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Drewnowski's index to measure lifespan variation: Revisiting the Gini coefficient of the life table

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ABSTRACT

The Gini coefficient of the life table is a concentration index that provides information on lifespan variation. Originally proposed by economists to measure income and wealth inequalities, it has been widely used in population studies to investigate variation in ages at death. We focus on the complement of the Gini coefficient, Drewnowski's index, which is a measure of equality. We study its mathematical properties and analyze how changes over time relate to changes in life expectancy. Further, we identify the threshold age below which mortality improvements are translated into decreasing lifespan variation and above which these improvements translate into increasing lifespan inequality. We illustrate our theoretical findings simulating scenarios of mortality improvement in the Gompertz model, and showing an example of application to Swedish life table data. Our experiments demonstrate how Drewnowski's index can serve as an indicator of the shape of mortality patterns. These properties, along with our analytical findings, support studying lifespan variation alongside life expectancy trends in multiple species.

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1. Introduction

The life table is an essential tool in mortality studies. It represents the current mortality experience of a population and it is usually summarized by life expectancy at birth (e_0). Life expectancy at birth is the average years a synthetic cohort of newborns is expected to live if individuals were to experience the current mortality conditions throughout their lives. It is, however, an average indicator that masks variability in ages at death, which can be substantial. This variability is often referred to as lifespan variation or lifespan inequality, and has received increasing attention over the past two decades (see, to mention a few, Wilmoth and Horiuchi, 1999; Edwards and Tuljapurkar, 2005; Smits and Monden, 2009; Baudisch, 2011; Vaupel et al., 2011; Fernández and Beltrán-Sánchez, 2015; Colchero et al., 2016; Ebeling et al., 2018; van Raalte et al., 2018; Permanyer and Scholl, 2019; Aburto et al., 2020; Vaupel et al., 2021). Lifespan variation reveals the

uncertainty about the eventual age at death at the individual level, and measures how evenly mortality conditions are shared at the population level. There exist several indicators to measure lifespan variation (for an overview, see Shkolnikov et al., 2003; van Raalte and Caswell, 2013), such as the entropy of the life table (Leser, 1955; Keyfitz, 1977; Demetrius, 1978), the standard deviation or variance of the age-at-death distribution (as applied by Tuljapurkar and Edwards, 2011), the coefficient of variation (as applied by Aburto and van Raalte, 2018; Aburto et al., 2018), years of life lost (e^+) (Goldman and Lord, 1986; Vaupel, 1986; Hakkert, 1987; Vaupel and Canudas Romo, 2003), or the Gini coefficient (Hanada, 1983).

Here we study in greater detail the Gini coefficient of the life table (G) and its complement, Drewnowski's index (D) (Drewnowski, 1982; Hanada, 1983), from a formal demographic perspective. While the properties of these two indicators in the demographic sense are analogous, D directly provides an added meaningful interpretation: it reflects the proportion of life expectancy that two newborns of a cohort are expected to live together when the life table assumptions hold. We additionally aim to understand how changes in age-specific mortality underpin trends in lifespan variation. We focus on how changes

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over time in D relate to changes in e_o , and highlight a new measure of absolute variation related to perturbation theory, named ϑ . This new indicator gives the value of life expectancy if the hazard is doubled at every age. We provide the mathematical foundation of how Drewnowski's index (and analogously the Gini coefficient) evolves over time, and give analytical formulae to find the threshold age below which mortality improvements are translated into decreasing lifespan variation and above which these improvements translate into increasing lifespan inequality.

2. The Gini coefficient and Drewnowski's index

The Gini coefficient is one of the most popular indices employed in social sciences to measure concentration in the distribution of a non-negative random variable (Gini, 1912, 1914). Originally proposed by economists to measure income or wealth inequality, this coefficient has been applied in demography and survival analysis to investigate within-group inequality in terms of ages at death (see, for instance, Hanada, 1983; Shkolnikov et al., 2003; Bonetti et al., 2009; Gigliarano et al., 2017; Barthold Jones et al., 2018; Diaz et al., 2018; Basellini and Camarda, 2019; Aburto et al., 2020).

2.1. Definition

As thoroughly discussed by Yitzhaki and Schechtman (2013), there are several equivalent ways to define the Gini coefficient. Let X be a non-negative random variable with probability density function $f(x)$ and expected value $E[X]$, a common definition is

$$G = \frac{1}{2E[X]} \int_0^\infty \int_0^\infty |x_1 - x_2| f(x_1) f(x_2) dx_1 dx_2 .$$

Accordingly, if X is a random variable of the ages at death in a population, the Gini coefficient expresses the average of absolute differences in individual lifespans relative to the mean length of life $E[X]$.

Michetti and Dall'Aglio (1957), and later Hanada (1983), suggest a re-formulation of the Gini coefficient in terms of the life table functions, given by

$$G = 1 - \frac{\int_0^\infty \ell(x, t)^2 dx}{\int_0^\infty \ell(x, t) dx} = 1 - \frac{\vartheta}{e_o} , \tag{1}$$

where $\ell(x, t)$ is the life table survival function at time t , $e_o = \int_0^\infty \ell(x, t) dx$ the life expectancy at birth at time t , and $\vartheta = \int_0^\infty \ell(x, t)^2 dx$ is the resulting life expectancy at birth of doubling the hazard at all ages. Barthold Jones et al. (2018) interpret ϑ as a measure of *shared life expectancy*, that is, the average time two newborns at time t are expected to survive together. For the purposes of this article, the definition of the Gini coefficient in (1) will be used in the following.

2.2. Main properties

The Gini coefficient takes values between 0 and 1, and can be interpreted as a *measure of inequality*. A value of 0 denotes equality in ages at death, i.e. when every individual in the population has the exact same length of life. The index increases approaching 1 as lifespans become more spread and unequal in the population. This makes the interpretation clear and intuitive: higher values correspond to greater within-group inequality in ages at death.

