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## The Inverse Relationship Between Life Expectancy-Induced Changes in the Old-Age Dependency Ratio and the Prospective Old-Age Dependency Ratio

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## The Inverse Relationship Between Life Expectancy-Induced Changes in the Old-Age Dependency Ratio and the Prospective Old-Age Dependency Ratio

### Abstract

Unlike other biological populations, the human population is experiencing long-run increases in life expectancy. Those lead to changes in age compositions not typical for other biological populations. Sanderson and Scherbov (2015a) demonstrated that, in many countries in Europe, faster increases in life expectancy lead to faster population aging when measured using the old-age dependency ratio and to slower population aging when measured using the prospective old-age dependency ratio that employs a dynamic old-age threshold. We examine this finding analytically and with simulations. We use an analytic decomposition of changes in mortality schedules into shift and compression processes. We show that shifts and compressions of mortality schedules push the two old-age dependency ratios in opposite directions. Our formal results are supported by simulations that show a positive effect of a mortality shift on the old-age dependency ratio and a negative effect of it on the prospective old-age dependency ratio. The effects are of opposite sign for a mortality compression. Our formal and simulation results generalize observed European trends and suggest that the inverse relationship between life expectancy and prospective old-age dependency would be observed more generally.

**Keywords:** Aging; Old-Age Dependency Ratio; Prospective Old-Age Dependency Ratio; Life-Table Population; Mortality Shift; Mortality Compression.

## 1. Introduction

Human populations share many properties, such as renewal, with other biological populations. They also have unique features among which are the long-run lengthening of lifespan. When age-specific survival probabilities are fixed, increases in chronological age are typically associated with decreases in average remaining lifetimes. When age-specific survival probabilities are declining, it is possible for average remaining lifetimes to increase even when the average number of years already lived also increases (Sanderson and Scherbov 2005).

When lifetimes are lengthening, population aging can be analyzed from two different perspectives, one based on the average number of years already lived and another based on the average number of years left to live. Sanderson and Scherbov (2005, 2015a) have shown, using historical data and population forecasts, that these two perspectives can often yield strikingly different results. Because their findings were based on patterns of survival from the recent past and on forecasts based on them, it is not clear what causes those differences and the extent to which their findings are more general or are specific to the era from which their data were derived.

In this paper, we provide a theoretical analysis of the old-age dependency ratio, a commonly used measure of human population aging, from the two perspectives. Old age dependency ratios are ratios of the number of people who are classified as being old to those who are classified as being in the working ages. Our analysis uses the theoretical framework in Ediev (2013a). This framework allows changes in human mortality schedules to be expressed using two parameters, one related to the shift of the age distribution of deaths and the other to its compression or expansion. Sanderson and Scherbov (2005, 2015a) showed that when changes in life expectancy occur it matters whether a perspective based on the number of years already lived or one based on the number of years left to live is used in computing old-age dependency ratios. Not only are the magnitudes of the changes different, but even the direction of the change. The theoretical results presented here show the analytic conditions under which this occurs.

We begin with formal definitions of the old-age dependency ratio and the prospective old-age dependency ratio.

The conventional Old-Age Dependency Ratio (OADR) is defined as:

$$OADR(t) = \frac{OAP(t)}{WAP(t)} = \frac{\int_{R_0}^{\infty} P(t,x)dx}{\int_W^{R_0} P(t,x)dx}. \quad (1)$$

Here,  $OADR(t)$  is the OADR at time  $t$ ,  $OAP(t)$  is the old-age population at time  $t$  obtained by summing the population  $P(t, x)$  over ages  $R_0$ , the fixed old age threshold to the maximum lifespan (the latter is replaced by infinity to facilitate further derivations). The working-age population at time  $t$ ,  $WAP(t)$ , is the population between the ages  $W$ , the onset of the working age interval, and  $R_0$ . In one commonly used version of the OADR,  $W$  is set to age 20 and  $R_0$  is set equal to 65.

The definition of the Prospective Old-Age Dependency Ratio (POADR) requires a time-varying old-age threshold,  $R(t)$ , that holds remaining life expectancy constant. To do this, we first choose some base year  $t_0$  and compute the time varying old age threshold as the age in year  $t$  where remaining life expectancy is the same as at age  $R_0$  in the base year  $t_0$ . For numerical illustrations, we use a rolling base  $t_0$ . For example, for year  $t$ , we use year  $t_0 = t - 1$  as the base year. The base-year relation  $R(t_0) = R_0$  assures a similar 'start' for both the conventional and prospective indicators of population aging. For example, if  $t_0$  were 2015 and  $R_0$  were 65, then we would first find the remaining life expectancy at age 65 in 2015. Let us suppose that this life expectancy was 20 years. If year  $t$  were 2016, we would determine  $R(2016)$  by finding the age in 2016 where remaining life expectancy was 20 years. When the time varying old age threshold  $R(t)$  is used instead of the fixed old age threshold,  $R_0$ , a new ratio is obtained where the numerator is the number of people at or above  $R(t)$  and the denominator is the number of people from age  $W$  to  $R(t)$ . This new measure is called the POADR. Specific implementations of the POADR concept can be found in (Sanderson and Scherbov, 2010, 2013, 2015b):

$$POADR(t) = \frac{\int_{R(t)}^{\infty} P(t,x)dx}{\int_W^{R(t)} P(t,x)dx}, \quad (2)$$

where  $POADR(t)$  is the POADR at time  $t$ .

POADRs, based on estimates and forecasts, at 5-year intervals from 1950 to 2100, have been published for most countries of the world (Sanderson and Scherbov, 2008; Wittgenstein Centre for Demography and Global Human Capital, 2014). In those POADRs, the old age threshold,  $R(t)$ , is set equal to the age at which remaining life expectancy equals 15 years. The POADR was designed to study population aging in an environment where the characteristics of older people could vary markedly over time and space. This was the motivation for making the old age threshold dynamic. For simplicity, the age at labor force entry,  $W$ , is kept constant.

**Figure 1: Annual change of life-table Prospective Old-Age Dependency Ratio (POADR) (2) vs the Old-Age Dependency Ratio (OADR) (1), entire Human Mortality Database, years 1950-2015: men, women, currently High- and Low-mortality countries**

**Figure 2: Increments of life-table Prospective Old-Age Dependency Ratio (POADR) (2) vs the Old-Age Dependency Ratio (OADR) (1) in model life tables: men, women, Coale-Demeny and United Nations model life table families. Note:  $e_0$  is the life expectancy at birth (years).**

Sanderson and Scherbov (2015a) used population forecasts for European countries to demonstrate a novel result. When the speed of aging is measured using the increase in OADR, faster increases in life expectancy lead to faster population aging. When the speed of aging is measured using the increase in the POADR, faster increases in life expectancy lead to slower increases in aging. Figure 1 shows a similar phenomenon in a different context. It shows the relationship between annual changes in OADRs and POADRs defined over life table populations

for the years 1950 to 2015 for currently low and high mortality countries in the Human Mortality Database (University of California (Berkeley) and Max Planck Institute for Demographic Research (Rostock), 2018). The list of currently ‘low mortality’ countries is obtained by excluding Eastern-European countries from the HMD. It includes Australia, Austria, Belgium, Canada, Chile, Denmark, England and Wales, Finland, France, Germany (total population), Ireland, Israel, Italy, Japan, Netherlands, New Zealand (Non-Maori), Northern Ireland, Norway, Portugal, Scotland, Spain, Sweden, Switzerland, Taiwan (China), USA, UK, and West Germany. We also exclude some relatively small populations (Iceland, New Zealand--Maori, and Luxemburg). “High-mortality” countries include the Eastern-European countries. The annual changes in the OADRs and POADRs in Figure 1 are both in response to the same changes in mortality conditions.

In Figure 1, we can see a strong negative relationship between the annual changes in OADRs and POADRs. In this paper, we want to understand whether the relationship extends beyond mortality schedules currently observed in HMD. The relationship between changes in old-age dependency ratios and changes prospective old-age dependency ratios in model life tables (Coale and Demeny 1966; United Nations 1982) suggests the relationship is general, with few exceptions. In Figure 2, we plot increments of conventional and prospective OADRs within the same model life table family against one year increments of life expectancy at birth. The model life tables cover a wide range of values of life expectancy at birth from  $e_0=20$  years to  $e_0=100$  years and – unlike the empirical schedules in Figure 1 – are free from stochastic variations. Although they have these advantages, they also have the disadvantage that they are based on imputations and may be misleading at the lower and upper extremes of life expectancy at birth. The model life tables show the same inverse relationship between OADR and POADR that is seen in the observed life tables with a few exceptions at low life expectancy at birth. The model life table-based relationship is curvilinear. We explain this below in the section on analytic derivations. The object

here is to explain analytically why this inverse relationship exists in both observed and model life tables.

