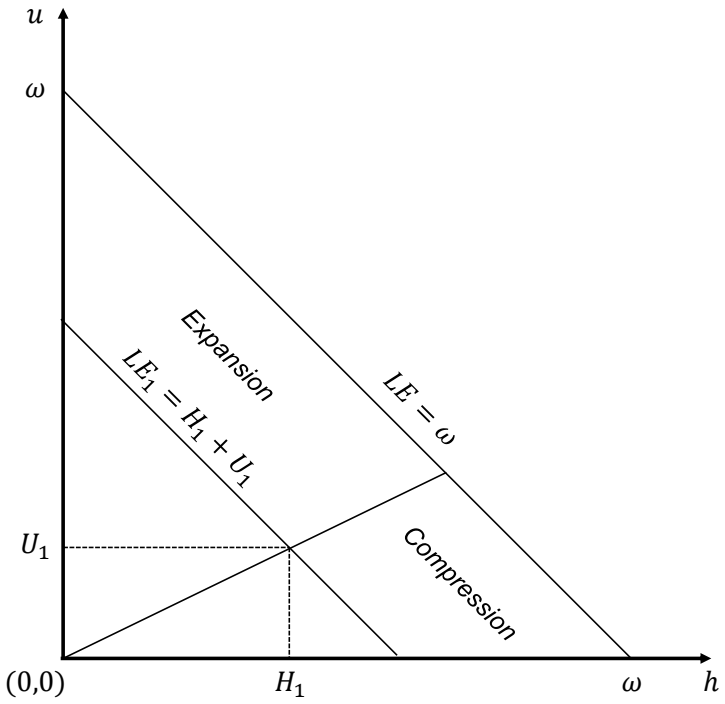

Figure 2. Traditional approach to test compression vs expansion of morbidity. Source: Authors' own elaboration.

Definition 1. For each age at death $x \in (0, \omega]$, let

$$\Psi_H(x) \coloneqq \int_0^x h \left[\frac{f(h, x-h)}{\int_0^x f(a, x-a)da} \right] dh = E[H|X = x] \tag{6}$$

$$\Psi_U(x) \coloneqq \int_0^x u \left[\frac{f(x-u, u)}{\int_0^x f(x-a, a)da} \right] du = E[U|X = x] \tag{7}$$

The first equation in Definition 1 is simply an average of the values of h for all individuals who died at age x. Thus, $\Psi_H(x)$ measures the average number of years lived in good health among those who died at age x. Likewise, $\Psi_U(x)$ measures the average number of years lived in less-than-good health among those who died at age x. The curve $\Psi_H(x)$ (resp. $\Psi_U(x)$) will be referred to as 'healthy years curve' (resp. 'unhealthy years curve'). Similarly, it is straightforward to define the relative version of the healthy and unhealthy year curves (i.e., the functions that, for each age at death x measure the proportion of years lived in good (resp. less-than-good) health among those who died at age x (see Appendix). It is easy to check (see Appendix) that, for any age at death $x \in (0, \omega]$,

$$0 \leq \Psi_H(x) \leq x$$
$$0 \leq \Psi_U(x) \leq x$$

And

$$\Psi_H(x) + \Psi_U(x) = x \tag{8}$$

That is: among those who die at age x, the average number of years lived in good health and the average number of years lived in less-than-good health add up to

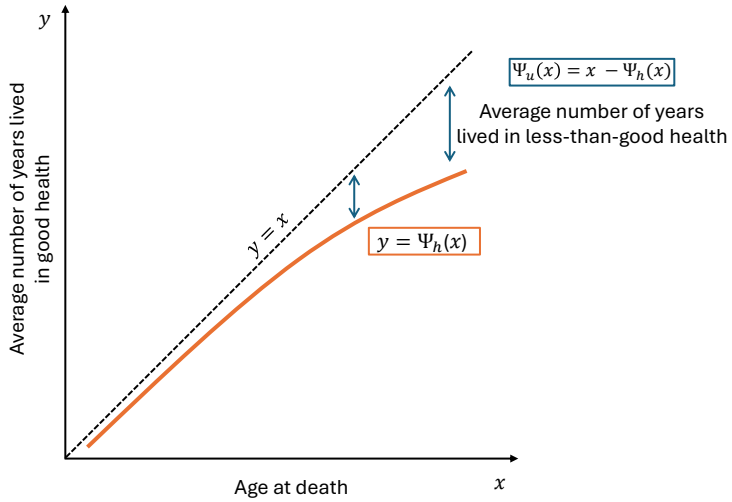

Figure 3. Illustration of the healthy $\Psi_H(x)$ and unhealthy $\Psi_U(x)$ year curves. Source: Authors' own elaboration.