An additional attractive feature of the Gini coefficient is that it fulfills three important properties for inequality indices (Sen, 1973; Anand, 1983): (i) it does not change if the number of individuals at each age at death is changed by the same proportion (*population-size independence*); (ii) it does not change if

each individual lifespan is changed by the same proportion (*scale independence*); and (iii) it decreases if years of life are transferred from a longer to a shorter lived individual (*Pigou-Dalton condition*). Note that property (i) enables straightforward comparison between populations, including comparisons between different species (Wrycza et al., 2015). Furthermore, the coefficient is not too sensitive to redistributions at early ages of life, and it well reflects changes at adult ages (Shkolnikov et al., 2003).

Being bounded between 0 and 1, the Gini coefficient can be readily transformed from a *measure of inequality* into a *measure of equality* of lifespans. From (1), its complement indicator of lifespan equality immediately derives, the Drewnowski index, defined as

$$D = 1 - G = \frac{\vartheta}{e_o} = \frac{\int_0^\infty \ell(x, t)^2 dx}{\int_0^\infty \ell(x, t) dx} , \tag{2}$$

and sharing all the important properties of G . In the case of the Pigou-Dalton condition, though, the effect is the opposite and D increases as years of life are transferred from a longer to a shorter lived individual without changing their relative ranks. According to Hanada (1983), this index was first proposed by Jan Drewnowski on a working group on health indicators at the World Health Organization in the early 1980s (Drewnowski, 1982).

In addition to its interpretation as the proportion of life expectancy that will be shared by two newborns (if mortality rates do not change over their lives), D is akin to already established demographic measures. Specifically, D is similar both in its construction and age pattern to e^+ (Vaupel and Canudas Romo, 2003), to the life table entropy (Leser, 1955; Keyfitz, 1977; Demetrius, 1978), and to the variance or standard deviation (Edwards and Tuljapurkar, 2005). Moreover, following (2), and similarly to other indicators, D has the advantage that it can be interpreted as the weighted average of the survival function (with weights equal to the survival function itself).

3. Changes over time in Drewnowski's index

In order to analyze changes over time in Drewnowski's index – or, equivalently, the Gini coefficient – we aim to find an analytical expression for the time derivative of D . In the following, a dot over a function will denote the partial derivative with respect to time, but variable t will be omitted for simplicity.

3.1. Relative derivative of D

Proposition 1. Let $D = \vartheta / e_o$ be Drewnowski's index, where $\vartheta = \int_0^\infty \ell(x)^2 dx$, $e_o = \int_0^\infty \ell(x) dx$ is the life expectancy at birth, and $\ell(x)$ the probability of surviving from birth to age x . Then, relative changes over time in D are given by

$$\frac{\dot{D}}{D} = \frac{\dot{\vartheta}}{\vartheta} - \frac{\dot{e}_o}{e_o} . \tag{3}$$

Proof. Note that $D = \vartheta / e_o$ implies that $D e_o - \vartheta = 0$. Differentiating with respect to time yields

$$\dot{D} e_o + D \dot{e}_o - \dot{\vartheta} = 0 .$$

Solving for \dot{D} and dividing both sides by D , we get (3). \square

Eq. (3) decomposes relative changes in D into relative changes of the shared life expectancy between two individuals ϑ , and relative changes in the life expectancy at birth e_o .

3.2. Time derivatives of e_0 and ϑ

Vaupel and Canudas Romo (2003) showed that changes over time in life expectancy at birth are a weighted average of the total rates of mortality improvement, expressed as

$$\dot{e}_0 = \int_0^\infty \rho(x) w(x) dx \tag{4}$$

Function $\rho(x) = -\dot{\mu}(x) / \mu(x)$ stands for the age-specific rates of mortality improvement, where $\mu(x)$ is the force of mortality (hazard rate) at age x . The weights $w(x) = \mu(x) \ell(x) e(x)$ are a measure of the importance of death at age x , where $e(x) = \int_x^\infty \ell(a) da / \ell(x)$ is the remaining life expectancy at age x . Following a similar approach, we aim to express the time derivative of ϑ as a weighted average of mortality improvements, but with different weights.

Definition 1. Let $\ell(x)$ be the probability of surviving from birth to age x . A measure of lifespan equality at age x is Drewnowski's index conditional upon survival up to age x , defined as

$$D(x) = \frac{1}{\ell(x)} \frac{\int_x^\infty \ell(a)^2 da}{\int_x^\infty \ell(a) da} \tag{5}$$

Proposition 2. Let $\vartheta = \int_0^\infty \ell(x)^2 dx$, where $\ell(x)$ is the probability of surviving from birth to age x . Then, its partial derivative with respect to time can be expressed as

$$\dot{\vartheta} = \int_0^\infty \rho(x) w(x) 2 \ell(x) D(x) dx, \tag{6}$$

where $\rho(x)$ are the age-specific rates of mortality improvement, $w(x)$ the same weights defined in (4), and $D(x)$ as defined in (5).

Proof. Applying the chain rule, the time derivative of ϑ is simply

$$\dot{\vartheta} = \int_0^\infty 2 \ell(x) \dot{\ell}(x) dx.$$

Using that $\dot{\ell}(x) = -\ell(x) \int_0^x \dot{\mu}(a) da$, and reversing the order of integration, we get

$$\begin{aligned} \dot{\vartheta} &= -2 \int_0^\infty \ell(x)^2 \int_0^x \dot{\mu}(a) da dx = -2 \int_0^\infty \dot{\mu}(a) \int_a^\infty \ell(x)^2 dx da \\ &= 2 \int_0^\infty \rho(x) \mu(x) \ell(x) e(x) \frac{\int_x^\infty \ell(a)^2 da}{\int_x^\infty \ell(a) da} dx \\ &= \int_0^\infty \rho(x) w(x) 2 \ell(x) D(x) dx, \end{aligned}$$

where $w(x) = \mu(x) \ell(x) e(x)$, which proves (6). \square

3.3. Changes over time in D in terms of mortality improvements

Eqs. (4) and (6) enable us to express changes over time in D in terms of mortality improvements. Using (2), and subsequently replacing (4) and (6) in (3), yields

$$\begin{aligned} \dot{D} &= D \left(\frac{\dot{\vartheta}}{\vartheta} - \frac{\dot{e}_0}{e_0} \right) = \frac{\dot{\vartheta} - \dot{e}_0 D}{e_0} \\ &= \int_0^\infty \rho(x) w(x) \frac{2 \ell(x) D(x) - D}{e_0} dx \\ &= \int_0^\infty \rho(x) w(x) W(x) dx. \end{aligned} \tag{7}$$