We focus on the effects of mortality change alone. To do this, we study dependency ratios in stationary populations with survival constant over time and equal to the survival  $l(t, x)$  of the year  $t$ . In that context, assuming unit radix  $P(t, 0) = l(t, 0) = 1$  of the life table population,  $P(t, x) = P(t, 0)l(t, x) = l(t, x)$ , where  $l(t, x)$  is the life table proportion surviving from birth to age  $x$  in the period life table for the year  $t$ .

The analytic framework that we use here is the shift-compression model of the age distribution of adult deaths in Ediev (2013a) and is described in Section 2. In Section 3, we show that the shift-compression model produces a closed form solution for dynamic old age threshold,  $R(t)$ . The analytic relationship between shifts and compressions of mortality schedules and changes in OADRs and POADRs is presented in Section 4. We quantify shifts and compressions in mortality schedules using data from the Human Mortality Database and show that the observed shifts and compressions produce the results seen in Figures 1 and 2 in Section 5. In the appendix, we summarize the notations used. For ease of following the analytical derivations, notations and the basic relations used in the paper are summarized in Table 1.

**Table 1: Notation and basic relations**

Notation	Description
$t$	time variable
$t_0$	base year used as a reference in computing prospective indicators of aging
$x$	age variable
$P(t, x)$	population density at age $x$ , time $t$

Notation	Description
$\omega$	the maximum lifespan
$W$	the starting age of the working age interval
$R_0$	the time-fixed starting age of the conventional old age interval
$R(t)$	the time-varying ‘old age threshold’ marking the starting age of the prospective old age interval at time $t$ , Eq. (5)
$OAP(t) = \int_{R_0}^{\omega} P(t, x) dx$	conventional old-age population
$WAP(t) = \int_W^{R_0} P(t, x) dx$	working-age population with the conventional definition of old age
$POAP(t) = \int_{R(t)}^{\omega} P(t, x) dx$	prospective old-age population above time-dependent old age threshold
$PWAP(t) = \int_W^{R(t)} P(t, x) dx$	working-age population with the prospective definition of old age
$OADR(t) = \frac{OAP(t)}{WAP(t)}$	conventional old-age dependency ratio, Eq. (1)
$POADR(t) = \frac{POAP(t)}{PWAP(t)}$	prospective old-age dependency ratio, Eq. (2)
$PO(t) = \frac{OAP(t)}{\int_0^{\omega} P(t, x) dx}$	proportion of the population who are old (conventional definition)
$PPO(t) = \frac{POAP(t)}{\int_0^{\omega} P(t, x) dx}$	proportion of the population who are old (prospective definition)
$\omega(t, x)$	the age distribution of life table adult deaths at time $t$ , Eq. (3)

Notation	Description
$A_0$	mortality model's "pivotal age" used to mark the amount of mortality shift
$A(t)$	mortality model's parameter used to describe the amount of shift ( $A(t) - A_0$ ) at the pivotal age. $A(t_0) = A_0$ by definition.
$k(t)$	mortality model's parameter used to describe the amount of compression or expansion of the distribution of ages at death
$A_0^* = A_0 + \frac{A(t) - A_0}{k(t) - 1}$	age at which the mortality model produces no shift
$\Delta(t, x)$	mortality shift at age $x$ and time period $t$ , Eq. (4)
$\delta(t) = A(t) - A(t - 1)$	the annual mortality shift
$e(t, x)$	remaining life expectancy at age $x$ and time $t$
$\mu(t, x)$	the death rate (the force of mortality) at age $x$ and time $t$
$l(t, x)$	the life table survival to age $x$ in the period life table for the year $t$
$\sigma(t, x)$	the standard deviation of ages at death above age $x$ , Eq. (27)
Base year correspondence between the conventional and prospective indicators:	$R(t_0) = R_0$ $POAP(t_0) = OAP(t_0)$ $PWAP(t_0) = WAP(t_0)$ $POADR(t_0) = OADR(t_0)$ $PPO(t_0) = PO(t_0)$

## 2. The mortality model

If arbitrary mortality changes are allowed, one may easily construct mortality scenarios with any pattern of change of the old-age dependency ratios. Neither the OADR nor the POADR are affected by mortality changes at ages below the working age. Mortality changes in the working ages push

the two old-age dependency ratios in a similar direction. However, mortality changes at ages beyond the old-age threshold  $R_0$  may generate complex patterns of change depending on how they affect: (1) the old-age threshold  $R(t)$ , (2) the life table person-years below that age and (3) the survival probability to  $R(t)$ . Therefore, the possibility that the inverse relationship between changes of OADR and POADR might be general is ruled out. The form of the relationship depends on the pattern of combinations of mortality change at different ages.

To study patterns of age-specific mortality rate changes, we consider the two most important aspects of those changes: (1) changes in the mean age at death (shift) and (2) changes in the spread of the distribution of ages at death around the mean age (compression/expansion). To this end, we use the shift-compression/expansion model of the age distribution of adult deaths (Ediev, 2013a). Here the age distribution of adult deaths is expressed in relation to a baseline age distribution. The age distribution of adult deaths can be expressed as:

$$d(t, x) \propto d\left(t_0, A_0 - \frac{A(t)-x}{k(t)}\right), \quad (3)$$

where  $x$  is age,  $t$  is time,  $d(t, x)$  is the age distribution of life table adult deaths at time  $t$ ,  $t_0$  is the base year against which we study changes in the dependency ratios. The relationship between the age distribution of adult deaths in year  $t$  and year  $t_0$  depends on two parameters  $A(t)$  and  $k(t)$ .  $A(t) - A_0$  is the amount of age shift of the distribution at the arbitrarily chosen pivotal age  $A_0 = A(t_0)$  in the original distribution of life table deaths. Hereinafter, subscript '0' refers to the base year  $t_0$ . The parameter  $k(t)$  describes the amount of compression or expansion of the distribution and  $k_0 = 1$  by definition. When  $A(t) - A_0 > 0$ , the age distribution of deaths in year  $t$  is shifted rightwards relative to the distribution in year  $t_0$ . In other words, it is shifted towards longer lifespans. When  $k(t) = 1$ , the mortality shift occurs without a change in the shape of the distribution of ages at death. When  $k(t) > 1$ , mortality expands and deaths stretch over more years of life. When  $k(t) < 1$ , the distribution of age at death is compressed.

**Figure 3: Illustrative scenarios of mortality model (3): age at death distributions (dx). Notes: The vertical broken line indicates the pivotal age  $A_0 = 65$ . (a) The original distribution of life table deaths is shifted by 10 years. (b) The original distribution of life table deaths is compressed by 25%. (c) The original distribution of life table deaths is expanded by 25%. (d) The original distribution of life table deaths is shifted by 10 years and compressed by 25%.**

In Figure 3, we present four schematic scenarios for shift and compression or expansion with the pivotal age  $A_0 = 65$ . In panel (a), the original schedule is shifted rightwards by 10 years without compression or expansion. In panel (b), compression occurs without shift. The transformed schedule is also scaled upwards so as to sum up to the same total number of deaths as in the original schedule. In panel (c), the distribution expands without a shift. In panel (d), we present the more realistic scenario of shift and compression. Typically, period mortality decline manifests itself in positive shifts combined with compression (Fries, 1980; Wilmoth and Horiuchi, 1999; Kannisto, 2000; Canudas-Romo, 2008; Thatcher et al., 2010), although cohort mortality decline may also manifest itself without compression or even with expansion (Ediev 2011, 2013b).

Before proceeding to the implications of the mortality model, it may be useful to develop a better intuition for how the model works. The choice of the pivotal age does not affect the fit of the model to empirical data and, in that sense, is arbitrary. For any pivotal age, it is always possible to set the model parameters so that the model produces exactly the same distribution of deaths. This choice makes a difference, however, for the definition and interpretation of the shift parameter. Unlike the compression/expansion parameter  $k(t)$  that has no link to any particular age, the mortality shift may only be defined with respect to a given age. Indeed, if mortality would exhibit a universal shift, equal for all ages, there could have been no compression or expansion of

the mortality distribution. All parts of the curve would be shifting without changing the distance between them. Such a pure shift, however, is a rare case. A more plausible scenario involves age-specific shifts. In our model, the variety of age-specific shifts, say,  $\Delta(t, x)$  at some age  $x$  and time  $t$  and  $\Delta(t, y)$  at another age  $y$ , are linked to each other through the compression/expansion parameter  $k(t)$ :  $[y + \Delta(t, y)] - [x + \Delta(t, x)] = k(t)(y - x)$  and, therefore,

$$\Delta(t, y) = \Delta(t, x) + [k(t) - 1](y - x). \quad (4)$$

This makes it possible to describe the whole set of age-specific shifts of the death distribution curve by the shift at one (arbitrarily chosen) age  $A_0$  and by the compression/expansion coefficient  $k(t)$ .

