Frailty and mortality at old ages.
Some remarks

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IAA - Mortality Working Group
Vancouver - October 2015
Agenda

- Introduction & motivation
- Old age mortality data. Some controversial issues
- Mortality laws: assumptions, relations, implications
- Actuarial aspects
- Concluding remarks & outlook
INTRODUCTION & MOTIVATION

Very extensive literature on mortality at old and very old ages, in particular focussing on:

- longevity limits (maximum length of life)
- (possible) deceleration in the age-pattern of mortality at old and/or very old ages
- dynamic aspects, i.e. mortality trends
- impact of heterogeneity

Research involving:

- demography
- actuarial sciences
- gerontology
- biology
- epidemiology
- . . . . .
Our aims:

- to look at some (controversial) conclusions derived from data analysis
- to focus on mathematical models (i.e. “laws”) which can be adopted to represent the age-pattern of mortality, in particular at old and very old ages
- to single out the assumptions underlying some models, and to check their consistency with (reasonable) features of the old age mortality
- to focus on some actuarial aspects, in terms of relation between:
  - mortality law adopted
  - consequent assessment of expected values and riskiness inherent in a life annuity portfolio or a pension plan
OLD AGE MORTALITY DATA. 
SOME CONTROVERSIAL ISSUES

Archive on population data on aging

See: Thatcher [1999]

Deaths at age 80 and over, in 30 countries, since 1960 (at least)

Database currently held at University of Odense and Max Planck Institute, Rostock

Data closer to the logistic model ($\Rightarrow$ heterogeneity), than to the Gompertz model or the Weibull model

Three explanations suggested:

1. (fixed) individual frailty
2. stochastic process model $\Rightarrow$ individuals moving through health classes
3. genetic
Old age mortality data. Some controversial issues (cont’d)

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

See: Horiuchi and Wilmoth [1998]

Mortality data by cause of death (COD)

Deceleration can be explained by:

1. *heterogeneity hypothesis* ⇒ demographic explanation, relying on population composition
   ▶ more frail individual tend to die earlier

2. *individual-risk hypothesis* ⇒ gerontological explanation, in terms of senescent process
   ▶ less energy expenditure
   ▶ more protected environment
   ▶ . . . . .

Heterogeneity explored by Horiuchi and Wilmoth ⇒ heterogeneity hypothesis supported by results
Old age mortality data. Some controversial issues (cont’d)

New aspect singled out: *timing of deceleration*, i.e. when the deceleration phase starts

Predictions about timing of deceleration can be made according to CODs

Three predictions proposed:

(a) deceleration due to “selective” CODs
    ⇒ early

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

(c) deceleration due to overall mortality (all CODs aggregated)
    ⇒ late
Social Security Administration Death Master File (DMF)

See: Gavrilov and Gavrilova [2011]

Deaths in the United States, over age 85. Method adopted: extinct generations

According to the Authors, three critical aspects can lead to deceleration:

1. mixing different cohorts ⇒ heterogeneity among cohorts
2. standard assumptions in estimate procedures non appropriate (e.g. constant mortality rate over one-year age intervals)
3. exaggerated recorded age at death

From DMF analysis:

- deceleration almost negligible up to age 106
- deceleration probably caused by poor data
- better data for more recent cohorts ⇒ less deceleration ⇒ Gompertz law appropriate
**MORTALITY LAWS: ASSUMPTIONS, RELATIONS, IMPLICATIONS**

*Mortality laws vs polynomial and spline graduation*

Biological, physiological and possibly behavioral assumptions underpin many mortality laws, or components of mortality laws.

Polynomial and splines graduations:

- only aim at fitting and smoothing
- also adopted to extrapolate age pattern of mortality beyond ages for which reliable observations are available

**Remark**

Linking basic biology of humans to life table functions first proposed by Gompertz, 1825, and Brownlee, 1919; see Olshansky and Carnes [1997]
The senescence process according to Gompertz

Senescence assumption (with \( \mu_x \) = force of mortality):

\[
\Delta \mu_x = \beta \mu_x \Delta x \tag{1}
\]

i.e. given the age interval \( \Delta x \), the higher \( \mu_x \) \( \Rightarrow \) the higher the increment \( \Delta \mu_x \)

\( \Rightarrow \) Gompertz law, 1825:

\[
\mu_x = \alpha e^{\beta x} \tag{2}
\]

Quantity \( \alpha e^{\beta x} \) as a term in other laws, e.g.