This result shows that changes over time in D (and analogously in G) are a total average of mortality improvements weighted by

$w(x) W(x)$, where $w(x) = \mu(x) \ell(x) e(x)$ are the same weights as in (4) and

$$W(x) = \frac{2 \ell(x) D(x) - D}{e_0} \tag{8}$$

4. The threshold age

4.1. Positive and negative contributions to lifespan equality

Because Drewnowski's index is a measure of equality, $\dot{D} > 0$ indicates that lifespan variation decreases over time, whereas $\dot{D} < 0$ implies that lifespan variation increases over time. Eq. (7) can then be used to analyze the existence of a threshold age that separates positive from negative contributions to lifespan equality as a result of mortality improvements.

In the assumption that mortality improvements occur at all ages, $\rho(x) = -\dot{\mu}(x) / \mu(x) > 0$ is a strictly positive function. Therefore, from (7),

1. Those ages x for which $w(x) W(x) > 0$ will contribute positively to Drewnowski's index D and increase lifespan equality;
2. Those ages x for which $w(x) W(x) < 0$ will contribute negatively to Drewnowski's index D and favor lifespan inequality;
3. Those ages x for which $w(x) W(x) = 0$ will have no effect on the variation over time of D .

Any existing threshold age that separates positive from negative contributions to lifespan equality will occur whenever $w(x) W(x) = 0$. Since $\mu(x)$, $\ell(x)$, and $e(x)$ are all positive functions, so are $w(x)$ and e_0 . Hence,

$$w(x) W(x) = 0 \iff 2 \ell(x) D(x) - D = 0 \tag{9}$$

4.2. Existence and uniqueness of the threshold age

By means of the following two propositions and one theorem, we aim to prove that when mortality improvements occur at all ages and $\rho(x) > 0$ for all $x \geq 0$, there exists a unique threshold age a^D that separates positive from negative contributions to lifespan equality (measured by D) as a result of those improvements. The assumption of $\rho(x)$ being a strictly positive function is necessary to ensure that the existence of a threshold age and its uniqueness only depend on the weights and the ages at which $w(x) W(x) = 0$. The same assumption was made in previous works that identified the threshold age of other lifespan variation indicators (Zhang and Vaupel, 2009; Gillespie et al., 2014; Aburto et al., 2019). If $\rho(x)$ were to take both positive and negative values, the threshold age may not be unique. Following (7), the threshold age would also be unique if $\rho(x)$ were strictly negative and there were mortality increases for all ages over time. However, we did not explore that scenario.

Remark. Following (2), Drewnowski's index D is bounded between 0 and 1, reaching a value of 1 at complete equality in the ages at death within a population. A score of 0 would express maximum inequality in the ages at death. By definition, however, this value can never be reached:

$$\begin{aligned} D = 0 &\iff \frac{\int_0^\infty \ell(x)^2 dx}{\int_0^\infty \ell(x) dx} = 0 \iff \int_0^\infty \ell(x)^2 dx = 0 \\ &\iff \ell(x) = 0 \end{aligned} \tag{10}$$

for all ages $x \geq 0$. This implies that the denominator in (10) equals 0 because $\ell(x) \geq 0$ is always positive and, consequently, D would be undefined. Hence, $0 < D \leq 1$.

Proposition 3. Let $\ell(x)$ be the probability of surviving from birth to age x , D Drenowski's index as defined in (2), and $D(x)$ as defined in (5). Define the function $g(x) := 2 \ell(x) D(x) - D$. Then, there exists at least one age a^D such that $g(a^D) = 0$.

Proof. At age $x = 0$,

$$g(0) = 2 \ell(0) D(0) - D = 2 D - D = D > 0 \tag{11}$$

by definition, since $0 < D \leq 1$.

When ages become arbitrarily large,

$$\lim_{x \rightarrow \infty} g(x) = \lim_{x \rightarrow \infty} (2 \ell(x) D(x) - D) = 2 \lim_{x \rightarrow \infty} \ell(x) D(x) - D,$$

which only depends on the behavior of $\ell(x) D(x)$. Because $\ell(x) \in [0, 1]$ for all ages $x \geq 0$, we have that $0 \leq \ell(x)^2 \leq \ell(x)$ and

$$0 \leq \lim_{x \rightarrow \infty} \int_x^\infty \ell(a)^2 da \leq \lim_{x \rightarrow \infty} \int_x^\infty \ell(a) da. \tag{12}$$

Let $e(x)$ be the remaining life expectancy at age x , since

$$\lim_{x \rightarrow \infty} \int_x^\infty \ell(a) da = \lim_{x \rightarrow \infty} e(x) \ell(x) = 0,$$

both integrals in (12) tend to 0 as x approaches ∞ . Consequently,

$$\lim_{x \rightarrow \infty} \ell(x) D(x) = \lim_{x \rightarrow \infty} \frac{\int_x^\infty \ell(a)^2 da}{\int_x^\infty \ell(a) da}$$

is indeterminate, but applying L'Hôpital's rule, we get

$$\begin{aligned} \lim_{x \rightarrow \infty} \ell(x) D(x) &= \lim_{x \rightarrow \infty} \frac{\frac{\partial}{\partial x} \int_x^\infty \ell(a)^2 da}{\frac{\partial}{\partial x} \int_x^\infty \ell(a) da} = \lim_{x \rightarrow \infty} \frac{-\ell(x)^2}{-\ell(x)} \\ &= \lim_{x \rightarrow \infty} \ell(x) = 0. \end{aligned}$$

As a result,

$$\lim_{x \rightarrow \infty} g(x) = 2 \lim_{x \rightarrow \infty} \ell(x) D(x) - D = 0 - D < 0. \tag{13}$$

Finally, using (11) and (13), on a continuous framework the intermediate value theorem guarantees the existence of at least one positive age a^D at which $g(a^D) = 0$. \square

Proposition 4. Let $\ell(x)$ be the probability of surviving from birth to age x , D Drenowski's index as defined in (2), and $D(x)$ as defined in (5). Then, $g(x) := 2 \ell(x) D(x) - D$ is a strictly decreasing function.