x. In Figure 3, we show hypothetical examples of how these $\Psi_H(x)$ and $\Psi_U(x)$ curves could look like.
Importantly, the healthy and unhealthy year curves satisfy the following identities (proofs shown in Appendix)

$$\int_0^\omega \Psi_H(x)\,\varphi(x)\,dx = HLE \tag{9}$$

$$\int_0^\omega \Psi_U(x)\,\varphi(x)\,dx = ULE \tag{10}$$

That is: weighting the mean years lived in good (resp. less-than-good) health among those who die at age x by the share of deaths occurring at that age gives the expected average number of years lived in good (resp. less-than-good) health for the entire population. These identities show how, in our setting, HLE and ULE can be derived after averaging simpler age-at-death-specific estimates of the number of years individuals spend in good and less-than-good health, respectively.

Having introduced the healthy year curves, we can now present our new criteria to test alternative hypotheses in the compression vs expansion of morbidity debate.

Definition 2. Let $\Psi_H(x)$ and $\tilde{\Psi}_H(x)$ be the healthy year curves for the population under study at two different points in time t_1 and t_2, with $t_1 < t_2$.

Case (i). Whenever $\Psi_H(x) \geq \tilde{\Psi}_H(x)$ for each age at death $x \in (0, \omega]$, we say that there has been an expansion of morbidity between t_1 and t_2.

Case (ii). Whenever $\Psi_H(x) \leq \tilde{\Psi}_H(x)$ for each age at death $x \in (0, \omega]$, we say that there has been a compression of morbidity between t_1 and t_2.

Case (iii). Whenever $\Psi_H(x) < \tilde{\Psi}_H(x)$ for some values of $x \in (0, \omega]$ but $\Psi_H(x) > \tilde{\Psi}_H(x)$ for some other values of $x \in (0, \omega]$, we cannot clearly say whether morbidity has expanded or compressed between t_1 and t_2.

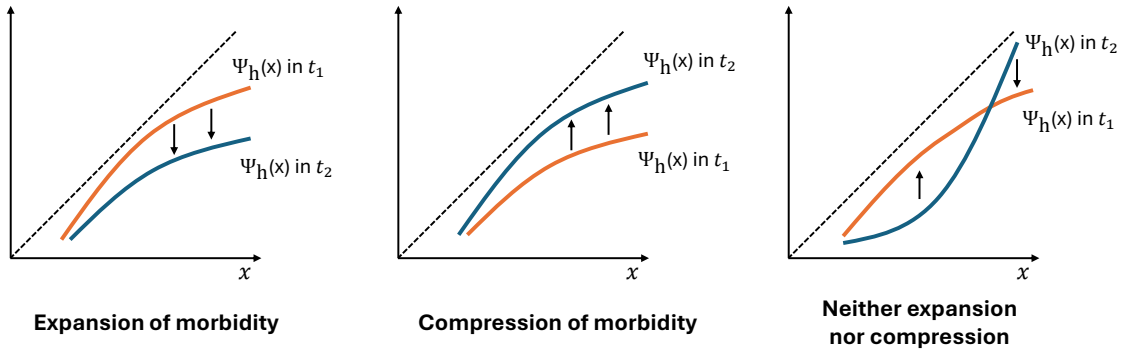

Figure 4. Testing the compression vs expansion of morbidity debate using the healthy year curves $\Psi_H(x)$ in two time points $t_1 < t_2$. Source: Authors' own elaboration.