The choice of the pivotal age is arbitrary and has no consequences for the kinds of changes the model describes. If one wishes to opt for a different pivotal age, it is only necessary to recalculate the amount of shift at new pivotal age according to (4). When making statements about the shift, however, one must always be clear about the age to which the shift applies. For ease of interpretation, we set  $A_0 = R_0 = 65$  in all numerical and empirical illustrations. Indeed, the model simplifies the analysis of real-life mortality change as it reduces the change to two parameters by assuming a universal compression/expansion of the age distribution of deaths. A limitation of this simplification becomes evident when one notices that the model produces no shift at the age  $A_0^* = A_0 + \frac{A(t) - A_0}{k(t) - 1}$ . At ages below and above the age  $A_0^*$ , the distribution of deaths is shifted in different directions. Adult mortality, typically, changes in the same direction across all ages. That means that the model assumption of no shift at age  $A_0^*$  might be not realistic. However, for a typical mortality change, the age  $A_0^*$  is beyond the range where most of the mortality change occurs. In our data, the mean annual shift was about 0.2 years at age 65 and the mean annual compression,  $k(t) - 1$ , was 0.003. With these parameters, the model would produce non-positive shifts, on average, only at ages above  $A_0^* = 65 + \frac{0.2}{0.003} \approx 132$  years. It may also be noticed that, when we

conducted tests of fit, not shown here, we found that the model performs marginally better than the Gompertz model (Gompertz 1825). The biggest advantage of the model for our study, however, comes from the convenience and accuracy it offers in deriving formal relations for indicators of population aging.

While our mortality model describes only adult deaths, real-life mortality also includes child (most importantly infant) deaths. The latter may not be subject to the shifting process and, therefore, may not be described by our model. Changes in mortality at ages younger than  $W$ , however, modify the stationary population at working and old ages in a similar proportion and, therefore, do not alter the dependency ratios. Neither do they alter the remaining life expectancies of working- or old-age adults and, as a consequence, they do not alter the old-age threshold  $R(t)$ . Therefore, we facilitate our formal derivations, without limiting the generality of our results, by assuming that the density function of infant and child deaths not covered by model (3) is time-constant before age  $W$ . A particular consequence of this assumption is time-invariance of the integral  $\int_0^\infty d(t, x)dx$  of the density function of the adult deaths (3). Furthermore, unless otherwise indicated, the life table functions discussed below will be assumed to apply to the working and old age populations, not to the ages below  $W$  where our mortality model is not fully functional.

### 3. The Analytic Expression for the Old Age Threshold $R(t)$

The old age threshold  $R(t)$ , adopted here (Sanderson and Scherbov, 2005, 2013) is determined based on a fixed remaining life expectancy. Given our consistency constraint  $R(t_0) = R_0$ , which assures the equality of the conventional and prospective indicators of aging in year 0, the old age threshold  $R(t)$  must follow the identity:

$$e(t, R(t)) \equiv e(t_0, R_0), \quad (5)$$

where  $e(t, x)$  is life expectancy at age  $x$  in year  $t$ . To explore solutions of this identity, we use the following implication of the shift-compression/expansion mortality model (3) (Edwards 2013a):

$$e(t, x) = k(t)e\left(t_0, A_0 - \frac{A(t)-x}{k(t)}\right). \quad (6)$$

Combining this with (5), we obtain the equation for  $R(t)$ :

$$e(t, R(t)) = k(t)e\left(t_0, A_0 - \frac{A(t)-R(t)}{k(t)}\right) = e(t_0, R_0). \quad (7)$$

The second equality in (7) is satisfied by the solution to

$$R(t) = A(t) + k(t) \left[ e^{-1}\left(t_0, \frac{e(t_0, R_0)}{k(t)}\right) - A_0 \right], \quad (8)$$

where  $e^{-1}(t_0, z)$  is the inverse function of the baseline remaining life expectancy with respect to age. In the pure mortality shift scenario,  $k(t) = 1$ , Equation (8) produces the following solution for the old age threshold:

$$R(t) = R_0 + A(t) - A_0. \quad (9)$$

Under the pure mortality shift scenario, the old age threshold shifts by exactly the same number of years as the age distribution of deaths. A positive mortality shift implies a time-invariant number of life-table person years in old-age and an increasing number of life-table person years in the working ages, defined here as the ages 20 (the fixed onset of the working age interval) to the old age threshold,  $R(t)$ . Hence, that shift would produce a fall in the POADR. A negative mortality shift would produce effects opposite to those of the positive shift.

Analytically more challenging is the general case of shift combined with compression or expansion, which is considered next.

#### 4. The Analytic Relationship between the OADR and the POADR under general mortality change

In a life table (stationary) population with unit radix, the OADR may be written as

$$OADR(t) = \frac{OAP(t)}{WAP(t)} = \frac{\int_{R_0}^{\infty} l(t,x)dx}{\int_W^{R_0} l(t,x)dx}, \quad (10)$$

where,  $OAP(t) = \int_{R_0}^{\omega} l(t,x)dx$  is the old-age life table population and  $WAP(t) = \int_W^{R_0} l(t,x)dx$  is the working-age life table population.

The life table POADR is

$$POADR(t) = \frac{POAP(t)}{PWAP(t)} = \frac{\int_{R(t)}^{\infty} l(t,x)dx}{\int_W^{R(t)} l(t,x)dx}, \quad (11)$$

where  $POAP(t) = \int_{R(t)}^{\infty} l(t,x)dx$  is the prospective old-age life table population and  $PWAP(t) = \int_W^{R(t)} l(t,x)dx$  is the working-age life table population (calculated using the prospective definition of who is old).

The two sets of aging indicators are identical in the base year  $t_0$ , so that  $R(t_0) = R_0$ ,  $POAP(t_0) = OAP(t_0)$ ,  $PWAP(t_0) = WAP(t_0)$ , and  $POADR(t_0) = OADR(t_0)$ .

Under model (3), assuming a fixed total count of adult life-table deaths ( $\int_0^{\infty} d(t,x)dx \equiv const$ ) subject to the model,  $d(t,x) = \frac{\int_0^{\infty} d(t_0,y)dy}{\int_0^{\infty} d(t_0,A_0 - \frac{A(t)-y}{k(t)})dy} d\left(t_0, A_0 - \frac{A(t)-x}{k(t)}\right)$ . Assuming,

additionally, that the support of the age distribution of adult deaths does not include age 0 at all times:

$$l(t,x) = \int_x^{\infty} d(t_0,y)dy = \frac{\int_0^{\infty} d(t_0,y)dy}{\int_0^{\infty} d\left(t_0, A_0 - \frac{A(t)-y}{k(t)}\right)dy} \int_x^{\infty} d\left(t_0, A_0 - \frac{A(t)-y}{k(t)}\right)dy =$$

$$\frac{\int_0^{\infty} d(t_0,z)dz}{k(t) \int_0^{\infty} d(t_0,z)dz} k(t) \int_{A_0 - \frac{A(t)-x}{k(t)}}^{\infty} d(t_0,z)dz =$$

$$\int_{A_0 - \frac{A(t)-x}{k(t)}}^{\infty} d(t_0, z) dz = l\left(t_0, A_0 - \frac{A(t)-x}{k(t)}\right). \quad (12)$$

Therefore,

$$\begin{aligned} OAP(t) &= \int_{R_0}^{\infty} l(t, x) dx = \int_{R_0}^{\infty} l\left(t_0, A_0 - \frac{A(t)-x}{k(t)}\right) dx = k(t) \int_{A_0 - \frac{A(t)-R_0}{k(t)}}^{\infty} l(t_0, z) dz \approx \\ &k(t)OAP(t_0) - k(t)l(t_0, R_0) \left[ A_0 - \frac{A(t)-R_0}{k(t)} - R_0 \right] \approx \\ &OAP(t_0) \left\{ 1 + (A(t) - A_0) \frac{l(t_0, R_0)}{OAP(t_0)} - [1 - k(t)] \left[ 1 + \frac{l(t_0, R_0)}{OAP(t_0)} (R_0 - A_0) \right] \right\}. \end{aligned} \quad (13)$$

$$\begin{aligned} WAP(t) &= \int_W^{R_0} l(t, x) dx = \int_W^{R_0} l\left(t_0, A_0 - \frac{A(t)-x}{k(t)}\right) dx = k(t) \int_{A_0 - \frac{A(t)-W}{k(t)}}^{A_0 - \frac{A(t)-R_0}{k(t)}} l(t_0, z) dz \approx \\ &k(t)WAP(t_0) + [A(t) - A_0][l(t_0, W) - l(t_0, R_0)] - \\ &[1 - k(t)][l(t_0, R_0)(R - A_0) - l(t_0, W)(A_0 - W)] - \\ &WAP(t_0) \left\{ 1 + [A(t) - A_0] \frac{l(t_0, W) - l(t_0, R_0)}{WAP(t_0)} - [1 - k(t)] \left[ 1 + \frac{l(t_0, R_0)(R - A_0) - l(t_0, W)(A_0 - W)}{WAP(t_0)} \right] \right\}. \end{aligned} \quad (14)$$