Proof. In order to demonstrate that $g(x)$ is a strictly decreasing function it suffices to show that its first derivative is negative for all ages $x \geq 0$. Note that since D does not depend on age,

$$\frac{\partial}{\partial x} g(x) < 0 \iff \frac{\partial}{\partial x} (\ell(x) D(x)) < 0.$$

Applying the quotient rule together with the fundamental theorem of calculus, we get

$$\begin{aligned} \frac{\partial}{\partial x} (\ell(x) D(x)) &= \frac{\partial}{\partial x} \left(\frac{\int_x^\infty \ell(a)^2 da}{\int_x^\infty \ell(a) da} \right) \\ &= \frac{\int_x^\infty \ell(a) da \frac{\partial}{\partial x} (\int_x^\infty \ell(a)^2 da) - \int_x^\infty \ell(a)^2 da \frac{\partial}{\partial x} (\int_x^\infty \ell(a) da)}{(\int_x^\infty \ell(a) da)^2} \\ &= \frac{\int_x^\infty \ell(a) da (-\ell(x)^2) - \int_x^\infty \ell(a)^2 da (-\ell(x))}{(\int_x^\infty \ell(a) da)^2}. \end{aligned}$$

Hence,

$$\begin{aligned} \frac{\partial}{\partial x} g(x) < 0 &\iff \ell(x) \int_x^\infty \ell(a)^2 da - \ell(x)^2 \int_x^\infty \ell(a) da < 0 \\ &\iff \frac{1}{\ell(x)^2} \int_x^\infty \ell(a)^2 da < \frac{1}{\ell(x)} \int_x^\infty \ell(a) da. \end{aligned}$$

Note that $\ell(x) = \exp[-\int_0^x \mu(a) da]$ for a given age-specific hazard function $\mu(x)$. Therefore, $\ell(x)^2 = \exp[-\int_0^x 2 \mu(a) da]$ can be interpreted as the survival schedule with doubling hazard $2 \mu(x)$ at all ages $x \geq 0$. We can then define $\tilde{e}(x) = \int_x^\infty \ell(a)^2 da / \ell(x)^2$ as the remaining life expectancy at age x of a population with survival schedule $\ell(x)^2$ and age-specific force of mortality $2 \mu(x)$. Accordingly,

$$\begin{aligned} \frac{\partial}{\partial x} g(x) < 0 &\iff \frac{1}{\ell(x)^2} \int_x^\infty \ell(a)^2 da < \frac{1}{\ell(x)} \int_x^\infty \ell(a) da \\ &\iff \tilde{e}(x) < e(x) \end{aligned}$$

for all $x \geq 0$, which holds true since doubling the hazard corresponds to a lower remaining life expectancy, in the reasonable assumption that $\mu(x) > 0$ for all ages. \square

Theorem. Let $D = \vartheta / e_0$ be Drenowski's index, where $\vartheta = \int_0^\infty \ell(x)^2 dx$, $e_0 = \int_0^\infty \ell(x) dx$ is the life expectancy at birth, and $\ell(x)$ the probability of surviving from birth to age x . Assume mortality improvements over time occur at all ages. Then, there exists a unique threshold age a^D that separates positive from negative contributions to lifespan equality, measured by D , as a result of those improvements.

Proof. Following (7), changes over time in D can be expressed as a weighted average of mortality improvements, given by

$$\dot{D} = \int_0^\infty \rho(x) w(x) W(x) dx,$$

where $\rho(x)$ are the age-specific rates of mortality improvement over time, and $w(x) W(x)$ the weights. By assumption, $\rho(x) > 0$ for all ages $x \geq 0$. Therefore, any threshold age that separates positive from negative contributions to lifespan equality as a result of mortality improvements will occur whenever $w(x) W(x) = 0$. From (9),

$$w(x) W(x) = 0 \iff 2 \ell(x) D(x) - D = 0,$$

where $D(x)$ is as defined in (5). Proposition 3 guarantees the existence of at least one positive age a^D such that $2 \ell(a^D) D(a^D) - D = 0$. In addition, from Proposition 4 the function $g(x) := 2 \ell(x) D(x) - D$ is strictly decreasing. Hence, assuming continuity, $g(x)$ is a one-to-one function and therefore the threshold age a^D is unique. \square

Corollary. Let G be the Gini coefficient as defined in (1). Provided that $G = 1 - D$, following (7)

$$\dot{G} = -\dot{D} = - \int_0^\infty \rho(x) w(x) W(x) dx.$$

Hence, G and D have the same threshold age, which is unique in the assumption that mortality improvements occur at all ages. Improvements below the threshold age will reduce lifespan variation (lowering G , but enlarging D), and improvements above will increase lifespan inequality (enlarging G , but lowering D).

5. Application

We illustrate our theoretical findings first by simulating scenarios under the Gompertz mortality model, and next by showing an example of application to Swedish life table data from the 20th century. The R code (R Core Team, 2021) and data to reproduce all the figures and results presented in the following are publicly available for research purposes from the GitHub repository <https://github.com/panchoVG/Drenowski>.