In Case (i), the average number of years individuals have spent in good health at time at death has decreased across all possible ages at death. When this happens, it seems reasonable to conclude that morbidity has expanded between the two time periods. Likewise, if the average number of years individuals have spent in good health at time at death has increased across all possible ages at death (Case (ii)), it seems reasonable to conclude that morbidity has compressed between t_1 and t_2. Instead, whenever the average number of years spent in good health has increased for some ages at death but decreased for others, it is not obviously clear whether morbidity has expanded or compressed overall, so no conclusion is reached (Case (iii)). These different scenarios are graphically illustrated in Figure 4.

How do the classical and the new approach proposed here differ from each other? While the classical one compares what fraction of life expectancy is spent in good health at the population level, the new one performs a similar exercise but across all possible ages at death (i.e., it inspects what fraction of life has been spent in good health among all those who die at a given age x, across all possible ages at death). The finer detail we are working with in the new approach comes at a cost: since we are imposing unanimity in the comparisons across all possible ages at death, there might be instances where a firm conclusion cannot be reached (as in Case (iii), when the corresponding healthy year curves cross). However, such lack of conclusiveness should not be necessarily seen as a limitation. Using the healthy year curves, we can identify the specific age ranges that have benefited the most (or the least) from health changes over time. We will show an example in the empirical application below.

4 Data

The data we have access to and that will be used in the complete version of the paper is extracted from the mortality and health records for the entire population of Denmark (roughly 5.8 Million inhabitants) from 2008 to 2019. Such records capture the ages at which individuals residing in the country die or are diagnosed from one of the following major chronic diseases: diabetes; myocardial infarction; angina pectoris; other diseases of the heart; stroke; chronic bronchitis/chronic

obstructive pulmonary disease/emphysema; cirrhosis of the liver; malignant tumor; parkinsonism; Alzheimer's disease and chronic renal failure. Linking mortality and health information, we are able to compute age-at-first-diagnosis and age-at-death for all individuals dying at a given year, from which we infer the corresponding values of x, h and u.

5 Preliminary Results

5.1 Standard/Classical indicators

Table 1 reports the values of LE, HLE and ULE for the Danish population aged above 50. Results are shown for women and men separately. The three indicators increase between 2008 and 2018 for both sexes. As expected, LE is higher among women for all years, but the gap with respect to men slightly decreases (from 3.6 years in 2008 to 3.3 years in 2018). While LE, HLE, and ULE tend to increase over time, the rates at which these indicators grow are not necessarily the same, leading to changes in the HLE/LE ratio. According to the classical criterion for testing the compression vs. expansion of morbidity hypotheses presented in Section 3.2.1, morbidity is expanding for men, as the proportion of life expectancy spent in good health has declined. For women, however, the increases in LE and HLE have remained more closely aligned, resulting in a relatively stable HLE/LE ratio.

Sex	Year	LE	HLE	ULE	HLE/LE
F	2008	31.6	24.6	7.0	0.779
F	2018	34.7	26.9	7.8	0.777
M	2008	28.0	21.3	6.3	0.759
M	2018	31.4	23.5	7.9	0.748

Table 1. Values of life expectancy (LE), healthy life expectancy (HLE) and unhealthy life expectancy (ULE) for Danish females (F) and males (M) at age 50 in 2008 and 2018 using the presence of at least one chronic condition to define less-than-good health.

5.2 Results for bivariate distributions

In Figure 5, Panel A shows the age-at-death distributions for Danish females and males who died without any chronic conditions ($u = 0$) in 2008 and 2018. For both sexes, we observe an increase in the number of healthy deaths occurring at older ages, suggesting improvements in morbidity-free survival over the decade.
Panel B presents the joint density functions $f(h, u > 0)$ for individuals who died after living at least one year with a chronic condition. We observe a rightward shift, representing increased unhealthy longevity, along with a significant reduction in the number of deaths occurring between ages 70 and 80 among those who lived more than 15 years with a chronic condition at the time of death. The lightest colours are concentrated on the horizontal axis: most individuals die after spending less than 5 years with a chronic condition.