Combining this with (10) and (13):

$$\begin{aligned} OADR(t) &\approx OADR(t_0) \times \\ &\frac{1 + [A(t) - A_0] \frac{l(t_0, R_0)}{OAP(t_0)} - [1 - k(t)] \left[ 1 + \frac{l(t_0, R_0)}{OAP(t_0)} (R_0 - A_0) \right]}{1 + [A(t) - A_0] \frac{l(t_0, W) - l(t_0, R_0)}{WAP(t_0)} - [1 - k(t)] \left[ 1 + \frac{l(t_0, R_0)(R - A_0) - l(t_0, W)(A_0 - W)}{WAP(t_0)} \right]} \approx \\ &OADR(t_0) \left\{ 1 + [A(t) - A_0] \left[ \frac{l(t_0, R_0)}{OAP(t_0)} - \frac{l(t_0, W) - l(t_0, R_0)}{WAP(t_0)} \right] - \right. \\ &\left. [1 - k(t)] \left[ \frac{l(t_0, R_0)(R_0 - A_0)}{OAP(t_0)} + \frac{l(t_0, R_0)(R_0 - A_0) + l(t_0, W)(A_0 - W)}{WAP(t_0)} \right] \right\}. \end{aligned} \quad (15)$$

The effect of the mortality shift in (15) will be positive for human mortality change, because

$$\frac{l(t_0, R_0)}{OAP(t_0)} - \frac{l(t_0, W) - l(t_0, R_0)}{WAP(t_0)} = \frac{1}{e(t_0, R_0)} - \frac{l(t_0, W) - l(t_0, R_0)}{l(t_0, W)e(t_0, W) - l(t_0, R_0)e(t_0, R_0)} =$$

$\frac{1}{e(t_0, R_0)} \left[ 1 - \frac{l(t_0, W) - l(t_0, R_0)}{\frac{e(t_0, W)}{e(t_0, R_0)} l(t_0, W) - l(t_0, R_0)} \right] > 0$  assuming  $\frac{e(t_0, W)}{e(t_0, R_0)} > 1$ . The effect of the compression, on the

other hand, will be negative for all pivotal ages  $A_0 \leq R_0$ . Setting  $A_0 = R_0$ , as we do in our empirical assessments, and assuming the limiting case of negligible low mortality in the working ages, it follows from (15) that:

$$OADR(t) \approx OADR(t_0) \left\{ 1 + [A(t) - A_0] \frac{1}{OAP(t_0)} - [1 - k(t)] \right\}. \quad (16)$$

To express the POADR analytically, we first need to estimate the old age threshold  $R(t)$ . To this end, we derive the first-order approximation for the inverse function in Eq. (8) (noticing that  $k(t_0) = 1$  by definition):

$$e^{-1} \left( t_0, \frac{e(t_0, R_0)}{k(t)} \right) \approx e^{-1} (t_0, e(t_0, R_0)) + \frac{1}{e'_x(t_0, R_0)} \left[ \frac{e(t_0, R_0)}{k(t)} - e(t_0, R_0) \right] = R_0 + \frac{1}{e'_x(t_0, R_0)} \left[ \frac{e(t_0, R_0)}{k(t)} - e(t_0, R_0) \right], \quad (17)$$

where,  $e'_x(t_0, R_0)$  is the partial derivative with respect to age of  $e(t, x)$  at  $x = R_0$  and  $t = t_0$ .

Substituting (17) into (8), we get the desired relation for the old age threshold:

$$R(t) \approx A(t) + k(t) \left\{ R_0 + \frac{1}{e'_x(t_0, R_0)} \left[ \frac{e(t_0, R_0)}{k(t)} - e(t_0, R_0) \right] - A_0 \right\} = A(t) + k(t)(R_0 - A_0) + [1 - k(t)] \frac{e(t_0, R_0)}{e'_x(t_0, R_0)}, \quad (18)$$

To simplify the equation for the old age threshold (18), we note that  $e'_x(t_0, R_0) = -1 + \mu(t_0, R_0)e(t_0, R_0)$  and rewrite Eq. (18):

$$R(t) \approx A(t) + k(t)(R_0 - A_0) - [1 - k(t)] \frac{e(t_0, R_0)}{1 - \mu(t_0, R_0)e(t_0, R_0)}. \quad (19)$$

The first two terms in (19) describe the shift of the death distribution at the age corresponding to  $R_0$  in the original distribution. The last term shows the additional correction to the old age threshold due to mortality compression/expansion. In the case of mortality expansion, the old age threshold increases by more than the pure shift. In the case of compression, it increases by less.

Having obtained the change in the old age threshold (19), we move on to estimate the corresponding change in the survival function. This is necessary in order to determine the old-age population. Substituting  $x = R(t)$  in (12),

$$l(t, R(t)) = l\left(t_0, A_0 - \frac{A(t) - R(t)}{k(t)}\right). \quad (20)$$

Combining this with (19) and using first-order approximations leads to

$$\begin{aligned} l(t, R(t)) &\approx l\left(t_0, R_0 - \frac{1}{k(t)} \frac{[1-k(t)]e(t_0, R_0)}{1-\mu(t_0, R_0)e(t_0, R_0)}\right) \approx l(t_0, R_0) - l'_x(t_0, R_0) \frac{1}{k(t)} \frac{[1-k(t)]e(t_0, R_0)}{1-\mu(t_0, R_0)e(t_0, R_0)} = \\ &l(t_0, R_0) + \frac{\mu(t_0, R_0)l(t_0, R_0)}{k(t)} \frac{[1-k(t)]e(t_0, R_0)}{1-\mu(t_0, R_0)e(t_0, R_0)} \approx l(t_0, R_0) \left\{1 + [1-k(t)] \frac{\mu(t_0, R_0)e(t_0, R_0)}{1-\mu(t_0, R_0)e(t_0, R_0)}\right\}. \end{aligned} \quad (21)$$

The life-table survival to the old age threshold is not influenced by the mortality shift. It is only affected by the mortality compression or decompression. It increases with compression and decreases with mortality expansion.

Because the life-table population at old ages is the product of the number surviving to the old age threshold multiplied by the remaining life expectancy at that age and because the latter is, by definition, time constant in the definition of the prospective old age, a relation similar to (21) applies to the life table prospective old-age population:

$$POAP(t) = \int_{R(t)}^{\infty} l(t, x) dx \approx POAP(t_0) \left\{1 + [1-k(t)] \frac{\mu(t_0, R_0)e(t_0, R_0)}{1-\mu(t_0, R_0)e(t_0, R_0)}\right\}. \quad (22)$$

Equation (22) shows that the prospective old-age population does not depend on the mortality shift, but does depend on the extent of compression/expansion. It will increase with mortality compression and decrease with expansion. Another consequence of (22) is that the POADR will fall in the case of a mortality decline accompanied by an expansion or pure shift.

Using the equation for the survival function, an alternative approximation of the denominator in (11) is:

$$PWAP(t) = \int_{W}^{K(t)} l\left(t_0, A_0 - \frac{A(t) - x}{k(t)}\right) dx = k(t) \int_{A_0 - \frac{A(t) - W}{k(t)}}^{A_0 - \frac{A(t) - R(t)}{k(t)}} l(t_0, z) dz \approx$$

$$k(t)PWAP(t_0) - k(t)l(t_0, W) \left[ A_0 - \frac{A(t)-W}{k(t)} - W \right] + k(t)l(t_0, R_0) \left[ A_0 - \frac{A(t)-R(t)}{k(t)} - R_0 \right]. \quad (23)$$

Substituting  $R(t)$  from (18), rearranging and keeping only the first-order terms

$$\begin{aligned} PWAP(t) &\approx k(t)PWAP(t_0) - l(t_0, W)\{k(t)A_0 - A(t) + [1 - k(t)]W\} - \\ &l(t_0, R_0)[1 - k(t)] \frac{e(t_0, R_0)}{1 - \mu(t_0, R_0)e(t_0, R_0)} \approx k(t)PWAP(t_0) + l(t_0, W) \{ \\ &\{A(t) - A_0 + [1 - k(t)][A_0 - W]\} - [1 - k(t)] \frac{OAP(t_0)}{1 - \mu(t_0, R_0)e(t_0, R_0)}. \end{aligned} \quad (24)$$