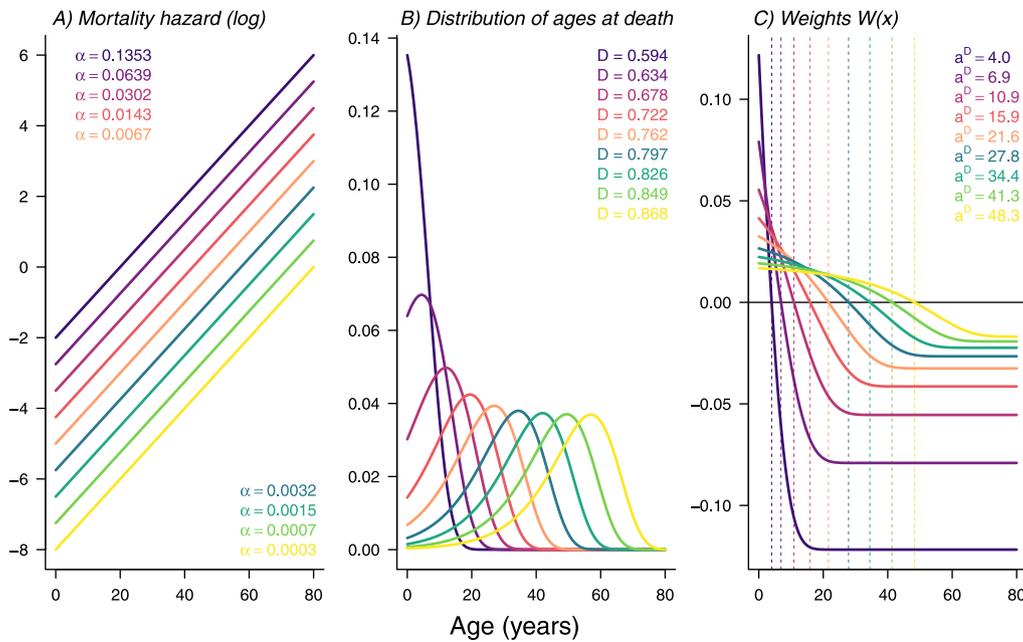


Fig. 1. Gompertz mortality model with positive aging ($\beta > 0$) for different levels of baseline mortality α and a fixed rate of aging $\beta = 0.1$.

5.1. The Gompertz model and the threshold age

We explore different scenarios of mortality improvement depending on level versus rate changes in the Gompertz mortality model (Gompertz, 1825). This model captures an exponential change of the force of mortality $\mu(x) = \alpha \cdot e^{\beta x}$ over age x , where α is the baseline level of mortality and β the rate of aging. As previously mentioned, for any hazard function $\mu(x)$ the corresponding survival is $\ell(x) = \exp[-\int_0^x \mu(a) da]$, and the distribution of deaths $d(x) = \mu(x)\ell(x)$ follows immediately. The survival function of the Gompertz model is given by

$$\ell(x) = \exp\left[\frac{\alpha}{\beta} (1 - e^{\beta x})\right]. \tag{14}$$

Applying (14) to (2), (5), (8) and (9), and using different values of α and β , we calculate the corresponding Drenowski indices D , weights $W(x)$, and threshold ages a^D reported in Figs. 1 to 5.

Specifically, we explore patterns of increasing, constant, and decreasing mortality with age. While our simulations are hypothetical, these patterns are found across the tree of life (Jones et al., 2014). Humans and non-human primates experience increasing exponential mortality from early ages (Colchero et al., 2021), but constant patterns of mortality with unchanged risk of death over age are observed in organisms such as *Hydra* (Schaible et al., 2015). Some species like turtles may show declining rates of mortality in adulthood (Jones et al., 2014), something that is commonly referred to as ‘negative senescence’ (Vaupel et al., 2004; Baudisch, 2008; Cayuela et al., 2019).

5.1.1. Gompertz force of mortality with positive aging: Level vs. rate change

Our first scenario demonstrates how improvements in mortality affect our outcome variables when progress results from reducing the level of mortality, but not from slowing the rate of aging. Fig. 1 depicts the Gompertz mortality function on a log-scale (Panel A), the corresponding age-at-death distribution (Panel B), and associated weights $W(x)$ (Panel C), for a fixed rate of aging $\beta = 0.1$ and changing levels of baseline mortality α . Values of Drenowski’s index D and corresponding threshold ages a^D are reported in Panels B and C, respectively.

Lower levels of baseline mortality α translate into vertical downward shifts in the log-hazard (Panel A). Correspondingly, lifespan variation falls as deaths concentrate at older ages, as captured by larger values of D (Panel B). Lower levels of mortality raise the threshold age a^D at which the weights $W(x)$ switch sign from positive to negative (Panel C). Positive weights at some age imply that saving lives at that age increases equality, i.e. enlarging D ; negative weights imply that saving lives decreases equality, i.e. lessening D . The threshold age a^D marks the boundary between these positive and negative effects of life saving on lifespan equality and is indicated by the dashed vertical lines. Panel C shows that under the assumption of mortality improvements over all ages, this threshold age is unique and that the magnitude of the weights $W(x)$ over age changes depending on the different shapes of the distribution of deaths, supporting our theoretical results.

Complementing these findings, Fig. 2 analyzes how slowing the rate of aging β affects lifespan variation. For a fixed baseline level of mortality $\alpha = e^{-6} \approx 0.0025$, Panel A illustrates how lower values of β translate, as expected, into a slower rise in the log-hazard. In contrast to the first scenario, Panel B reveals that slowing the rate of aging β reduces D (lighter colors) and widens the distribution of ages at death. That is, extending lifespan by slowing the rate of aging increases lifespan inequality, which is consistent with previous research by Colchero et al. (2021). The threshold age a^D increases as lifespan equality decreases. While life saving has opposite effects on lifespan variability and the level of the threshold age in the two scenarios, the general dynamics of the weights $W(x)$ remain similar. Saving lives before the threshold age reduces inequality; saving lives later increases inequality. The threshold age a^D remains unique, independent of specific values of the mortality parameters, further supporting our theoretical predictions.

5.1.2. Zero rate of aging

Fig. 3 captures a scenario in which the rate of aging equals zero. Under varying but constant levels of mortality (Panel A), lifespan variation remains unchanged at $D = 0.5$ and the largest number of deaths occur at the earliest ages (Panel B). The lower the level of mortality, the longer the length of life, and the later the threshold age (Panel C).

