Article from

ARCH 2017.1 Proceedings

High age mortality and frailty. Some remarks and hints for actuarial modeling [1]

Ermanno Pitacco [2]

Abstract

This paper provides some introductory remarks to critical biometric aspects underlying risk identification and risk assessment for life annuity portfolios and pension funds. On the one hand, statistical evidence shows, in many populations, a deceleration in mortality increase at very old ages, in particular a non-exponential increase in the age-pattern of mortality. On the other hand, causes of this feature of the age-pattern of mortality constitute a rather controversial issue. Nevertheless, a deceleration in the mortality increase can analytically be explained by the (reasonable) assumption of heterogeneity with respect to mortality inside a cohort, and, in particular, in terms of non-observable risk factors, which can be represented, for each individual in the population, by his/her "frailty" level.

The presence of mortality heterogeneity heavily impacts on the riskiness of a life annuity portfolio (or a pension fund), and hence should carefully be taken into account in the risk management process. In particular, appropriate parametric models can help in assessing the impact of heterogeneity among annuitants.

Keywords: Heterogeneity, Frailty, Mortality deceleration, Force of mortality, Parametric models, Longevity limits, Life annuities

[1] The research reported herein was performed in the framework of activities of the Mortality Working Group of the International Actuarial Association, http://www.actuaries.org/

[2] Dpt. DEAMS, University of Trieste (Italy), ermanno.pitacco@deams.units.it

1 Introduction

A very extensive literature, mainly developed in the last decades, focusses on mortality at old and very old ages and relevant possible causes. The related research work involves demography, actuarial sciences, gerontology, biology, biostatistics, epidemiology, etc. The following list of contributions, although largely incomplete, aims at showing the main topics addressed. We note that any classification and the related inclusion of contributions into the various categories are affected by some degree of arbitrariness. Nonetheless, a classification can help in singling out the main areas of scientific interest and research work.

The longevity limits, i.e. the maximum length of life and the modal age at death have recently been focussed, in particular, by Aarssen and de Haan (1994), Horiuchi et al. (2013), Le Bras (1976) and Thatcher (1999). However, earlier interest in these issues is witnessed in Greenwood and Irwin (1939) (see also the references therein).

The (possible) deceleration in the age-pattern of mortality at very old ages (see Sect. 2.1) and the underlying causes have recently been addressed, in particular, by Gampe (2010), Gavrilov and Gavrilova (2011, 2015), Horiuchi and Wilmoth (1998), Wilmoth (1995) and Steinsaltz and Wachter (2006). However, it is worth noting that the awareness of such a deceleration can be dated back to the first half of the 19th century; see Greenwood and Irwin (1939). For a detailed survey, the reader can refer to Olshansky (1998).

Dynamic aspects of mortality at high ages, i.e. mortality trends and related forecasts, have been dealt with by Buettner (2002) and Currie (2011).

Country-related features of high age mortality have been analyzed in particular by Bourbeau and Desjardins (2007), CMI Working Paper 85 (2015), Coale and Kisker (1990), Coelho et al. (2007), Gallop (2002), Horiuchi and Wilmoth (1998), Kannisto (1994), Lindbergson (2001), Maccheroni and Billari (1996) and Thatcher et al. (1998). It is worth noting that international comparisons as well as methodological contributions are also provided in many of the papers cited above.

While the earliest contributions to the construction of mortality tables and the definition of parametric models (i.e., "mortality laws") representing the age-pattern of mortality may be dated back to the 17th and 18th centuries (see, for example, the references in Haberman (1996), Haberman and Sibbett (1995), Hald (1987) and Pitacco (2004)), the oldest contributions focussing on biological and physiological features of the age-pattern of mortality date back to the 19th century; see, in particular, Gompertz (1825, 1860), Makeham (1867, 1890), Thiele (1871), Graf (1905), Brownlee (1919), Greenwood (1928) and Greenwood and Irwin (1939). Interesting reviews on the biodemography

of aging and mortality modeling according to biological and physiological perspectives are respectively provided by Olshansky (1998) and Yashin et al. (2000).

Parametric models for representing mortality at high ages have been addressed by Perks (1932), Beard (1959), Coale and Kisker (1990), Kannisto (1994), Thatcher (1999), Lindbergson (2001) and Saikia and Borah (2014). Details on this topic are given in Sects. 3.4 to 3.8. A state-of-art review is provided by Calduch Verdiell (2007). Doray (2008) focuses on inference problems for the logistic class of mortality laws.

General parametric mortality models are described and discussed by many textbooks in the field of life insurance mathematics and technique; see, for example, Pitacco et al. (2009) and the references therein. We also cite the detailed review provided by Forfar (2004).

The impact of heterogeneity, in particular due to non-observable risk factors, on the age pattern of mortality has been focussed by Beard (1959), Vaupel et al. (1979), Yashin and Iachine (1997) and Steinsaltz and Wachter (2006). A compact review is provided by Haberman and Olivieri (2014).

While Beard (1959) and Vaupel et al. (1979) express the non-observable heterogeneity in terms of fixed individual "frailty", due to genetic factors, other approaches to mortality heterogeneity have been proposed. In particular, the model proposed by Le Bras (1976) relies on the concept of frailty which stochastically changes with age, that is, throughout the individual life, viz because of physiological changes and environmental influences. The two approaches are compared by Thatcher (1999). Markov aging models, which generalize Le Bras's assumption, have been adopted by Su and Sherris (2012), Lin and Liu (2007), Liu and Lin (2012) and Sherris and Zhou (2014).

It is worth noting that Yashin et al. (1994) show that changing frailty models cannot be distinguished from a fixed frailty model: hence, a given observed age-pattern of mortality can be explained in (at least) two different ways, which involve two different concepts of changing probability of death with age on the individual level.

Heterogeneity with respect to mortality, frailty and the related impact on the results of a life annuity portfolio (or a pension fund) are addressed by significant research work in the actuarial field. See, in particular, Butt and Haberman (2002, 2004) and Olivieri (2006). Among the most recent contributions, Meyricke and Sherris (2013) analyze the impact of heterogeneity and frailty on the actuarial values of both standard life annuities and underwritten life annuities (i.e., "special rate" life annuities). Conversely, the impact of heterogeneity on tail risk and solvency requirements is focused by Sherris and Zhou (2014).

The remainder of the present paper is organized as follows. In Sect. 2 some

(controversial) conclusions derived from data analysis are briefly reported.

Parametric models which can be adopted to represent the age-pattern of mortality, in particular at old and very old ages, are presented in Sect. 3. The assumptions underlying some models are singled out, to check their consistency with (reasonable) features of the high age mortality. We stress that the parametric models addressed in the present paper comply with a "static" perspective, whereas trend aspects and relevant forecasting problems are not considered.

Some actuarial aspects constitute the object of Sect. 4, in terms of relation between the mortality law chosen and the consequent assessment of expected values and riskiness inherent in a life annuity portfolio or a pension plan.

Sect. 5 concludes the paper with some final remarks and an outlook for future research.

We stress that, from a scientific perspective, no original contribution is given by this paper. It can be considered as a "primer", which just aims to provide some guidelines for future research work in the field of actuarial aspects of mortality at high ages.