Finally, combining this with (11) and (22) and noticing the base-year equalities between the conventional and prospective indicators, we obtain:

$$\begin{aligned} POADR(t) &= \frac{POAP(t)}{PWAP(t)} \approx \\ &\frac{OAP(t_0) \left\{ 1 + [1 - k(t)] \frac{\mu(t_0, R_0)e(t_0, R_0)}{1 - \mu(t_0, R_0)e(t_0, R_0)} \right\}}{k(t)WAP(t_0) + l(t_0, W)\{A(t) - A_0 + [1 - k(t)][A_0 - W]\} - [1 - k(t)] \frac{OAP(t_0)}{1 - \mu(t_0, R_0)e(t_0, R_0)}} \approx \\ POADR(t_0) &\left[ 1 + [1 - k(t)] \frac{\mu(t_0, R_0)e(t_0, R_0)}{1 - \mu(t_0, R_0)e(t_0, R_0)} + [1 - k(t)] - \frac{l(t_0, W)}{WAP(t_0)} \times \right. \\ &\left. \{A(t) - A_0 + [1 - k(t)][A_0 - W]\} - [1 - k(t)] \frac{OAP(t_0)}{WAP(t_0)[1 - \mu(t_0, R_0)e(t_0, R_0)]} \right] = \\ POADR(t_0) &\left\{ 1 - [A(t) - A_0] \frac{l(t_0, W)}{WAP(t_0)} + \right. \\ &\left. [1 - k(t)] \left[ 1 - \frac{l(t_0, W)}{PWAP(t_0)} (A_0 - W) + \frac{OADR(t_0) + \mu(t_0, R_0)e(t_0, R_0)}{1 - \mu(t_0, R_0)e(t_0, R_0)} \right] \right\}. \end{aligned} \quad (25)$$

The effect of a positive shift in (25) is always negative, while the sign of the effect of compression depends on the pivotal age  $A_0$ . When  $A_0$  is set equal to  $R_0$ , as we do here, the effect of the compression in (25) is typically positive, because the ratio  $\frac{WAP(t_0)}{l(t_0, W)}$ , the average duration of life at ages  $W$  to  $R_0$  for people surviving to age  $W$ , does not differ much from the upper limit  $R_0 - W$ . For example, in the case of French women, with  $W = 20$  and  $R_0 = 65$ , the expression  $1 -$

$\frac{l(t_0, W)}{WAP(t_0)}(R_0 - W)$  was -0.23 in 1900, -0.07 in 1950 and only -0.02 in 2013. These numbers were small as compared to the magnitude of the last summand in the expression for the compression effect in (25) where  $\frac{OADR(t_0) + \mu(t_0, R_0)e(t_0, R_0)}{1 - \mu(t_0, R_0)e(t_0, R_0)}$  was 1.04 in 1900, 0.78 in 1950, and 0.71 in 2013. Hence, the effects of both shift and compression on the change of the POADRs are typically opposite in sign to their effects on the OADRs.

One may find it counterintuitive that the compression effect in (25) depends on the choice of the parameter  $A_0$  describing the mortality shift. Parameter  $A_0$  determines the age to which the mortality shift refers. Shifts of the mortality curve at all other ages are described by the combination of the shift and the compression parameters (4). The higher the age  $A_0$ , the more the compression effect in (25) is ‘contaminated’ by the effects of shifts at younger ages. Our numerical assessments, not presented here in detail, suggest the compression effect remains positive at all  $A_0$  below age 90. One important implication of this observation is that the effect of compression with respect to the modal age, often used to describe the mortality shift, is positive.

Typically, the effect of a shift at age  $A_0 = R_0$  in (25) dominates the effect of compression and the net effect of mortality decrease is negative. To develop the intuition for this observation, consider the limit case of a population with negligible mortality at ages between  $W$  and  $R_0$ . In that population, (25) simplifies to:

$$POADR(t) \approx POADR(t_0) \left\{ 1 - [A(t) - A_0] \frac{1}{R_0 - W} + [1 - k(t)] OADR(t_0) \right\}, \quad (26)$$

and the effect of a shift will be about  $-\frac{1}{65-20} \approx -0.02$  when the mortality shift is one year, while the effect of a one percent compression will be  $0.01 \cdot OADR(t_0)$ , that is about 0.003 per one percent of compression assuming a typical  $OADR(t_0) \approx 0.3$ . To have an idea of the relative contributions of a shift and a compression at age  $R_0 = 65$ , consider a case when mortality compresses to age  $A_0^* = 100$  years. In such a scenario, a one percent compression will result in a

shift of  $0.01(100 - 65) = 0.35$  years at age 65. Given the shift and compression effect coefficients in (26), the scenario will result in a change in the POADR of  $-0.02 \cdot 0.35 = -0.007$  due to the shift and a change of 0.003 due to the compression effect.

Empirically, the relative effect of shifts is even stronger because mortality declines over time even at age 100. The average annual mortality shift at age 65 in low-mortality Human Mortality Database countries after 1950 was about 0.2 years and the annual compression was about 0.3%. The effect of the observed shifts on the POADR has been stronger than the effect of the observed compressions by about four times,  $0.02 \cdot 0.2 / (0.003 \cdot 0.2) \approx 4.4$ . Hence, the POADR, typically, decreases, as mortality declines.

One may naturally be interested in whether our conclusion about the oppositely signed effects of shifts and compressions on the two old-age dependency ratios, when the effect of a shift exceeds the effect of a compression, is more generally applicable to cases where  $R_0$  is higher than 65. We did not study this aspect in detail, but numerical analysis of (25) and (15) suggests that our conclusions about the signs of the effects remain valid at all  $R_0$ 's up to age 90.

Comparing (16) to (26), we see that the change in the POADR will, approximately, be  $-OADR(t_0)$ , i.e., currently, about -0.3, times the change in the conventional OADR for a population with negligibly low mortality at working ages.

## 5. Empirical assessment

We examine the accuracy of the first-order approximations based on empirical data from the Human Mortality Database (University of California (Berkeley) and Max Planck Institute for Demographic Research (Rostock), 2018) that covers 31 currently low-mortality populations and 12 currently higher mortality populations.

In our empirical study, we set  $R_0$  equal to age 65 for consistency with the commonly used old age threshold. We also define  $A_0 = R_0 = 65$  to simplify the interpretation of the shift

parameter. To estimate the amounts of mortality shift and compression,  $k(t)$ , we used Eq. (6) and a similar equation for the standard deviation of ages at death above age  $x$  (Ediev 2013a):

$$\sigma(t, x) = k(t)\sigma\left(t_0, A_0 - \frac{A(t)-x}{k(t)}\right). \quad (27)$$

We apply both equations to the pivotal age  $A(t)$  to obtain:

$$\frac{\sigma(t, A(t))}{\sigma(t_0, A_0)} = \frac{e(t, A(t))}{e(t_0, A_0)} = k(t) \quad (28)$$

We estimate the annual shift  $\delta(t) = A(t) - A(t-1)$  by setting  $t_0 = t - 1$  and solving the first equality in (28) numerically and then estimating the compression coefficient  $k(t)$  from the second equality.

**Figure 4: Estimates of annual shifts and compressions of the life table distributions of deaths, entire Human Mortality Database: men, women**

In Figure 4, we show estimates of annual changes in the shift and compression parameters, computed separately for men and women, using data from the Human Mortality Database countries. In agreement with the previous empirical studies and theories (Fries, 1980; Wilmoth and Horiuchi, 1999; Ediev, 2013a, 2013b), a positive period mortality shift is, typically, accompanied by the compression of the age at death distribution. Negative shifts, on the other hand, usually accompany mortality expansion, which is also in agreement with theory (Ediev 2011). Our database covers a wide range of the shift and compression values, and of their combinations. The large number of negative shifts in low mortality countries seen in the figure is not a surprise as the annual mortality changes are rather volatile and may easily go against the main trend. On average, though, the distributions of age at death were shifting rightwards with a slight compression. The annual mortality shift in low-mortality Human Mortality Database countries after 1950 was about 0.2 years and the annual compression was about 0.3%.

Using the Human Mortality Database data, we ran linear regressions with no intercept using annual changes in the OADR and POADR as independent variables and taking annual shifts and compressions as dependent variables. We present the results in Table 2. There we pooled together results for both the high- and low-mortality populations, except for observations based on very small populations like the New Zealand-Maori that were excluded from the analysis. All effects are highly significant. The p-values are not shown in the table because they are all negligible. As expected from our formal inquiry, the effects of shift and compression are of opposite sign, and the effects change sign when switching from the conventional to the prospective old-age dependency ratio. Our empirical estimates of effects, about -0.006 for a one-year shift and 0.2 for a one-percent compression, on the change in the POADR are somewhat different from the rough assessments based on the first-order approximation (26), which were -0.02 and 0.3 respectively. The empirical relation between the changes of the POADR and the OADR, however, with the POADR change being approximately -0.38 times the change in the OADR, is close to our first-order approximation of -0.3 times, as we discussed above.