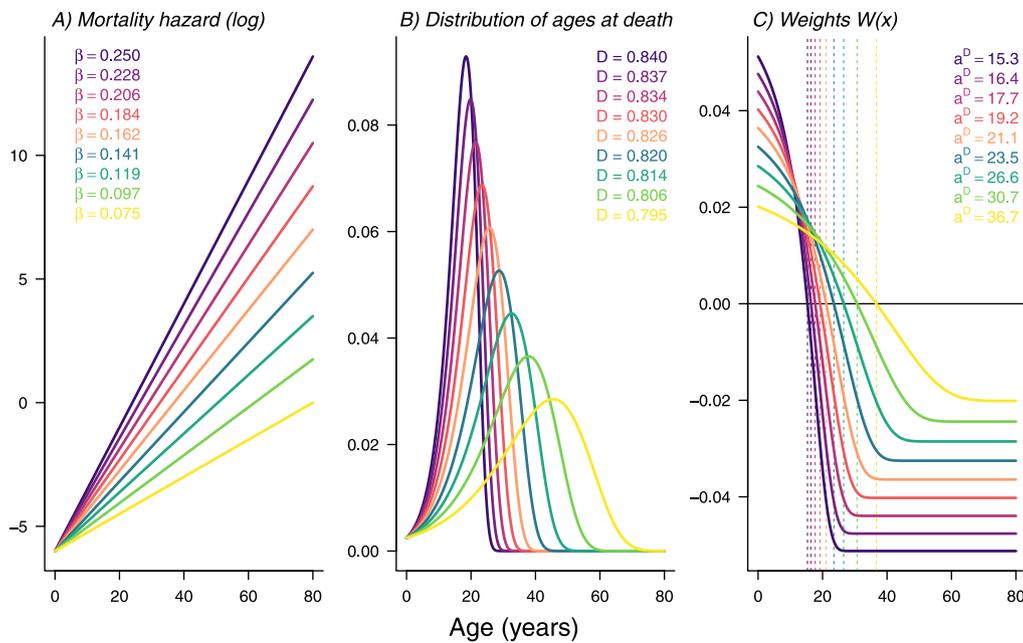


Fig. 2. Gompertz mortality model with positive aging for different rates of aging $\beta > 0$ and a fixed level of mortality $\alpha = e^{-6}$.

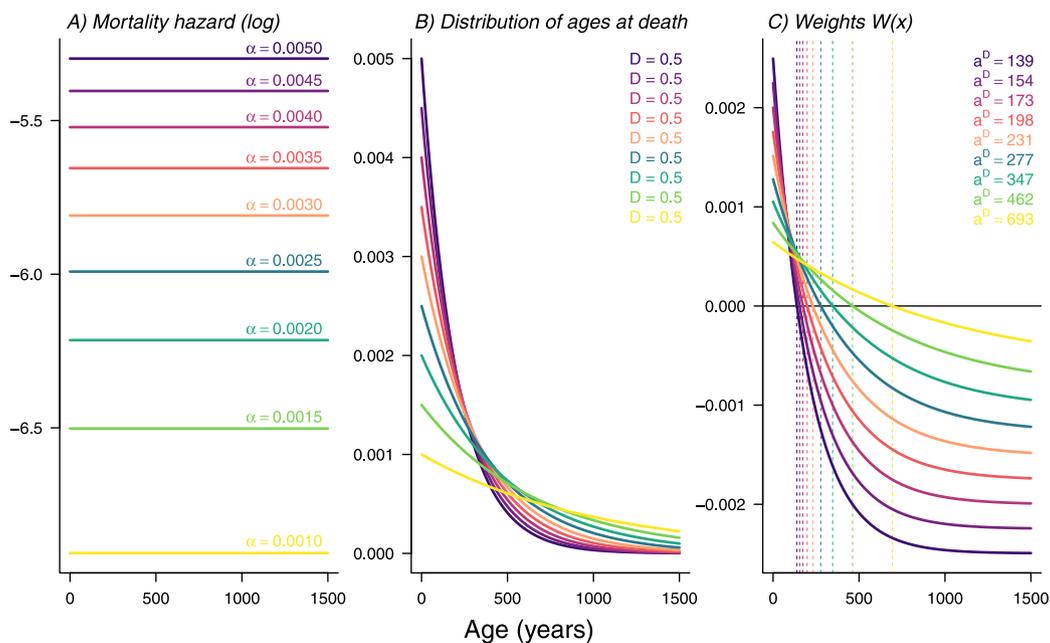


Fig. 3. Gompertz mortality model with zero rate of aging ($\beta = 0$) and different levels of baseline mortality α .

This can also be verified analytically. When $\beta = 0$, the survival function for the Gompertz mortality model simplifies to $\ell(x) = e^{-\alpha x}$. Then, life expectancy as the integral of the survivorship over all ages becomes $1/\alpha$, which is the inverse of the force of mortality. Following Definition 1, in this scenario the conditional Drenowski index $D(x)$ is

$$D(x) = \frac{1}{e^{-\alpha x}} \frac{e^{-2\alpha x} / 2\alpha}{e^{-\alpha x} / \alpha} = 0.5 .$$

In other words, when mortality is constant over age, $D(x) = 0.5$ for all ages and it is independent of the baseline mortality level α . Accordingly, from Proposition 3

$$g(x) = 0 \iff 2 e^{-\alpha x} 0.5 - 0.5 = 0 \iff x = \ln(2) / \alpha ,$$

which implies that the threshold age is $a^D = \ln(2) / \alpha$ and $\ell(a^D) = e^{-\alpha a^D} = 0.5$. This means that, when mortality is constant, the threshold age and the median age at death are the same.