2 The age-pattern of mortality at high ages: statistical data and controversial issues

We first focus on possible features of the mortality pattern at high ages. Then, we briefly describe some statistical studies and the related achievements, concerning high age mortality.

2.1 The "deceleration" in late-life mortality

In several traditional mortality models (e.g. the mortality laws proposed by Gompertz, Makeham and Thiele), it is assumed that the force of mortality (or hazard rate) increases exponentially, at least definitely, and hence at a constant rate.

Conversely, a "deceleration" phenomenon occurs when the force of mortality eventually increases at a decreasing rate. Generally speaking. i.e. not restricting the context to the mortality of humans, the following mortality profiles, which decelerate at high ages, can be recognized (see Fig. 1).

- The force of mortality increases at a decreasing rate, for example because it eventually follows a linear profile (or approaches a slant linear asymptote).

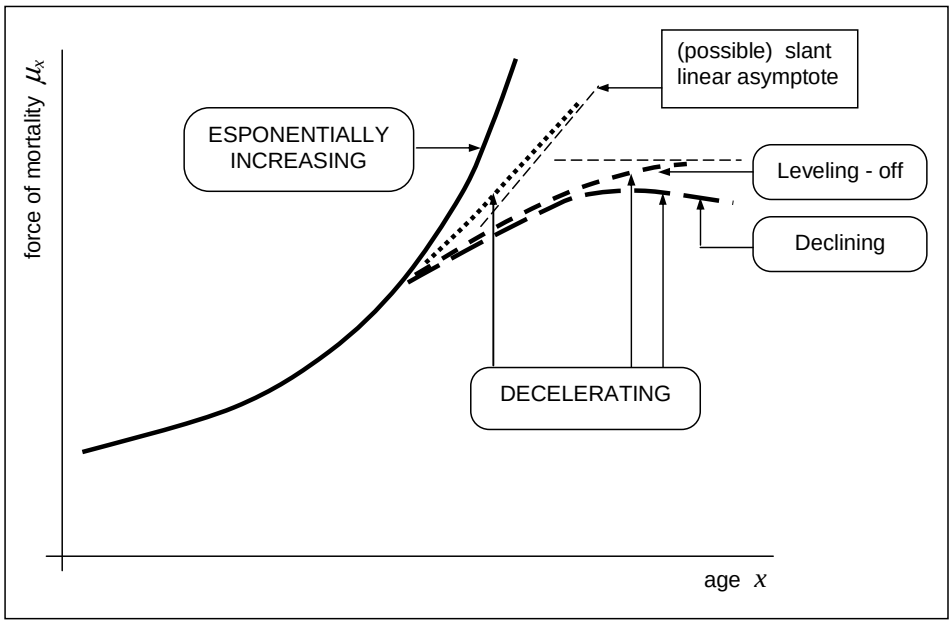

Figure 1: Force of mortality: exponentially increasing vs decelerating

- The force of mortality stops increasing (or tends to a horizontal asymptote), and then proceeds at a constant rate (or approximately constant rate). Hence, the rate of increase is (or tends to be) equal to zero. In this case we say that a mortality "leveling-off" occurs (or a mortality "plateau" is reached).

- In some species, the force of mortality can eventually decline at old ages, and this obviously results in a negative rate of increase (the meaning of "old" being of course related to the species addressed).

2.2 The archive on population data on aging

Thatcher (1999) focused on number of deaths at age 80 and over, observed in 30 countries, since 1960 (at least). The relevant database is currently held at the University of Odense and the Max Planck Institute in Rostock.

The age-pattern of mortality resulting from the data appears to be closer to the mortality profile given by a logistic model, than to that given by the Gompertz model or the Weibull model (see Weibull (1951)).

Three explanations have been suggested, which formally correspond to different aging models:

(1) the fixed individual frailty model, originally proposed by Beard (1959) (see Sect. 3.9);

(2) the stochastic process model, according to which individuals move through health classes, originally proposed by Le Bras (1976);

(3) the biological theory of aging, according to which the deterioration process may not continue indefinitely, so that the force of mortality first increases with age but then reaches a plateau, or even (for some species) shows a fall (as noted by Thatcher (1999), this theory is controversial).

2.3 Mortality data of Sweden (1861 - 1990) and Japan (1951 - 1990)

Horiuchi and Wilmoth (1998) focussed on mortality experience in Sweden and Japan. In particular, mortality data by cause of death (COD) have been analyzed and late-life mortality deceleration has been observed.

Two main causes of deceleration have been proposed, each one formally underpinned by a specific hypothesis:

(1) population heterogeneity hypothesis, that is, a demographic explanation which relies on the population composition; according to such hypothesis, more frail individuals tend to die earlier;

(2) individual-risk hypothesis, that is, a gerontological explanation, in terms of senescent process; in this context, mortality deceleration can be explained by less energy expenditure at high ages, more protected environment, etc.

The heterogeneity explored by Horiuchi and Wilmoth (1998) and the relevant results support the population heterogeneity hypothesis.

Further, a new aspect has been singled out: the timing of deceleration, i.e. the age at which the deceleration phase starts. Predictions about timing of mortality deceleration can be made according to CODs. Three predictions have been proposed:

(a) deceleration of mortality due to "selective" CODs tends to occur earlier than deceleration due to other causes; a COD is considered selective when the risk of death for that particular cause differs among individuals of the same age;

(b) timing of deceleration of mortality due to most of CODs depends on individual vulnerability to diseases which constitute CODs;

(c) the overall deceleration (that is, relative to mortality due to all CODs aggregated) occurs in late-life.

2.4 Social Security Administration Death Master File (DMF)

Gavrilov and Gavrilova (2011) focus on the numbers of deaths in the United States, over age 85. The method of extinct generations has been adopted in the analysis.

According to the Authors, three critical aspects can cause deceleration in the estimated age-pattern of mortality:

(1) mixing different cohorts (as usual when working with census data), which leads to heterogeneity among cohorts belonging to the population;

(2) non-appropriate standard assumptions in the estimation procedures (e.g. the assumption of constant mortality rate over one-year age intervals);

(3) exaggerated recorded age at death.

From the DMF analysis, the following features can be recognized:

(a) the mortality deceleration is almost negligible up to age 106;

(b) the deceleration is probably caused by poor quality data;

(c) better quality data (in particular for more recent cohorts) show a negligible deceleration, so that the Gompertz law provides a correct representation of the age-pattern of mortality.

2.5 United Kingdom: ONS and CMI data

A detailed analysis of mortality experience is presented and discussed in CMI Working Paper 85 (2015). In particular, population data provided by the Office for National Statistics (ONS) and market data collected and elaborated by the Continuous Mortality Investigation of the Institute and Faculty of Actuaries are analyzed with major focus on high age mortality.

In the report, critical features of statistical data are singled out, which might lead to deceleration in the age-pattern of mortality, in particular:

- unreported deaths;
- misstatement of age at death.

A what-if-analysis has also been performed, by assuming the presence of heterogeneity, that is, the presence of groups with different Gompertz age-patterns of mortality. Numerical results show that heterogeneity implies the mortality deceleration phenomenon.

2.6 Some remarks

From the results briefly described in Sects. 2.2 to 2.5, it clearly emerges that both the presence of mortality deceleration as well as the causes of possible deceleration constitute controversial issues. Indeed, conflicting results obtained from statistical data do not allow to derive univocal conclusions.