**Table 2: Regression results for the shift and compression effects (linear model, no intercept) on the annual changes of  $OADR(t)$  and  $POADR(t)$ : entire Human Mortality Database, men, women, excluding small-size populations (Iceland, New Zealand--Maori, and Luxemburg)**

Indicator	Sex	Shift effect	Compression effect	R <sup>2</sup>
$OADR(t)$	women	0.0170 (0.0001)	-0.356 (0.002)	0.961
$OADR(t)$	men	0.0139 (0.0001)	-0.294 (0.002)	0.929
$POADR(t)$	women	-0.0061 (0.0001)	0.171 (0.001)	0.862
$POADR(t)$	men	-0.0058 (0.0001)	0.159 (0.002)	0.750

**Note:** numbers in the parentheses indicate standard errors of regression coefficients.

**Figure 5: Increments of life-table Prospective Old-Age Dependency Ratio (POADR) divided by the initial Old-Age Dependency Ratio (OADR) vs the increments of Old-Age Dependency Ratio (OADR) in model life tables: men, women, Coale-Demeny and United Nations model life tables' families.**

Our analytical approximations for regimes with low mortality in the working ages suggest the POADR change should be roughly equal to the initial OADR multiplied by the OADR change. To check validity of this approximation, in Figure 5 we present the POADR increments divided by the initial OADR versus OADR increments in model life tables (each model life table compared to another one with one year longer life expectancy at birth from the same family of model life tables). With the exception of cases with low life expectancy at birth (below 60 years) the model life tables produce patterns consistent with our analytical findings. The anomalous results at low life expectancies may be due to two factors. First, in that range our approximations may be too rough. Second, at low life expectancy the model life tables themselves could be problematic. UN and Coale-Demeny model life tables differ most from one another when life expectancy is low, possibly because in that range they are based more on analytic approximations and less on observations.

## 6. Conclusion

The OADR assumes that the old age starts at some fixed age regardless of time or place. Nevertheless, in a world where life expectancy is increasing, where people are often healthier at given ages than they were in the past, where age-specific cognitive functioning is improving, where older people are now more educated than they were in the past, and where people in OECD countries will generally be facing higher normal pension ages, another measure of aging, consistent with these changes, seems appropriate. The POADR is such a measure.

Population aging, viewed from the perspective of the POADR looks very different from the picture provided by the OADR. Sanderson and Scherbov (2015a) showed that faster increases in life expectancy lead to slower rates of population aging when measured by the percentage increase in the POADR, in contrast to the faster rates of population aging when measured by the percentage increase in the OADR.

We show why those differences were observed and, indeed, that they were predictable given the sorts of shifts and compressions that have been observed. Here, we have shown that, in a wide variety of life table populations, annual changes in OADRs and POADRs move in opposite directions. Ediev's (2013a) shift-compression model provides an analytic two-parameter specification of the age distribution of adult deaths. We used that model to provide analytic expressions for both the OADR and the POADR in terms of shift and compression parameters. The theoretical expressions that we obtained predicted that the observed negative relationship between annual changes in OADRs and POADRs is exactly what we should expect to see. We estimated the shift and compression parameters using data from the countries in the Human Mortality Database. The data showed that the change in the POADR was around -0.38 times the change in the OADR. Our theoretical approximation predicted that it would be around -0.3 times the change in the OADR.

Decreasing period mortality in developed countries has been typically accompanied by a gradually diminishing mortality compression. Here, we show that these sort of changes will result in increasing numbers of people considered old even using the prospective definition. That number will stop increasing, however, if mortality compression stalls (Bongaarts 2005; Canudas-Romo 2008) and gives way to a pure mortality shift. In either scenario, be it mortality compression or shift, our results show that, in life table populations, the number of people below the old age threshold marking the onset of the old age will grow faster than the number of people old, so that POADR will fall as lifespans increase. In non-stationary populations, there are factors, other than

mortality, that influence age structure. An earlier study (Sanderson and Scherbov 2015) of OADR and POADR changes in observed populations suggests, however, that the effects of fertility and migration on the changes in those aging indicators might be secondary compared to the effects of mortality that we have elucidated here.

## References

- Bongaarts, J., 2005. Long-Range Trends in Adult Mortality: Models and Projection Methods. *Demography* 42, 23–49. <https://doi.org/10.1353/dem.2005.0002>
- Canudas-Romo, V., 2008. The modal age at death and the shifting mortality hypothesis. *Demographic Research* 19, 1179–1204. <https://doi.org/10.4054/DemRes.2008.19.30>
- Coale, A.J., Demeny, P.G., 1966. *Regional Model Life Tables and Stable Populations*. Princeton University Press, Princeton, NJ.
- Ediev, D.M., 2013a. Decompression of Period Old-Age Mortality: When Adjusted for Bias, the Variance in the Ages at Death Shows Compression. *Mathematical Population Studies* 20, 137–154.
- Ediev, D.M., 2013b. Mortality compression in period life tables hides decompression in birth cohorts in low-mortality countries. *Genus* 69, 53–84. <https://doi.org/10.4402/GENUS-451>
- Ediev, D.M., 2011. Life Expectancy in Developed Countries is Higher Than Conventionally Estimated. Implications from Improved Measurement of Human Longevity. *Journal of Population Ageing* 4, 5–32. <https://doi.org/10.1007/s12062-011-9040-x>
- Fries, J.F., 1980. Aging, natural death, and the compression of morbidity. *The New England journal of medicine* 303, 130–5. <http://doi.org/10.1056/NEJM198007173030304>
- Gompertz, B., 1825. On the Nature of the Function Expressive of the Law of Human Mortality, and on a New Mode of Determining the Value of Life Contingencies. *Philosophical Transactions of the Royal Society of London* 115, 513–583.
- Kannisto, V.V., 2000. Measuring the Compression of Mortality. *Demographic Research* 3, [24] p. <https://doi.org/10.4054/DemRes.2000.3.6>
- Sanderson, W.C., Scherbov, S., 2015a. Faster increases in human life expectancy could lead to slower population aging. *PLoS ONE* 10, e0121922. <https://doi.org/10.1371/journal.pone.0121922>
- Sanderson, W.C., Scherbov, S., 2015b. Are we overly dependent on conventional dependency ratios? *Population and Development Review* 41, 687–708. <https://doi.org/10.1111/j.1728-4457.2015.00091.x>
- Sanderson, W.C., Scherbov, S., 2013. The characteristics approach to the measurement of population aging. *Popul. Dev. Rev.* 39, 673–685. <https://doi.org/10.1111/j.1728-4457.2013.00637.x>
- Sanderson, W.C., Scherbov, S., 2010. Remeasuring aging. *Science* 329, 1287–1288. <https://doi.org/10.1126/science.1193647>
- Sanderson, W.C., Scherbov, S., 2008. Conventional and Prospective Measures of Population Aging, 1995–2005, 2025, 2045.
- Sanderson, W.C., Scherbov, S., 2005. Average remaining lifetimes can increase as human populations age. *Nature* 435, 811–813.
- Thatcher, A.K., Cheung, S.L.K., Horiuchi, S., Robine, J.-M., 2010. The compression of deaths above the mode. *Demographic Research* 22, 505–538. <https://doi.org/10.4054/DemRes.2010.22.17>

- United Nations, 1982. Model life tables for developing countries (No. ST/ESA/SER.A/77). United Nations, New York.
- University of California (Berkeley), Max Planck Institute for Demographic Research (Rostock), 2018. Human Mortality Database. Online database sponsored by University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany) [WWW Document]. URL [www.mortality.org](http://www.mortality.org) (accessed 5.15.18).
- Wilmoth, J.R., Horiuchi, S., 1999. Rectangularization revisited: variability of age at death within human populations. *Demography* 36. <https://doi.org/10.2307/2648085>
- Wittgenstein Centre for Demography and Global Human Capital, 2014. Wittgenstein Centre Data Explorer, Version 1.1 [WWW Document]. URL <http://witt.null2.net/shiny/wittgensteincentredataexplorer/> (accessed 9.27.14).