5.1.3. Negative rate of aging

To complete the analyses, Figs. 4 and 5 capture scenarios of decreasing mortality, which are consistently marked by values of D below one half (Panels B). Different to the scenario with positive aging rates, here effects of both level and rate changes point in the same direction: higher mortality results in less lifespan variation (higher values of D in Panels B). Both Drenowski's index and the threshold ages barely change, and the latter remain close to the earliest age (Panels C). This is because in these scenarios most

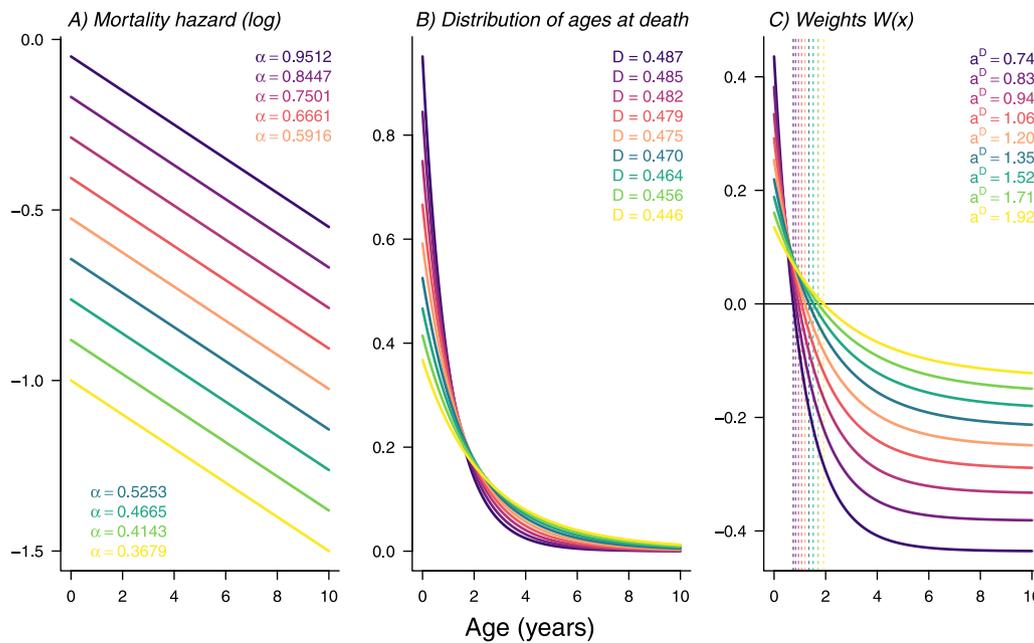


Fig. 4. Gompertz mortality model with negative rate of aging ($\beta < 0$) for different levels of baseline mortality α and a fixed rate of aging $\beta = -0.05$.

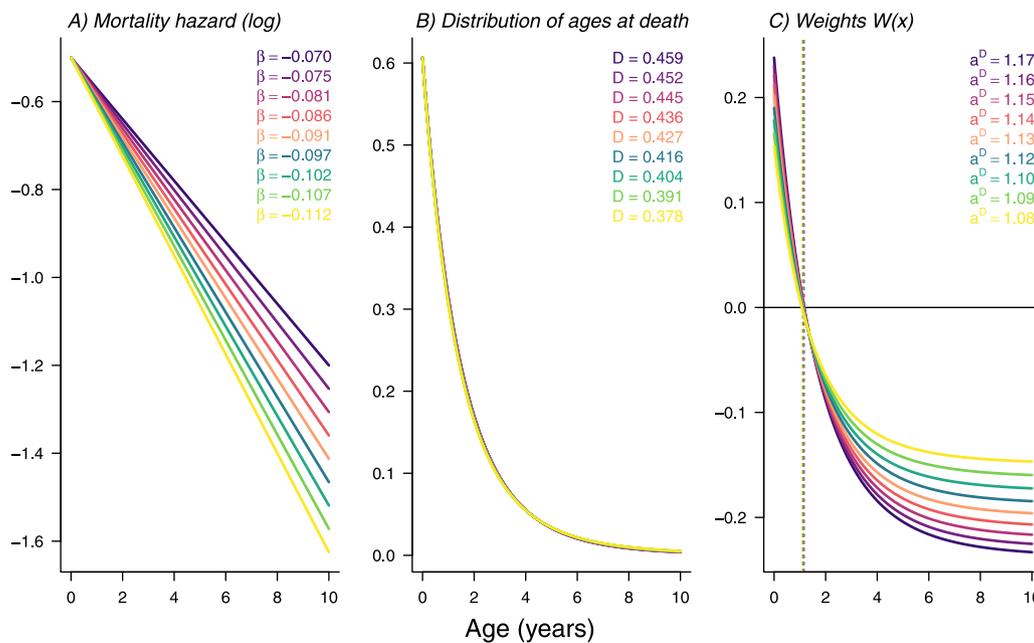


Fig. 5. Gompertz mortality model with negative rate of aging ($\beta < 0$) for different rates of aging β and a fixed level of mortality $\alpha = e^{-0.5}$.

individuals die young, and life expectancy is extremely short for all cases.

The two negative aging scenarios substantially differ, however, in the maximum age for the survivors (not shown). This reflects the exceeding disparity among the majority of individuals who die upon – or shortly after – birth, and those few surviving to manifold higher ages as they benefit from the improvements in mortality over age. These differences are also reflected in the density functions: while varying the rate of aging leaves no distinguishable effect on the density function (Fig. 5, Panel B), varying the baseline level of mortality α distinctly affects the shape of the density function with lower mortality levels yielding an increasing rectangularization (Fig. 4, Panel B).

5.2. Rates of mortality improvement among Swedish females

To show the practical usefulness of our framework, we analyze mortality patterns for Swedish females, whose life expectancy has improved over the last decades. We apply the discrete approximation suggested by Vaupel (1986) to estimate age-specific rates of mortality improvement. Let ${}_nq_x^{t_1}$ denote the probability of death between ages x and $x + n$ at time t_1 in a given population, and ${}_nq_x^{t_2}$ the corresponding probability at time $t_2 > t_1$. Then, the rate of mortality improvement from age x to $x + n$ between times t_1 and t_2 can be approximated as

$${}_n\rho_x = \frac{\ln(-\ln(1 - {}_nq_x^{t_1})) - \ln(-\ln(1 - {}_nq_x^{t_2}))}{t_2 - t_1} \tag{15}$$