As regards possible causes of deceleration, Preston et al. (1999) investigate the influence of age misreporting on measures of mortality at older ages. Five types of mortality estimates are considered, and the effect of such misreporting on mortality estimates is identified. In particular, the Authors show that the downward bias in death rates at older ages may not be solely attributable to age exaggeration.

Among the most recent contributions on problems originated by unreliable population data, we cite Cairns et al. (2016). A framework is developed, which allows to assess data reliability and to identify anomalies. The Authors also propose methods that can be used to improve estimates of population exposures.

Although data quality can constitute an obstacle in deriving univocal conclusions, on the one hand the deceleration phenomenon emerges from numerous mortality experiences and, on the other, the presence of deceleration inside a heterogeneous cohort can also be explained in formal terms. In the next sections (and, in particular, in Sects. 3.4 to 3.9) we will focus on this aspect.

3 Graduation via mortality laws

The term "graduation" denotes an adjustment procedure applied to a set of estimated quantities, in order to obtain adjusted quantities which are close to a reasonable pattern and, in particular, do not exhibit an erratic behavior. We note that previous experience and intuition usually suggest a smooth progression.

In actuarial science (and demography), graduation procedures are typically applied to raw mortality rates which result from statistical observation. Graduated series of period mortality rates should exhibit a progressive change over a series of ages, without sudden and/or huge jumps, which cannot be explained by intuition or supported by past experience.

3.1 Mortality laws vs polynomial and spline graduation

Various approaches to graduation can be adopted. In particular, two broad categories can be recognized, i.e., parametric approaches, involving the use of mortality laws, and non-parametric approaches.

According to a parametric approach, a functional form is chosen, and the relevant parameters are estimated in order to find the parameter values which provide the best fit to the observed data, e.g., to mortality rates. Various fitting criteria can be adopted for parameter estimation, for example maximum likelihood, based on a Generalized Linear Models formulation.

The choice of a particular functional form is avoided when a non-parametric graduation method is adopted. Important methods in this category are: weighted moving average methods, kernel methods, the Whittaker-Henderson model, methods based on polynomials and spline functions.

In particular, polynomial and splines graduations only aim at fitting and smoothing raw mortality rates. These graduation approaches are also adopted to extrapolate the age pattern of mortality beyond ages for which reliable observations are available.

Conversely, biological, physiological and possibly behavioral assumptions underpin many mortality laws, or components of mortality laws. As noted by Olshansky and Carnes (1997), linking basic biology of humans to life table functions was first proposed by Gompertz (1825), and later analyzed by Brownlee (1919).

3.2 The senescence process according to Gompertz

As is well known, a new era for the actuarial science started in 1825 with the law proposed by Benjamin Gompertz (see Gompertz (1825)), the pioneer of a new approach to survival modeling. Gompertz's ideas can be properly expressed in terms of what we now call force of mortality (or instantaneous intensity, or hazard rate). Gompertz's law constitutes one of the most influential proposals in the early times of survival modeling. Actually, many contributions in the field of mortality laws, throughout the latter half of the 19th century, generalize Gompertz's law or, anyhow, proceed from

Gompertz's ideas. Remarkable examples are given by the laws proposed by Makeham (1867) and Thiele (1871) (see Sect. 3.3).

Let μ_x denote the force of mortality at age x, defined as follows:

$$\mu_x = \lim_{t \to 0} \frac{\mathbb{P}[T_x \leq t]}{t} \tag{1}$$

where T_x denotes the random remaining lifetime of an individual age x. The senescence assumption is expressed, in the Gompertz model, by:

$$\Delta\mu_x = \beta\, \mu_x\, \Delta x + o(\Delta x) \tag{2}$$

with $\beta > 0$. Hence, given the age interval $(x, \Delta x)$, the higher is the initial value μ_x, the higher is the increment $\Delta\mu_x$. This assumption leads to the differential equation:

$$\frac{\mathrm{d}\mu_x}{\mathrm{d}x} = \beta\, \mu_x \tag{3}$$

Finally, from Eq. (3) we obtain the Gompertz law:

$$\mu_x = \alpha\, \mathrm{e}^{\beta\, x} \tag{4}$$

with $\alpha > 0$.

As noted by Olshansky and Carnes (1997), the idea that "one simple function" (like the one given by Eq. (4)) cannot represent the age-pattern of mortality over the whole life span was first expressed by Gompertz (1860), who proposed four age intervals, i.e. (in years):

$$(0, 1),\ (1, 20),\ (20, 60),\ (60, 100)$$

which should be separately considered for modeling purposes.

This idea was then implemented in particular by Thiele (1871) and Heligman and Pollard (1980), although adopting a different approach to single out interval-specific components of the age-pattern of mortality.

3.3 Generalizing the Gompertz model

The Gompertz exponential function, i.e. the term $\alpha\, \mathrm{e}^{\beta\, x}$, enters as the senescent component of the force of mortality in various parametric models.

The first Makeham law (see Makeham (1867)) generalizes the Gompertz model, by assuming:

$$\mu_x = \gamma + \alpha\, \mathrm{e}^{\beta\, x} \tag{5}$$

where the term $\gamma > 0$ represents age-independent mortality, e.g. because of accidents. The second Makeham law (see Makeham (1890)) also includes a linear term:

$$\mu_x = \gamma + \rho\, x + \alpha\, \mathrm{e}^{\beta\, x} \tag{6}$$

and hence constitutes a further generalization of the Gompertz law.

In 1867, W. Lazarus proposed another generalization (see Graf (1905)) by adding to the Makeham law a negative exponential term, which decreases as the age increases and can hence represent the infant mortality:

$$\mu_x = \varphi\, \mathrm{e}^{-\psi\, x} + \gamma + \alpha\, \mathrm{e}^{\beta\, x}; \quad \varphi, \psi > 0 \tag{7}$$

The law proposed by Thiele (1871) aims at representing the age-pattern of mortality over the whole life span:

$$\mu_x = \varphi\, \mathrm{e}^{-\psi\, x} + \gamma\, \mathrm{e}^{-\tau (x-\xi)^2} + \alpha\, \mathrm{e}^{\beta\, x}; \quad \varphi, \psi, \gamma, \tau, \xi > 0 \tag{8}$$

We note that the second term in the right-hand side of Eq. (8), which has a "Gaussian" shape, can quantify the mortality hump (mainly due to accidents) at young-adult ages.

Other generalizations of the Gompertz law have been proposed. A significant example is given by the GM class of models (namely, the Gompertz-Makeham class of models), proposed by Forfar, McCutcheon and Wilkie (see Forfar et al. (1988)). The GM structure is as follows:

$$\mu_x = \sum_{i=0}^{r-1} a_i\, x^i + \exp\left[\sum_{j=0}^{s-1} b_j\, x^j\right] \tag{9}$$

with the proviso that when $r = 0$ the polynomial term is absent, and when $s = 0$ the exponential term is absent. The general model represented by (9) is usually labelled as GM(r, s). Note that, in particular, GM$(0, 2)$ denotes the Gompertz law, GM$(1, 2)$ the first Makeham law and GM$(2, 2)$ the second Makeham law. Models used by the Continuous Mortality Investigation Bureau in the UK to graduate the force of mortality μ_x are of the GM(r, s) type. In particular, models GM$(0, 2)$, GM$(2, 2)$ and GM$(1, 3)$ have widely been adopted. See, for example, **?**.