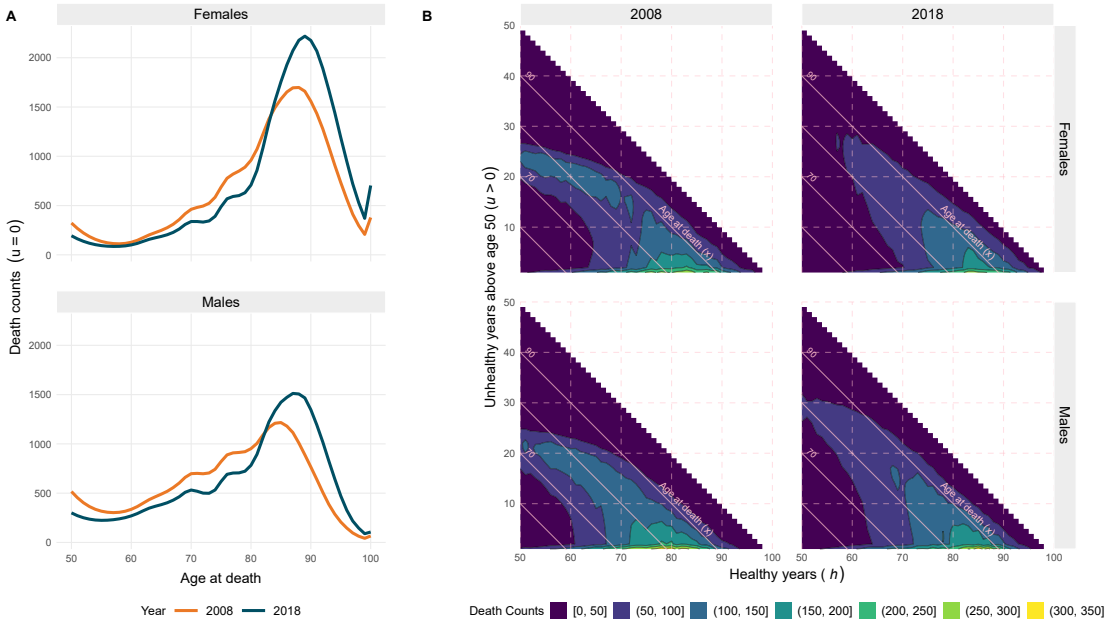

Figure 5. Graphical representation of the joint density function $f(h, u)$ for Danish women and men aged 50+ in 2008 and 2018 using the diagnosis of at least a chronic condition as a measure of less-than-good health. In Panel A, the age-at-death distributions of those dying healthy ($u = 0$); in Panel B, the age-at-death distributions of those dying unhealthy ($u > 0$).

The expected (i.e., mean) values of these (H, U) distributions were shown in the LE, HLE and ULE columns of Table 1.

The healthy year curves $\Psi_H(x)$ associated with the 2008 (orange) and 2018 (blue) (H, U) distributions are shown in Figure 6 (women on the left, men on the right). A clear cross-over point appears around age 80 for both sexes (81 for females, 78 for males), placing this case in the third scenario described in Figure 4. For individuals dying before age 65, the number of healthy years lived appears unchanged between the two time periods, as the curves closely overlap. However, between ages 65 and the cross-over point, the 2018 curve lies above the 2008 curve for both women and men, revealing that those dying in this age range in 2018 had accumulated on average more years in good health than those who died at the same ages in 2008. The trend reverses at older ages, where the 2018 curve stays consistently below the 2008 curve, indicating fewer healthy years, on average, among those dying at advanced ages in 2018 compared to 2008.

When compared to the results based solely on the ratio, it is clear that the curves provide a richer and more informative picture, revealing dynamics and differences that a single summary statistic cannot capture. This is particularly true for women, for which the ratio does not change between the two periods.

[Not sure it makes sense] To try to understand which theory is ultimately unfolding, Table 2 presents the share of total deaths occurring before and after the age at which the curves cross, for both sexes and years. The percentage of individuals