## Figures

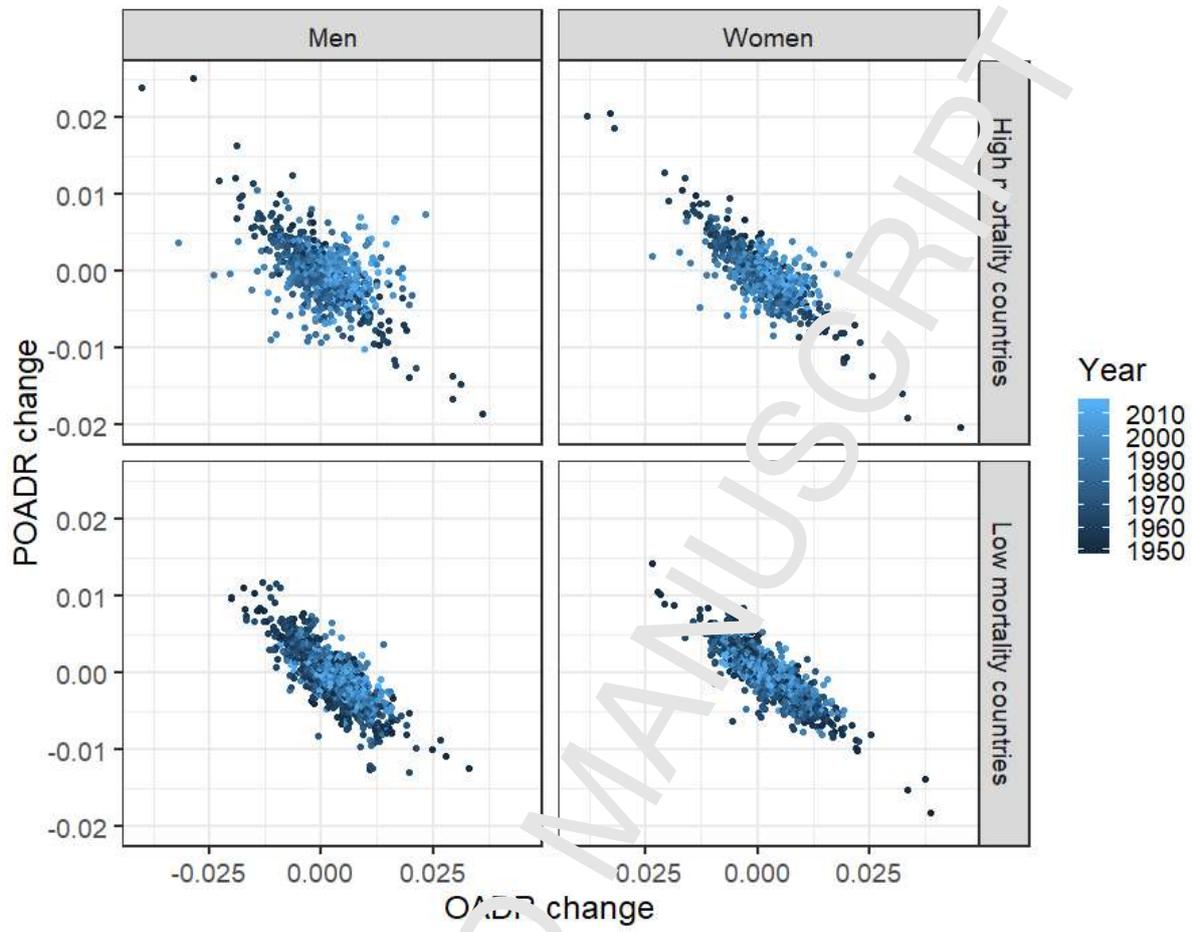


Fig. 1.

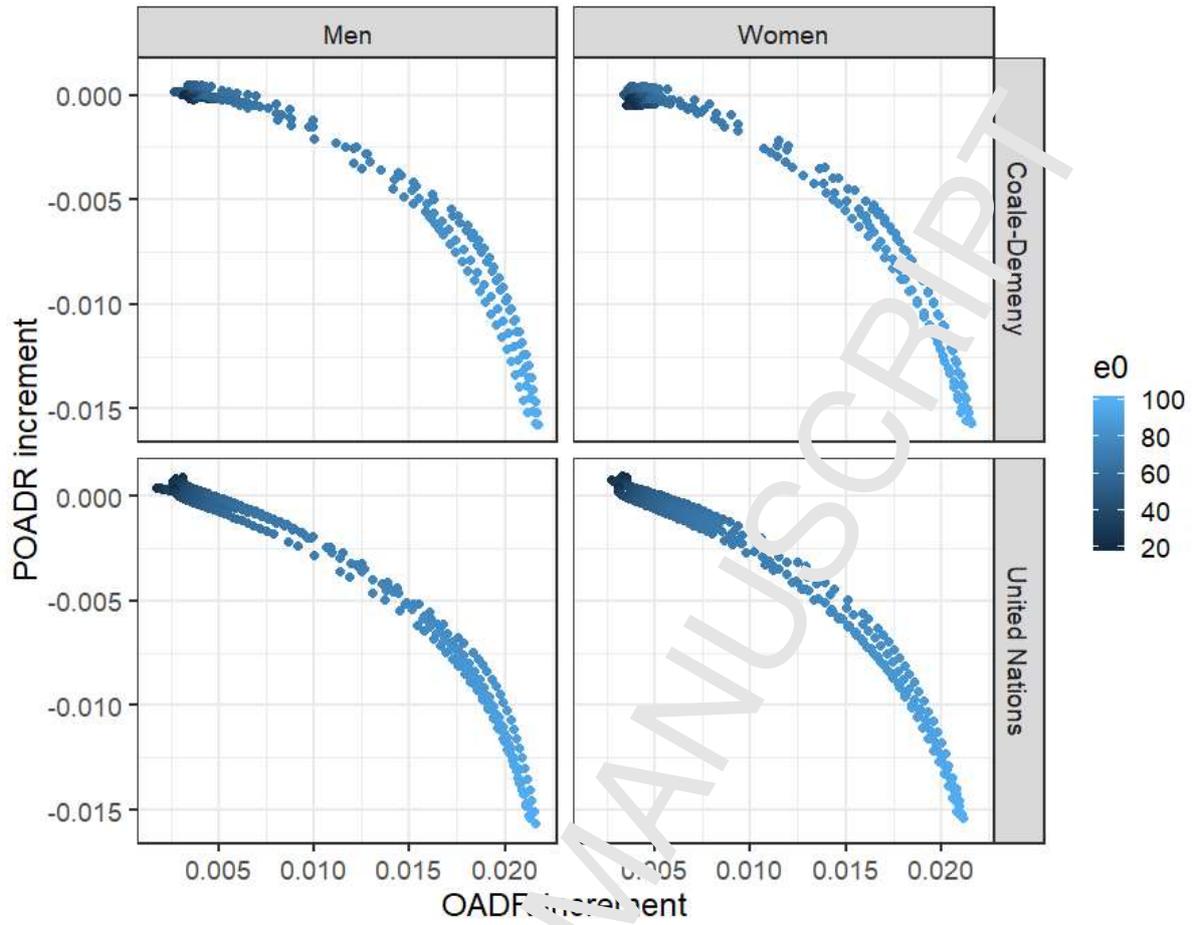


Fig. 2.

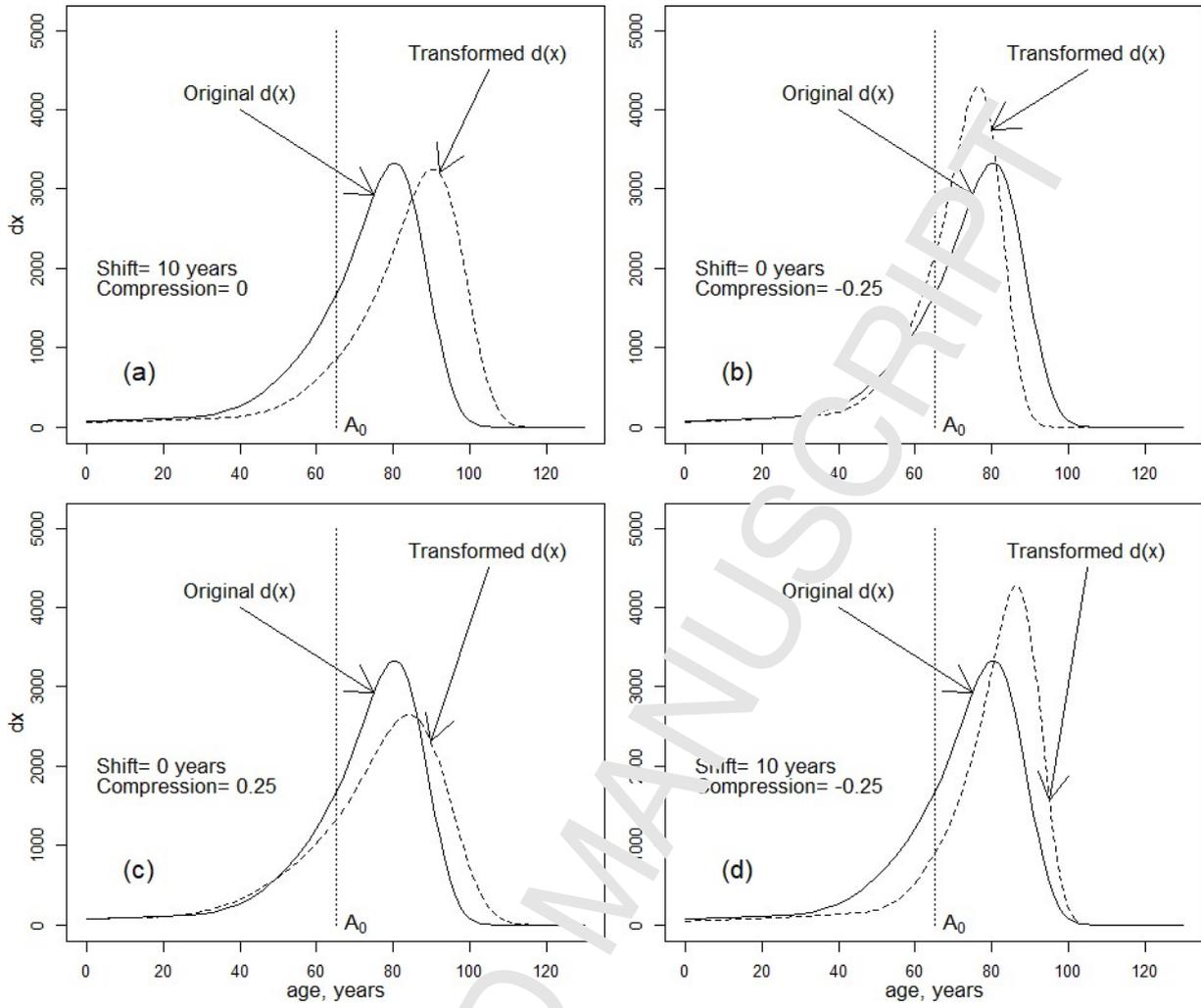


Fig. 3.