3.4 Deceleration in the senescence process

The mortality laws so far considered assume an exponential growth in the age-pattern of mortality because of senescence (see, for example, the Gompertz hypothesis expressed by Eq. (2)). We note that the exponential growth

implies a constant rate of increase in senescent mortality, which, in its turn, leads to a constant rate of increase in the Gompertz model, that only allows for senescent mortality, and an approximately constant rate of increase in the models which generalize the Gompertz law also including other mortality components.

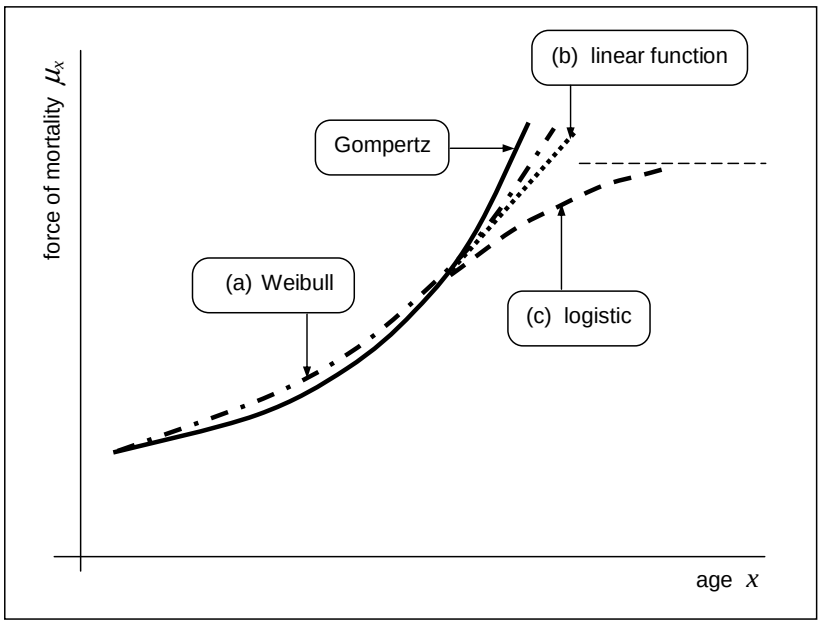

Figure 2: Exponential vs non-exponential old-age pattern of mortality

A non-exponential increase in the age-pattern of mortality can be represented in several ways. We consider the following modeling choices (see Fig. 2).

(a) An example of non-exponential force of mortality (over the whole life span), is given by the model proposed by Weibull (1951):

$$\mu_x = A\, x^B. \tag{10}$$

(b) A non-exponential increase at very old ages (and hence a non-constant rate of increase) can be obtained by, e.g., a linear or an asymptotically linear component of the force of mortality; see Sect. 3.6.

(c) A mortality leveling-off at very old ages can be achieved by adopting logistic models; see Sect. 3.5.

We note that modeling choices (b) and (c) imply a deceleration in the age-pattern of mortality, with respect to the Gompertz or Gompertz-related exponential models (see Sect. 2.1).

3.5 Logistic-type models

Several models have been proposed, that are strictly related each other and share the purpose of representing a mortality leveling-off (see Sect. 2.1). In formal terms, the common feature of these models consists in a horizontal asymptote of the force of mortality.

In 1932 W. Perks proposed two mortality laws; see Perks (1932). The first Perks law is as follows:

$$\mu_x = \frac{\alpha\, \mathrm{e}^{\beta x} + \gamma}{\delta\, \mathrm{e}^{\beta x} + 1} \tag{11}$$

while the second Perks law has the following, more general, structure:

$$\mu_x = \frac{\alpha\, \mathrm{e}^{\beta x} + \gamma}{\delta\, \mathrm{e}^{\beta x} + \epsilon\, \mathrm{e}^{-\beta x} + 1} \tag{12}$$

The following law was proposed by Beard (1959):

$$\mu_x = \frac{\alpha\, \mathrm{e}^{\beta x}}{\delta\, \mathrm{e}^{\beta x} + 1} \tag{13}$$

We note that (13) can be obtained from the first Perks law (11) by setting $\gamma = 0$.

Among the most recent contributions, we first recall the law proposed by Kannisto (1994):

$$\mu_x = \frac{\alpha\, \mathrm{e}^{\beta x}}{\alpha\, \mathrm{e}^{\beta x} + 1} \tag{14}$$

which can be obtained by setting $\gamma = 0$ and $\delta = \alpha$ in the first Perks law.

Thatcher (1999) proposed the following expression for the force of mortality:

$$\mu_x = \frac{\nu\, \alpha\, \mathrm{e}^{\beta x}}{\alpha\, \mathrm{e}^{\beta x} + 1} + \kappa \tag{15}$$

The simplified version of (15), used in particular for studying long-term trends and forecasting mortality at very old ages, has $\nu = 1$ and hence only three parameters, namely α, β and κ:

$$\mu_x = \frac{\alpha\, \mathrm{e}^{\beta x}}{\alpha\, \mathrm{e}^{\beta x} + 1} + \kappa \tag{16}$$

3.6 Linear behavior at very old ages

A late-life deceleration phenomenon can be the result of a linear (or asymptotically linear) behavior of the force of mortality.

Lindbergson (2001) proposed the following model:

$$\mu_x = \begin{cases} \gamma + \alpha\, \mathrm{e}^{\beta\, x} & \text{if } \; x \leq w \\ \gamma + \alpha\, \mathrm{e}^{\beta\, w} + \vartheta\, (x - w) & \text{if } \; x > w \end{cases} \tag{17}$$

where w represents an old age. We note that (17) is a modified version of the first Makeham law (5), obtained by simply replacing the exponential growth with a linear growth at very old ages.

The basic structure of Thiele's law (8) can also be found in the (first) law proposed by Heligman and Pollard (1980) to represent the mortality odds:

$$\frac{q_x}{1 - q_x} = A^{(x+B)^C} + D\, \mathrm{e}^{-E(\ln x - \ln F)^2} + G\, H^x \tag{18}$$

An interesting property of this law emerges in terms of the related force of mortality (see Thatcher (1999), Buettner (2002)). At old ages, assume the approximation:

$$\frac{q_x}{1 - q_x} \approx G\, H^x = a\, \mathrm{e}^{bx} \tag{19}$$

Hence:

$$\ln q_x - \ln(1 - q_x) \approx \ln a + b\, x \tag{20}$$

Accepting the approximation

$$\mu_x \approx -\ln(1 - q_x) \tag{21}$$

we finally find:

$$\lim_{x \to +\infty} \big[\mu_x - (\ln a + b\, x)\big] = 0 \tag{22}$$

where $\ln a + b\, x$ represents a slant linear asymptote. Then, the result is an asymptotically linear behavior of the force of mortality.