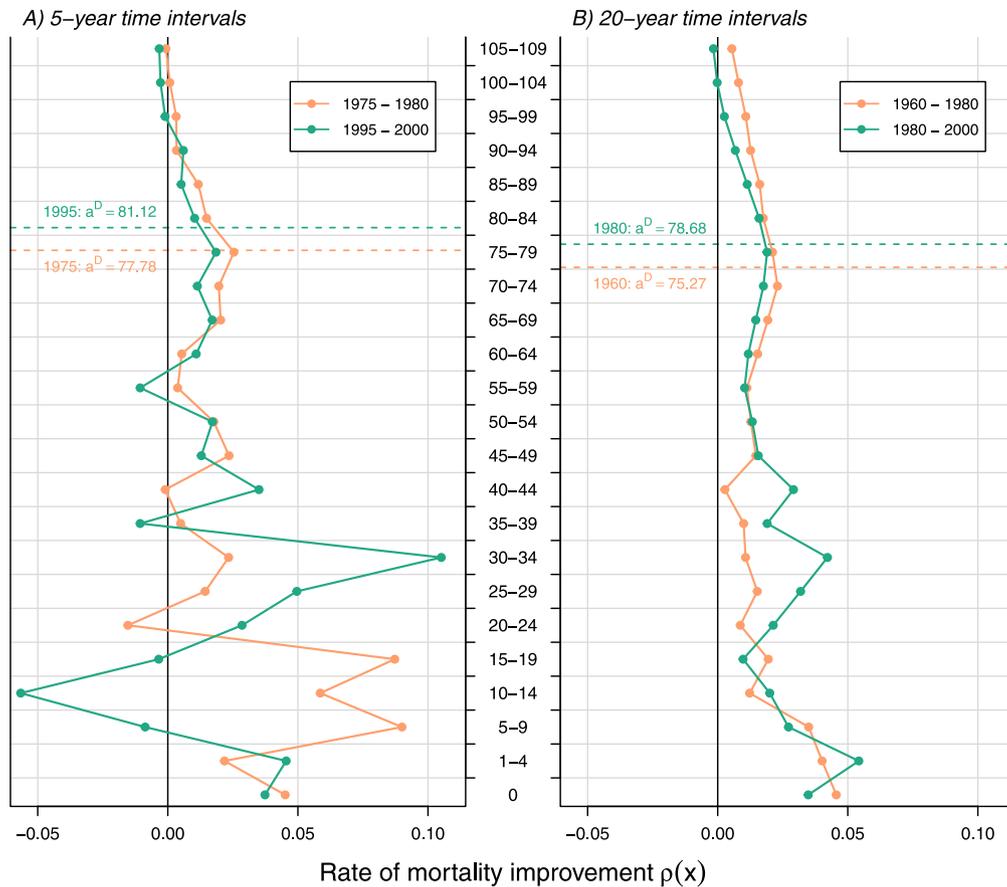


Fig. 6. Age-specific rates of mortality improvement (5-year age groups), Swedish females. Panel A shows estimated improvements in 5-year time intervals (1975–1980 and 1995–2000), whereas Panel B shows estimated improvements in 20-year time intervals (1960–1980 and 1980–2000). Dashed horizontal lines mark corresponding threshold ages. Source: Human Mortality Database, 2022.

Fig. 6 illustrates the results of applying (15) to life table data for Swedish females from the Human Mortality Database (2022). Panel A shows estimated improvements in 5-year time intervals (1975–1980 and 1995–2000), whereas Panel B shows estimated improvements for 20-year periods (1960–1980 and 1980–2000).

Although the assumption of $\rho(x)$ being a strictly positive function does not hold for the whole time period, in these time windows the threshold ages fall within the age ranges in which mortality improvements are observed. Over time, threshold ages are postponed to higher ages. Aligned with these dynamics, life expectancy for Swedish females increased from 74.88 in 1960 to 82.02 in 2000 (Human Mortality Database, 2022). The improvements depicted in Fig. 6 are responsible for these increments in life expectancy. During the same period, lifespan equality measured by D increased from 0.899 to 0.921. This means that, on average, lifespans increased and also became more predictable for Swedish females.

The example of Sweden can only serve as an illustration, and results may differ for other countries. In particular, mortality improvements may be more volatile in low- and middle-income countries, or during mortality crises such as the Covid-19 pandemic. Researchers should apply Eq. (15) to identify the age ranges in which the assumption of positive rates of mortality improvement holds.

6. Discussion

In this article we uncovered how age-specific patterns of mortality underpin trends in lifespan variation as measured by

Drewnowski’s index D , the complement of the Gini coefficient of the life table, by means of formal demography. We contribute to the literature by disentangling how changes in age patterns of mortality affect lifespan variation and provide an analytical proof for the existence of a threshold age a^D below which mortality improvements are translated into increasing lifespan equality and above which these improvements translate into increasing lifespan inequality. Previous research determined such age for the life table entropy (Aburto et al., 2019), the variance (Gillespie et al., 2014) and years of life lost (Zhang and Vaupel, 2009).

We test and illustrate our results under multiple scenarios of mortality changes with age, including positive, zero, and negative rates of aging. This is relevant because shapes of mortality patterns across the tree of life vary substantially (Jones et al., 2014). Our experiments demonstrate how Drewnowski’s index D can serve as an indicator of the ‘shape’ of mortality patterns (Baudisch, 2011; Wrycza et al., 2015), distinguishing between increasing mortality ($D > 0.5$), constant mortality ($D = 0.5$), and decreasing mortality ($D < 0.5$) over age. These properties, along with our analytical findings, support studying lifespan variation alongside life expectancy trends in multiple species.

The existence of a unique threshold age, though, is conditioned on having positive rates of mortality improvement throughout the whole age range, which is certainly a strong assumption. Using Sweden as a benchmark, we hypothesize that among human populations this may be likely to hold at adult ages (60–95 years) in which more deaths are concentrated, and where it may be more meaningful to identify a (unique) threshold age. Above that

range, one could argue that the small fraction of the remaining deaths does not suffice to counterbalance the uniqueness of the threshold age, though we have not tested that explicitly. Our results also show that our framework may be suitable for studying long term trends (20 years), so that mortality improvement can be substantiated across a wide age range.

This framework may also be applicable for non-human species when comparing populations, not chronologically over time, but ordered from harsher to milder conditions, so that improvements in survival can be studied across changing environments (Colchero et al., 2019). Such analyses may aid understanding the principle limits of the plasticity of aging in different species (Colchero et al., 2021)

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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