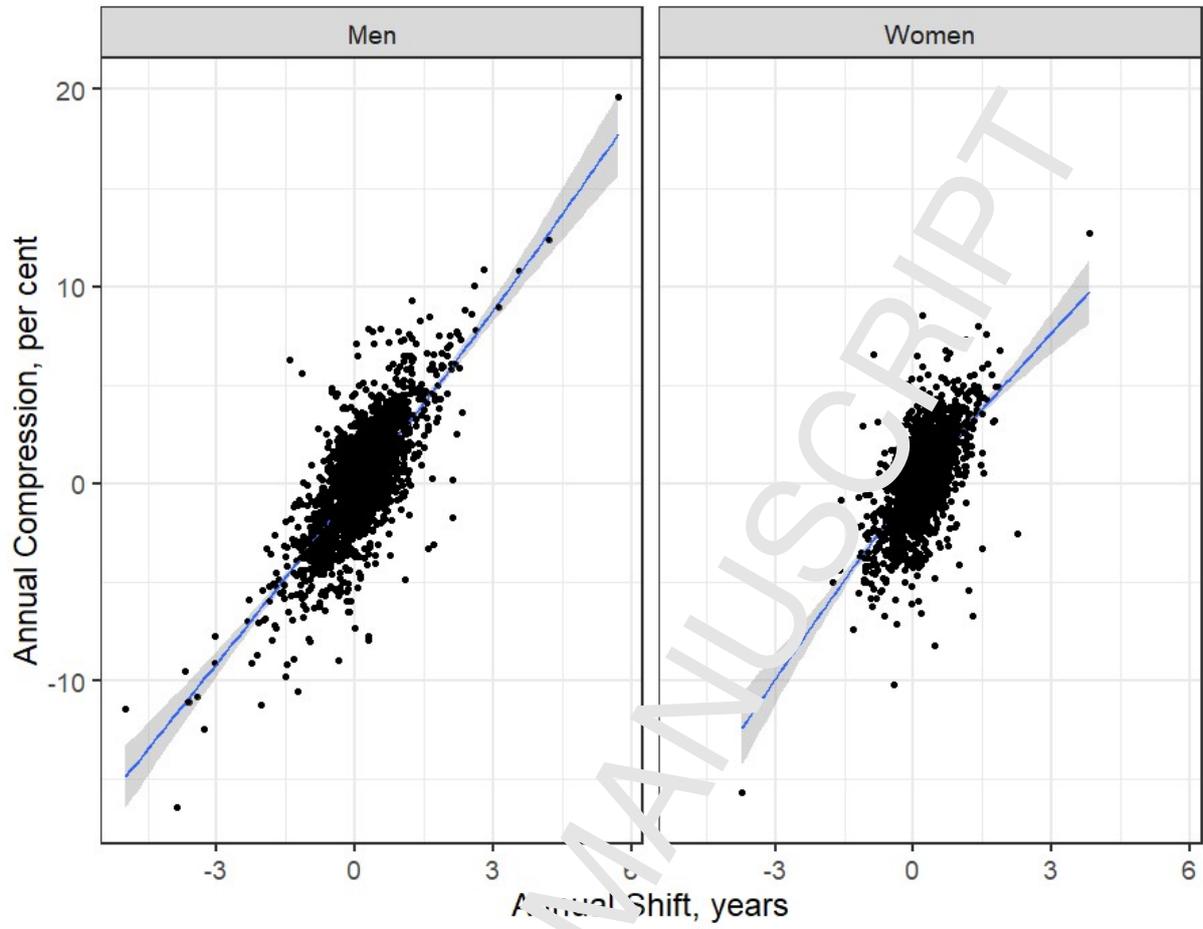


Fig. 4.

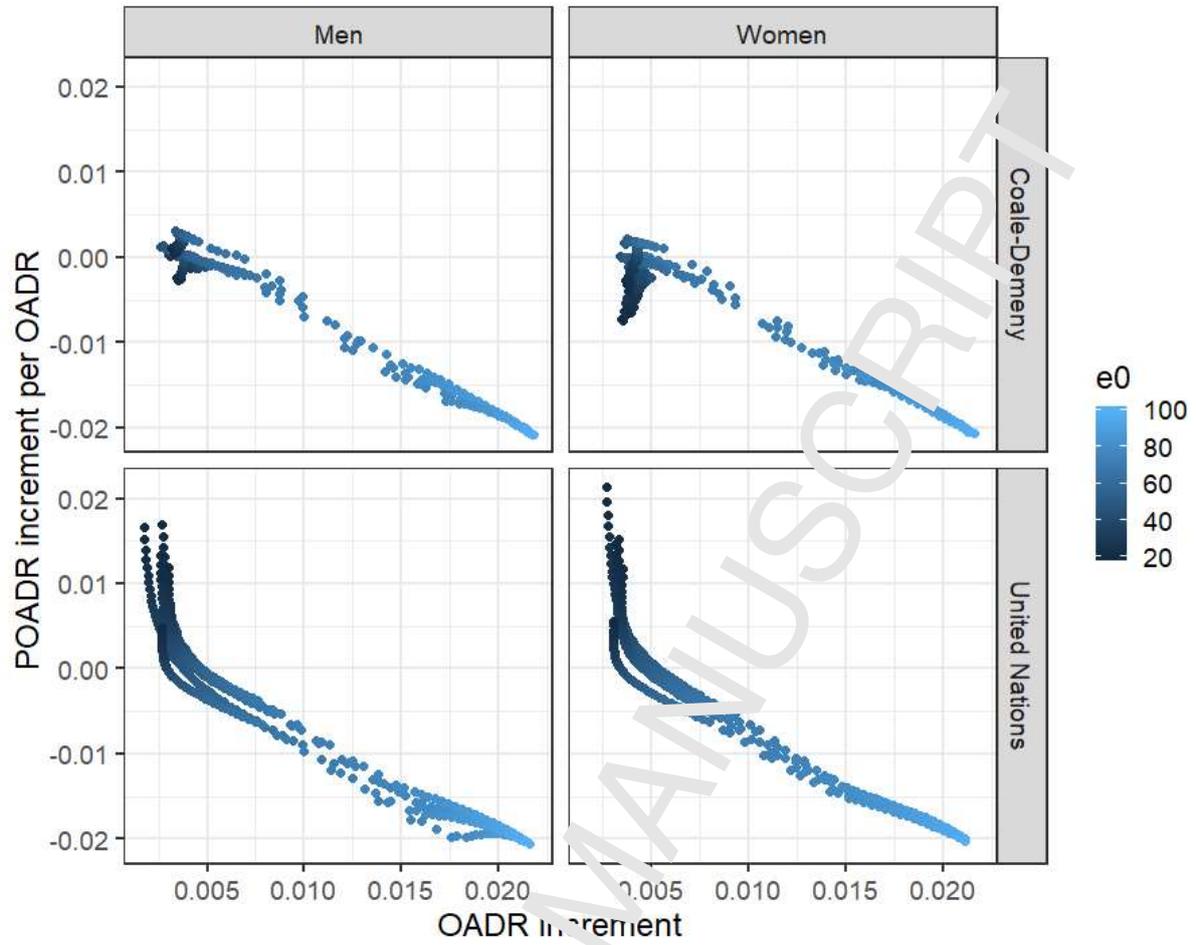


Fig. 5.

**The formal demography of prospective age:****The relationship between the old age dependency ratio and the prospective old age dependency ratio****Highlights**

- Inverse relationship between alternative responses of population ageing indicators to mortality is explained
- Mortality shift and mortality compression push ageing indicators in opposite directions
- Results of analysis of population dynamics depend crucially on whether the conventional or prospective old-age dependency ratios are used
- Mortality shift and compression model is useful in analytically studying effects of mortality change