3.7 The Coale-Kisker assumption

The model proposed by Coale and Kisker (1990) relies on the exponential age-specific rate of change of the central mortality rates m_x:

$$k_x = \ln \frac{m_x}{m_{x-1}} \tag{23}$$

(see also Buettner (2002) and Wilmoth (1995)). A linear profile of k_x beyond age 85 is assumed, that is:

$$k_x = k_{85} - (x - 85)\, s \tag{24}$$

The parameter s is determined by assuming that k_{85} is calculated from empirical data, whereas a predetermined value is assigned to the mortality rate m_{110}. For given values of k_x, $x = 85, 86, ..., 110$, we obtain from (23):

$$m_x = m_{85} \exp\left(\sum_{h=86}^{x} k_h\right) \tag{25}$$

From Eq. (25) it follows that the Coale-Kisker model implies an exponential-quadratic function for central death rates at the relevant ages, i.e.:

$$m_x = \exp(a\, x^2 + b\, x + c) \tag{26}$$

The model can be used to extrapolate the age pattern of mortality beyond ages for which reliable observations are available.

3.8 Mortality profile at very old ages according to parametric models: a summary and a further step

As regards the age pattern of mortality at old and very old ages, the parametric models we have presented in Sects. 3.2 to 3.7 offer the following modeling possibilities.

(a) An exponential growth, and hence a constant rate of increase, of the force of mortality is provided by the Gompertz law and by the "Gompertz term" in the Makeham laws, the Lazarus law and the Thiele law.

(b) A decreasing rate of increase in the force of mortality and, in particular, a mortality leveling-off are the features of mortality laws belonging to the logistic class.

(c) A decreasing rate of increase in the force of mortality is also implied by the presence of a linear term (see the Lindbergson model), or a linear asymptotic behavior of the force of mortality (see the Heligman-Pollard law).

(d) A different pattern of old-age central rates of mortality is provided by the Coale-Kisker assumptions.

An interesting link between features (a) and (b) can be established thanks to an appropriate interpretation of the force of mortality. More precisely, if we reasonably assume that a population (or an insurance portfolio) consists of individuals with different age-patterns of mortality, namely if a certain degree of heterogeneity in the population is allowed for, then we can distinguish between:

- the individual force of mortality, that is, a force of mortality with individual-specific (although unknown) parameters;
- the average force of mortality in the population.

In Sect. 3.9 we focus on a particular setting which aims at describing the heterogeneity in a population. Then, a particular link between features (a) and (b) will briefly be described.

3.9 Heterogeneity, frailty and mortality deceleration

Heterogeneity of a population in respect of mortality can be explained by differences among the individuals; some of these are observable (for example: age, gender, etc.), while others (for example: the individual's attitude towards health, some congenital personal characteristics) are unobservable (see, for example, Pitacco et al. (2009)).

When allowing for unobservable heterogeneity factors, two approaches can be adopted:

(1) A discrete approach, according to which heterogeneity is expressed through a (finite) mixture of appropriate functions (e.g., forces of mortality, survival functions, life tables in an age-discrete setting, etc.).

(2) A continuous approach, based on a non-negative real-valued variable, called the "frailty", whose role is to include all the unobservable factors influencing the individual mortality.

Approach (2) was proposed by Vaupel et al. (1979). In their seminal paper, they extend the earlier work of Beard (see Beard (1959, 1971)) and define the frailty as a non-negative quantity whose level expresses the unobservable risk factors affecting individual mortality. The underlying idea is that those people with a higher frailty die on average earlier than others. In what follows, we deal with this approach only.

With reference to a cohort (defined at age 0 and closed to new entrants), we consider people current age x. They represent a heterogeneous group,

because of unobservable factors. Let us assume that, for any individual, such factors are summarized by a non-negative variable, viz the frailty. The specific value of the frailty of each individual does not change over time, but remains unknown. On the contrary, because of deaths, the distribution of people in respect of frailty does change with age, given that people with low frailty are expected to live longer. We denote by Z_x the random frailty at age x, for which a continuous probability distribution with probability density function (pdf) $g_x(z)$ is assumed. It must be mentioned that the hypothesis of unvarying individual frailty, which is reasonable when thinking of genetic aspects, seems weak when referring to environmental factors, which may change over time then affecting the risk of death; however, there is empirical evidence which validates quite satisfactorily the above assumption.

Let $\mu_x(z)$ denote the (conditional) force of mortality of an individual current age x with frailty level z, that is:

$$\mu_x(z) = \lim_{t \to 0} \frac{\mathbb{P}[T_x \leq t | Z_x = z]}{t} \tag{27}$$

The individual (conditional) force of mortality can be linked, in various ways, to a "standard" force of mortality. In Vaupel et al. (1979) a multiplicative link has been proposed:

$$\mu_x(z) = z\, \mu_x \tag{28}$$

where $\mu_x = \mu_x(1)$ represents the force of mortality for an individual with $z = 1$, and is considered as the standard force of mortality.

It can be proved (see, for example, Pitacco et al. (2009)) that, given the pdf of the initial distribution of the frailty, $g_0(z)$, and the force of mortality $\mu_x(z)$ for $x, z > 0$, we can determine:

- the pdf of the frailty at age x, $g_x(z)$, for $x > 0$;
- the average force of mortality in the cohort:

$$\bar{\mu}_x = \int_0^{+\infty} \mu_x(z)\, g_x(z)\, \mathrm{d}z \tag{29}$$

which, according to the multiplicative link, is given by:

$$\bar{\mu}_x = \mu_x \int_0^{+\infty} z\, g_x(z)\, \mathrm{d}z = \mu_x\, \bar{z}_x \tag{30}$$

where $\bar{z}_x$ is the expected frailty at age x.

In order to further progress in analytical terms (and to find significant numerical results), some choices are needed, in particular as regards:

- the pdf of the frailty at a given age, e.g. age 0 and hence $g_0(z)$;
- the mortality law, that is, a specific parametric model for the standard force of mortality, μ_x.

We consider the model proposed by Beard (1959); see also Vaupel et al. (1979). Hence:

(1) the multiplicative model is adopted to link the frailty-specific force of mortality to the standard one; see Eq. (28);

(2) the probability distribution of the frailty is described by a Gamma with given parameters, Gamma(δ, θ);

(3) the Gompertz law or the Makeham law describes the standard mortality; in what follows, we adopt the Gompertz law $\mu_x = \alpha\, \mathrm{e}^{\beta\, x}$.

We then find, for the average force of mortality in the cohort:

$$\bar{\mu}_x = \frac{\alpha'\, \mathrm{e}^{\beta x}}{\delta'\, \mathrm{e}^{\beta x} + 1} \tag{31}$$

Thus, the Gompertz-Gamma model leads to the first Perks law, with $\gamma = 0$, that is the Beard law (see Eqs. (11) and (13)), with parameters α', δ' depending on the parameters δ, θ of the frailty distribution.

As the models in the logistic class (see Sect. 3.5) imply deceleration in the age-pattern of mortality, we can conclude that, in the setting described above, the deceleration is a consequence of the presence of frailty in the cohort.

For a formal approach and more details, see, for example, Pitacco et al. (2009) and the references therein.

We note that various generalizations of the Gompertz-Gamma or the Makeham-Gamma model can be conceived. For example, the generalization proposed by Martinelle (1987) relies on the use of a shifted Gamma distribution for the frailty, which only takes positive values on the interval $[z^*, +\infty)$, with $z^* > 0$.