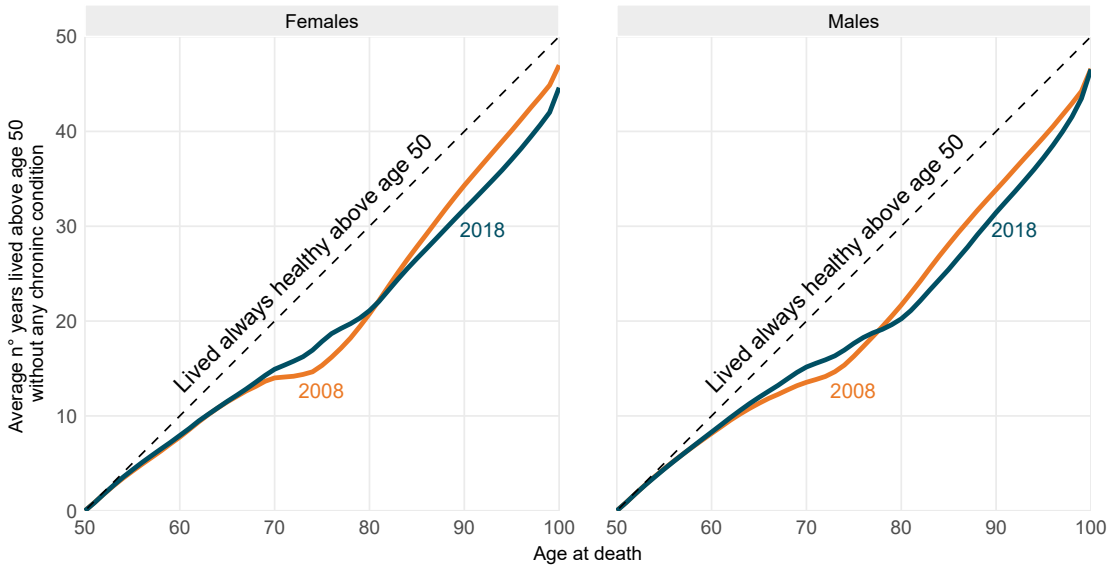

Figure 6. Healthy year curves $\Psi_H(x)$ for Danish females and males aged 50+ in 2008 and 2018 using the presence of at least one chronic condition to define less-than-good health.

dying above the cross-over age increases from 2008 to 2018 for both men and women. This suggests that more people are reaching older ages in 2018, and, as the curves indicate, they are more likely to accumulate additional years lived with a chronic condition before death. This aligns with the expansion of morbidity theory.

Sex	Year	CoA	% deaths before CoA	% deaths after CoA
F	2008	81	41%	59%
F	2018		26%	74%
M	2008	78	43%	57%
M	2018		28%	72%

Table 2. Share of total deaths happening before and after the cross-over age (CoA) for Danish females (F) and males (M) in 2008 and 2018.

6 Future Outlook

In this paper we have presented a more refined approach to assess whether morbidity is compressing or expanding over time. The traditional technique based on the evolution of the HLE/LE ratio suggests that morbidity has not changed for Danish women between 2008 and 2018, while it has expanded among the Danish males. The method proposed here relying on the relative position of the healthy years curves gives a more nuanced picture, pointing towards an expansion of morbidity for both sexes, while also showing for which ages at death the average number of years accumulated without a chronic condition has increased or decreased.

These results cohere with recent studies finding that, as survival prospects further improve in low-mortality countries, the health profiles of the elder become an increasingly heterogeneous mix of robust and frail individuals (Engelman et al., 2010), with an increasing prominence of the years that are lived in morbid states.

We acknowledge that the traditional dichotomy of "healthy" "unhealthy" tends to oversimplify the complexity of health, often overlooking individual circumstances and the interplay of multiple contributing factors. Nonetheless, simple models can still offer valuable insights. In this paper, we chose to adopt a broad list of chronic diseases to classify individuals as "unhealthy". In the future, we plan to produce the curves using a more restricted and specific set of conditions –such as cardiovascular diseases– and to possibly refer to them in a more neutral way, using terms like "condition-free" and "with condition" curves.

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Appendix

Proof of Eq. (8)

We prove that

$$\Psi_H(x) + \Psi_U(x) = x.$$

By definition

$$\Psi_H(x) + \Psi_U(x) = \frac{1}{\varphi(x)}\left[\int_0^x hf(h, x-h)dh + \int_0^x uf(x-u, u)du\right]$$

Substituting $u = x - h$, the second integral can be rewritten as

$$\begin{aligned}\int_0^x uf(x-u, u)du &= \int_x^0 (x-h)f(h, x-h)(-dh) \\ &= \int_0^x (x-h)f(h, x-h)dh\end{aligned}$$