Further, it is worth noting that a logistic model for the average force of mortality, $\bar{\mu}_x$, can be the result of different settings and assumptions. An interesting example is given by the stochastic process of ageing proposed by H. Le Bras. In Le Bras (1976) a cohort, assumed homogeneous at the birth, is considered; thus, all its members are supposed to be in the same state of health. Then, people move from one state of health to another, and hence heterogeneity develops throughout the life of the cohort.

4 Actuarial aspects

Research work in the actuarial field, in particular focussing on the impact of heterogeneity and frailty on the results of life annuity business and pension funds has been quoted in Sect. 1. In this Section, we only summarize some numerical results achieved by Olivieri (2006).

Refer to a portfolio of life annuities. All the annuitants are aged $x = 65$ initially, that is at time $t = 0$. The group is closed to new entrants; death is the only cause of decrement. The same annual benefit $b = 1$ is paid to all the annuitants; we assume that the annual benefit is paid continuously along the year; 2% is the annual interest rate adopted in calculating actuarial values.

As regards mortality in the cohort, two basic alternatives are considered:

- homogeneity assumption, with force of mortality μ_x given by the Gompertz law, with $\alpha = 9.712 \times 10^{-6}$ and $\beta = 0.109$;
- heterogeneity assumption, according to the Gompertz-Gamma model with Gamma(δ, θ), and standard force of mortality (that is, with unitary frailty) $\mu_x(1) = \mu_x$.

In the case of heterogeneity, we assume $\theta = \delta$, so that at age 0 the average frailty is equal to 1 (for details, again see Pitacco et al. (2009)). We also note that:

- ▷ lower δ $\Rightarrow$ stronger heterogeneity;
- ▷ higher δ $\Rightarrow$ weaker heterogeneity;
- ▷ $\delta \to +\infty$ $\Rightarrow$ homogeneity.

In particular, $\delta = 30$ will be considered as the heterogeneity assumption in some numerical comparisons (see also Butt and Haberman (2004)).

In the following tables, $Y_t^{(j)}$ denotes the random present value, assessed at time t, of the benefits paid to the generic annuitant j (i.e., the individual liability). The relevant expected value, i.e. the actuarial value of the benefits, has been calculated according to both the assumptions, that is, homogeneity and heterogeneity, and in the latter case assuming various parameter values. Conversely, $Y_t^{(\mathrm{P}_t)}$ denotes the random present value, assessed at time t, of the benefits paid to the portfolio in force at time t, P_t (i.e. the portfolio liability), that is:

$$Y_t^{(\mathrm{P}_t)} = \sum_{j \in \mathrm{P}_t} Y_t^{(j)}$$

Table 1: Expected values of individual liabilities. Source: Olivieri (2006)

$x+t$	$\mathbb{E}[Y_t^{(j)}]^{[\text{homog}]}$	$\frac{\mathbb{E}[Y_t^{(j)}]^{[\text{heter}]}}{\mathbb{E}[Y_t^{(j)}]^{[\text{homog}]}} - 1$				
		$\delta = 1$	$\delta = 20$	$\delta = 30$	$\delta = 40$	$\delta \to +\infty$
65	14.685	15.288%	1.048%	0.698%	0.523%	0.000%
70	12.027	22.084%	1.459%	0.972%	0.728%	0.000%
75	9.505	32.750%	2.122%	1.413%	1.060%	0.000%
80	7.219	49.684%	3.226%	2.149%	1.611%	0.000%
85	5.252	76.428%	5.107%	3.402%	2.551%	0.000%
90	3.657	117.537%	8.361%	5.573%	4.179%	0.000%
95	2.440	177.114%	14.029%	9.355%	7.017%	0.000%
100	1.568	253.609%	23.906%	15.951%	11.969%	0.000%

Table 2: Coefficient of variation of liabilities for some portfolios. Source: Olivieri (2006)

size n_t	$x = 65$		$x + 10 = 75$		$x + 20 = 85$	
	[homog]	[heter]	[homog]	[heter]	[homog]	[heter]
10	12.757%	14.921%	16.528%	20.367%	20.846%	27.092%
1 000	1.276%	8.090%	1.653%	12.467%	2.085%	18.173%
10 000	0.403%	8.001%	0.523%	12.372%	0.659%	18.071%

Numerical results in Table 1 show that disregarding heterogeneity in the portfolio lead to underestimation of the actuarial values and hence, in particular, of the individual reserves.

In Table 2 values of the coefficient of variation, that is

$$\mathbb{CV}[Y_t^{(\mathrm{P}_t)}] = \frac{\sqrt{\mathbb{V}\mathrm{ar}[Y_t^{(\mathrm{P}_t)}]}}{\mathbb{E}[Y_t^{(\mathrm{P}_t)}]}$$

for various portfolio sizes are reported (as regards the calculation of the variance, the reader can refer to Olivieri (2006)). Numerical results show that disregarding heterogeneity leads to underestimation of the (relative) riskiness in the portfolio, as expressed by the coefficient of variation, and hence to underestimation of the adequacy requirements, in terms of risk margin and/or solvency capital.

Although numerical results obviously depend on the probabilistic model adopted (the Gompertz-Gamma model in the above examples) and the choice

of the relevant parameter values, "qualitative" results (like the higher riskiness in the presence of heterogeneity) reasonably hold also in other settings.

5 Concluding remarks and outlook

High-age mortality data may be scanty, even when the reference population has a significant size. Of course, scarcity and possible bad quality of data cause severe problems in estimating the high-age mortality pattern. Some (controversial) conclusions have been presented in Sect. 2.

A possible problem in estimating the age pattern of mortality is given by population heterogeneity with respect to mortality. Heterogeneity may be due to:

(a) mixing several cohorts data;

(b) heterogeneity among individuals inside a given cohort.

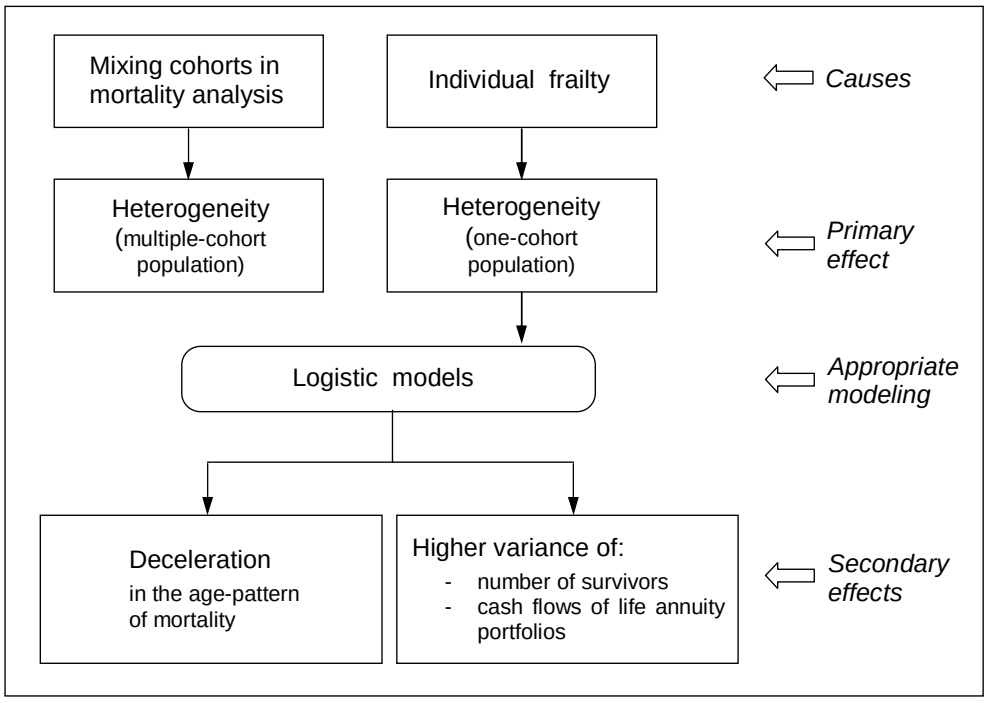

Figure 3: Heterogeneity, Deceleration, Variance

As regards point (b), assume homogeneity in the cohort in relation to observable mortality risk factors, e.g. the gender. A residual heterogeneity is

then due to non-observable risk factors. The individual specificity in relation to non-observable risk factors can be summarized in quantitative terms by the individual frailty level.