Therefore

$$\begin{aligned}\Psi_H(x) + \Psi_U(x) &= \frac{1}{\varphi(x)}\left[\int_0^x hf(h, x-h)dh + \int_0^x (x-h)f(h, x-h)dh\right] \\ &= \frac{1}{\varphi(x)}\int_0^x xf(h, x-h)dh \\ &= \frac{x}{\varphi(x)}\int_0^x f(h, x-h)dh \\ &= \frac{x}{\varphi(x)}\varphi(x) \\ &= x\end{aligned}$$

More compactly, in random variable notation:

$$\begin{aligned}\Psi_H(x) + \Psi_U(x) &= E[H|X = x] + E[U|X = x] \\ &= E[H + U|X = x] \\ &= E[X|X = x] \\ &= x\end{aligned}$$

Proof of Eq.(9)

We prove that

$$\int_0^\omega \Psi_H(x)\,\varphi(x)\,dx = HLE.$$

Replacing $\Psi_H(x)$ with its definition in (6) and then simplifying by $\varphi(x)$ the integral becomes

$$\int_0^\omega \Psi_H(x)\,\varphi(x)\,dx = \int_0^\omega \left[\frac{1}{\varphi(x)}\int_0^x hf(h,x-h)dh\right]\varphi(x)dx$$
$$= \int_0^\omega \int_0^x hf(h,x-h)dhdx$$

Inverting the order of integration we obtain

$$\int_0^\omega \int_0^x hf(h,x-h)dhdx = \int_0^\omega \int_h^\omega hf(h,x-h)dxdh$$

Finally, substituting $x-h=u$ we get

$$\int_0^\omega \int_h^\omega hf(h,x-h)dxdh = \int_0^\omega \int_0^{\omega-h} hf(h,u)dudh$$

which is equal to HLE as defined in Eq. (2).

The proof of Eq. (10) is equivalent.

Relative version of $\Psi_H(x)$, $\Psi_U(x)$

Definition A1. For each age at death $x \in (0,\omega]$, let

$$P_h(x) \coloneqq \frac{1}{x}\int_0^x h\left[\frac{f(h,x-h)}{\int_0^x f(a,x-a)da}\right]dh = \frac{\Psi_H(x)}{x}$$
$$P_u(x) \coloneqq \frac{1}{x}\int_0^x u\left[\frac{f(x-u,u)}{\int_0^x f(x-a,a)da}\right]du = \frac{\Psi_U(x)}{x}$$

The functions introduced in Definition A1 simply are the relative version of the 'healthy' and 'unhealthy year curves'. Thus, $P_h(x)$ (resp. $P_u(x)$) measures the proportion of years lived in good (resp. less-than-good) health among those who died at age x. Thus, it is easy to check that, for all possible ages at death $x \in (0,\omega]$, the following identity holds

$$P_h(x) + P_u(x) = 1$$

The relative versions of the healthy and unhealthy year curves yield the following identities

$$\int_0^\omega P_h(x)\,\varphi(x)\,dx = \int_0^\omega \int_0^x \left(\frac{h}{h+u}\right) f(h,x-h)dhdx = \int_0^\omega \int_0^x \frac{h}{x} f(h,x-h)dhdx$$
$$\int_0^\omega P_u(x)\,\varphi(x)\,dx = \int_0^\omega \int_0^x \left(\frac{u}{h+u}\right) f(h,x-h)dhdx = \int_0^\omega \int_0^x \frac{u}{x} f(h,x-h)dhdx$$

The first (resp. second) equation is an average of the fractions of life spent in good (resp. less-than-good) health across all the individuals in the population. Interestingly, this quantity does not necessarily coincide with the value of HLE/LE (and the same happens with the second equation and ULE/LE). Stated otherwise: the average of fractions of life spent in good health across individuals is typically different from the fraction of averages HLE/LE[1].

[1]The reason why these two quantities do not necessarily coincide is because arithmetic

averages are additive, while fractions are multiplicative – so to speak. For instance, if $x_1, \ldots, x_n, y_1, \ldots, y_n$ are real non-negative numbers, then $(1/n)\sum_i (x_i/y_i)$ generally differs from $((1/n)\sum_i x_i)/((1/n)\sum_i y_i)$.