The presence of heterogeneity inside a cohort implies a deceleration of the late-life mortality in the cohort. This feature can appropriately be represented by a force of mortality belonging to the logistic class, as results, for example, from the Gompertz-Gamma model.

Conversely, from an actuarial perspective disregarding heterogeneity leads, in particular, to underestimation of the risk profile, as shown in Sect.4.

Figure 3 summarizes the above conclusions.

The complexity of the problem, especially in the actuarial context, calls for further research. From a risk management perspective, models and results presented in Sects. 3 and 4 can be placed in the "risk identification", "risk assessment" and "impact assessment" phases (see, for example, Olivieri and Pitacco (2015) and references therein). Actually:

(a) risk identification relies on the awareness of mortality differentiation because of heterogeneity, in particular due to non-observable risk factors;

(b) risk assessment aims at finding appropriate probability distribution of lifetimes, and related typical values (expected value, modal value, variance, etc.) under various mortality assumptions;

(c) impact assessment calls for appropriate models aiming to quantify liability values under various mortality assumptions.

While the basic guidelines for performing phases (a) to (c) can be found in the previous Sections, a further phase must be implemented, that is, the choice of "risk management actions", among which the product design should carefully be considered, especially in relation to life annuities and the related pricing and reserving. Indeed, an appropriate product design can mitigate risks in terms of the relevant impact on portfolio results.

References

Remark

> Where links are provided, they were active as of the time this paper was completed but may have been updated since then.

Aarssen, K. and de Haan, L. (1994). On the maximal life span of humans. *Mathematical Population Studies*, 4(4):259–281.

Beard, R. E. (1959). Note on some mathematical mortality models. In Wolstenholme, C. E. W. and Connor, M. O., editors, *CIBA Foundation Colloquia on Ageing*, volume 5, pages 302–311, Boston.

Beard, R. E. (1971). Some aspects of theories of mortality, cause of death analysis, forecasting and stochastic processes. In Brass, W., editor, *Biological aspects of demography*, pages 57–68. Taylor & Francis, London.

Bourbeau, R. and Desjardins, B. (2007). Mortality at extreme ages and data quality: The Canadian experience. In Robine, J. M., Crimmins, E. M., Horiuchi, S., and Zeng, Y., editors, *Longevity, Individual Life Duration, and the Growth of the Oldest-Old Population*. Springer.

Brownlee, J. (1919). Notes on the biology of a life-table. *Journal of the Royal Statistical Society*, 82:34–77.

Buettner, T. (2002). Approaches and experiences in projecting mortality patterns for the oldest-old. *North American Actuarial Journal*, 6(3):14–25.

Butt, Z. and Haberman, S. (2002). Application of frailty-based mortality models to insurance data. Actuarial Research Paper 142, Dept. of Actuarial Science and Statistics, City University, London.

Butt, Z. and Haberman, S. (2004). Application of frailty-based mortality models using generalized linear models. *ASTIN Bulletin*, 34(1):175–197.

Cairns, A. J. G., Blake, D., Dowd, K., and Kessler, A. R. (2016). Phantoms never die: Living with unreliable population data. *Journal of the Royal Statistical Society: Series A (Statistics in Society)*. DOI: 10.1111/rssa.12159.

Calduch Verdiell, N. (2007). *Old age mortality: A state of the art*. PhD thesis, Universitat Autònoma de Barcelona. Departament de Geografia. Memòria de Recerca del Doctorat en Demografia. Available at: `http://www.recercat.cat/handle/2072/10100`.

CMI Working Paper 85 (2015). *Initial report on the features of high age mortality.* Continuous Mortality Investigation. High Age Mortality Working Party. Available at: https://www.actuaries.org.uk/learn-and-develop/continuous-mortality-investigation/cmi-working-papers/mortality-projections/cmi-wp-85.

Coale, A. and Kisker, E. (1990). Defects in data on old age mortality in the United States: New procedures for calculating approximately accurate mortality schedules and life tables at the highest ages. *Asian and Pacific Population Forum*, 4:1–31.

Coelho, E., Magalhaes, M. G., and Bravo, J. M. (2007). Mortality and longevity projections for the oldest-old in Portugal. In *Joint Eurostat/UNECE Work Session on Demographic Projections*, Bucharest. Available at: http://www.unece.org/stats/documents/ece/ces/ge.11/2007/wp.10.e.pdf.

Currie, I. D. (2011). Modelling and forecasting the mortality of the very old. *Astin Bulletin*, 41(2):419–427.

Doray, L. G. (2008). Inference for logistic-type models for the force of mortality. Presented at the Living to 100 and Beyond Symposium. Orlando, FL. Available at: https://www.soa.org/library/monographs/retirement-systems/ living-to-100-and-beyond/2008/january/subject-toc.aspx.

Forfar, D. O. (2004). Mortality laws. In Teugels, J. L. and Sundt, B., editors, *Encyclopedia of Actuarial Science*, volume 2, pages 1139–1145. John Wiley & Sons.

Forfar, D. O., McCutcheon, J. J., and Wilkie, A. D. (1988). On graduation by mathematical formulae. *Journal of the Institute of Actuaries*, 115:1–149.

Gallop, A. P. (2002). Mortality at advanced ages in the United Kingdom. Presented at: Living to 100 Symposium - Society of Actuaries, 2002. Available at: https://www.soa.org/library/monographs/life/living-to-100/2002/mono-2002-m-li-02-1-gallop.pdf.

Gampe, J. (2010). Human mortality beyond age 110. In Maier, H., Gampe, J., Jeune, B., Vaupel, J. W., and Robine, J. M., editors, *Supercentenarians*, Demographic Research Monographs, pages 219–230. Springer.

Gavrilov, L. A. and Gavrilova, N. S. (2011). Mortality measurement at advanced ages: A study of the Social Security Administration Death Master File. *North American Actuarial Journal*, 15(3):432–447.

Gavrilov, L. A. and Gavrilova, N. S. (2015). New developments in the biodemography of aging and longevity. *Gerontology*, 61:364–371. Available at: `http://www.karger.com/Article/Pdf/369011`.

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 Royal Society (Series A)*, 115:513–583.

Gompertz, B. (1860). On one uniform law of mortality from birth to extreme old age and on the law of sickness. In *International Statistical Congress*. Reproduced in: 1871, Journal of the Institute of Actuaries, 16:329–344.

Graf, J. (1905). *Die Rechnungsgrundlagen der k.u.k priv. Assicurazioni Generali in Triest*. Assicurazioni Generali, Trieste.

Greenwood, M. (1928). "Laws" of mortality from the biological point of view. *The Journal of Hygiene*, 28(3):267–294.

Greenwood, M. and Irwin, J. O. (1939). The biostatistics of senility. *Human Biology*, 11(1):1–23.

Haberman, S. (1996). *Landmarks in the history of actuarial science (up to 1919)*. Actuarial Research Paper No. 84, Dept. of Actuarial Science and Statistics, City University, London. Available at: `http://www.cass.city.ac.uk/arc/reports/84ARC.pdf`.

Haberman, S. and Olivieri, A. (2014). Risk Classification/Life. In *Wiley StatsRef: Statistics Reference Online*. Wiley.

Haberman, S. and Sibbett, T. A., editors (1995). *History of actuarial science*, London. Pickering & Chatto.

Hald, A. (1987). On the early history of life insurance mathematics. *Scandinavian Actuarial Journal*, (1):4–18.

Heligman, L. and Pollard, J. H. (1980). The age pattern of mortality. *Journal of the Institute of Actuaries*, 107:49–80.

Horiuchi, S., Ouellette, N., Cheung, S. L. K., and Robine, J.-M. (2013). Modal age at death: Lifespan indicator in the era of longevity extension. *Vienna Yearbook of Population Research*, 11:37–69. Available at: `http://www.oeaw.ac.at/vid/publications/VYPR2013/VYPR2013.shtml`.

Horiuchi, S. and Wilmoth, J. R. (1998). Deceleration in the age pattern of mortality at older ages. *Demography*, 35(4):391–412.

Kannisto, V. (1994). *Development of Oldest-Old Mortality, 1950-1990: Evidence from 28 Developed Countries*. Odense Monograph on Population Aging, 1. Odense University Press. Available at: `http://www.demogr.mpg.de/Papers/Books/Monograph1/OldestOld.htm`.

Le Bras, H. (1976). Lois de mortalité et age limite. *Population*, 31(3):655–692.

Lin, X. S. and Liu, X. (2007). Markov aging process and phase-type law of mortality. *North American Actuarial Journal*, 11(4):92–109.

Lindbergson, M. (2001). Mortality among the elderly in Sweden 1988-97. *Scandinavian Actuarial Journal*, (1):79–94.

Liu, X. and Lin, X. S. (2012). A subordinated Markov model for stochastic mortality. *European Actuarial Journal*, 2(1):105–127.

Maccheroni, C. and Billari, F. (1996). Mortality among the very old: A model and an application to Italy. *Giornale dell'Istituto Italiano degli Attuari*, 59(1-2):39–61.

Makeham, W. M. (1867). On the law of mortality. *Journal of the Institute of Actuaries*, 8:301–310.

Makeham, W. M. (1890). On the further development of Gompertz's law. *Journal of the Institute of Actuaries*, 28:152–159,185–192,316–332.

Martinelle, S. (1987). *A generalized Perks formula for old-age mortality*. R & D report / Statistics Sweden. Statistiska Centralbyrån, Stockholm.

Meyricke, R. and Sherris, M. (2013). The determinants of mortality heterogeneity and implications for pricing annuities. *Insurance: Mathematics & Economics*, 53(2):379–387.

Olivieri, A. (2006). Heterogeneity in survival models. applications to pension and life annuities. *Belgian Actuarial Bulletin*, 6:23–39. Available at: `http://www.actuaweb.be/frameset/frameset.html`.

Olivieri, A. and Pitacco, E. (2015). *Introduction to Insurance Mathematics. Technical and Financial Features of Risk Transfers*. EAA Series. Springer, 2nd edition.

Olshansky, S. J. (1998). On the biodemography of aging: A review essay. *Population and Development Review*, 24:381–393.

Olshansky, S. J. and Carnes, B. E. (1997). Ever since Gompertz. *Demography*, 34(1):1–15.

Perks, W. (1932). On some experiments in the graduation of mortality statistics. *Journal of the Institute of Actuaries*, 63:12–57.

Pitacco, E. (2004). From Halley to "frailty": a review of survival models for actuarial calculations. *Giornale dell'Istituto Italiano degli Attuari*, 67(1-2):17–47.

Pitacco, E. (2016). High age mortality and frailty. Some remarks and hints for actuarial modeling.

Pitacco, E., Denuit, M., Haberman, S., and Olivieri, A. (2009). *Modelling Longevity Dynamics for Pensions and Annuity Business*. Oxford University Press.

Preston, S. H., Elo, I. T., and Stewart, Q. (1999). Effects of age misreporting on mortality estimates at older ages. *Population Studies*, 53:165–177. Available at: `http://www.tandfonline.com/doi/abs/10.1080/00324720308075`.

Saikia, P. and Borah, M. (2014). A comparative study of parametric models of old-age mortality. *International Journal of Science and Research*, 3(5). Available at: `www.ijsr.net`.

Sherris, M. and Zhou, Q. (2014). Model risk, mortality heterogeneity, and implications for solvency and tail risk. In Mitchell, O. S., Maurer, R., and Hammond, P. B., editors, *Recreating Sustainable Retirement: Resilience, Solvency, and Tail Risk*. Oxford University Press.

Steinsaltz, D. R. and Wachter, K. W. (2006). Understanding mortality rate deceleration and heterogeneity. *Mathematical Population Studies*, 13(1):19–37.

Su, S. and Sherris, M. (2012). Heterogeneity of Australian population mortality and implications for a viable life annuity market. *Insurance: Mathematics & Economics*, 51(2):322–332.

Thatcher, A. R. (1999). The long-term pattern of adult mortality and the highest attained age. *Journal of the Royal Statistical Society*, A, 162:5–43.

Thatcher, A. R., Kannisto, V., and Vaupel, J. W. (1998). *The Force of Mortality at Ages 80 to 120*. Odense Monograph on Population Aging, 5. Odense University Press. Available at: `http://www.demogr.mpg.de/Papers/Books/Monograph5/start.htm`.

Thiele, P. N. (1871). On a mathematical formula to express the rate of mortality throughout the whole of life. *Journal of the Institute of Actuaries*, 16:313–329.

Vaupel, J. W., Manton, K. G., and Stallard, E. (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography*, 16(3):439–454.

Weibull, W. (1951). A statistical distribution function of wide applicability. *Journal of Applied Mechanics*, 18:293–297.

Wilmoth, J. R. (1995). Are mortality rates falling at extremely high ages? An investigation based on a model proposed by Coale and Kisker. *Population Studies*, 49(2):281–295.

Yashin, A. I. and Iachine, I. A. (1997). How frailty models can be used for evaluating longevity limits: Taking advantage of an interdisciplinary approach. *Demography*, 34:31–48.

Yashin, A. I., Iachine, I. A., and Begun, A. S. (2000). Mortality modeling: a review. *Mathematical Population Studies*, 8(4):305–332.

Yashin, A. I., Vaupel, J. W., and Iachine, I. A. (1994). A duality in aging: the equivalence of mortality models based on radically different concepts. *Mechanisms of Ageing and Development*, 74:1–14.