Makeham, 1867:

\[
\mu_x = \gamma + \alpha e^{\beta x} \tag{3}
\]

Thiele, 1871:

\[
\mu_x = \varphi e^{-\psi x} + \gamma e^{-\delta(x-\epsilon)^2} + \alpha e^{\beta x} \tag{4}
\]
Remark

Idea that “one simple function” (like Gompertz, or Makeham) cannot represent the age-pattern of mortality over the whole life span first expressed by Gompertz, 1872 (see Olshansky and Carnes [1997]); four age intervals (in years) proposed by Gompertz:

\[(0, 1), (1, 20), (20, 60), (60, 100)\]

Idea then implemented by Thiele, Heligman-Pollard, etc. (although referring to different age intervals)
**Mortality laws: assumptions, relations, implications (cont’d)**

*Deceleration in the senescence process*

Gompertz, Makeham, Thiele laws \( \Rightarrow \) exponential increase in the age-pattern of mortality (see Gompertz’s assumption)

Non-exponential increase can be represented by:

(a) non-exponential force of mortality over the whole life span, e.g. Weibull law, 1951

\[
\mu_x = A x^B 
\]  

(b) non-exponential increase, e.g. linear or asymptotically linear, at very old ages

(c) prevailing non-exponential increase at very old ages, e.g. via logistic models

Modeling choices (b) and (c) \( \Rightarrow \) deceleration in the age-pattern of mortality
Mortality laws: assumptions, relations, implications (cont’d)

Deceleration in the old-age mortality pattern

(a) Weibull

(b) linear function

(c) logistic

Gompertz

leveling-off (plateau)
Mortality laws: assumptions, relations, implications (cont’d)

**The logistic class**

1st Perks law, 1932 (see Perks [1932]):

\[
\mu_x = \frac{\alpha e^{\beta x} + \gamma}{\delta e^{\beta x} + 1}
\]  

(6)

2nd Perks law, 1932:

\[
\mu_x = \frac{\alpha e^{\beta x} + \gamma}{\delta e^{\beta x} + \epsilon e^{-\beta x} + 1}
\]  

(7)

Beard law, 1963 (\( \gamma = 0 \) in the 1st Perks law):

\[
\mu_x = \frac{\alpha e^{\beta x}}{\delta e^{\beta x} + 1}
\]  

(8)

Kannisto law, 1992 (\( \gamma = 0, \delta = \alpha \) in the 1st Perks law):

\[
\mu_x = \frac{\alpha e^{\beta x}}{\alpha e^{\beta x} + 1}
\]  

(9)
Mortality laws: assumptions, relations, implications (cont’d)

Linear behavior at very old ages

Lindbergson [2001] proposed:

\[
\mu_x = \begin{cases} 
\gamma + \alpha e^{\beta x} & \text{if } x \leq w \\
\gamma + \alpha e^{\beta w} + \delta (x - w) & \text{if } x > w 
\end{cases} 
\]  \tag{10}

First Heligman-Pollard law:

\[
\frac{q_x}{1 - q_x} = A(x+B)^C + D e^{-E(\ln x - \ln F)^2} + G H^x \text{ old-age} \]  \tag{11}

See Heligman and Pollard [1980]; see also Thatcher [1999], Buettner [2002].

At old ages, assume:

\[
\frac{q_x}{1 - q_x} = G H^x = a e^{bx} \]  \tag{12}
Hence:

\[ \ln q_x - \ln(1 - q_x) = \ln a + b x \]  \hspace{1cm} (13)

Accepting the approx

\[ \mu_x \approx \ln(1 - q_x) \]

we find:

\[ \lim_{x \to +\infty} \left[ \mu_x - \left( \ln a + b x \right) \right] = 0 \]  \hspace{1cm} (14)

slant linear asymptote
The Coale-Kisker assumption

See: Coale and Kisker [1990]. See also: Buettner [2002], Wilmoth [1995]

Model relying on the exponential age-specific rate of change of central death rates:

\[ k_x = \ln \frac{m_x}{m_{x-1}} \]  \hspace{1cm} (15)

Assumption: \( k_x \) linear over age 85

\[ k_x = k_{85} - (x - 85) \cdot s \]  \hspace{1cm} (16)

Parameter \( s \) calculated

▷ assuming \( k_{85} \) determined from empirical data
▷ assigning a predetermined value to \( m_{110} \)
We find from (15):

\[ m_x = m_{85} \exp \left( \sum_{h=86}^{x} k_h \right) \]  \hspace{1cm} (17)

and from (17):

\[ m_x = e^{ax^2 + bx + c} \] \hspace{1cm} (18)

The model can be used to extrapolate the age pattern of mortality beyond ages for which reliable observations are available.
Mortality laws: assumptions, relations, implications  (cont’d)

Heterogeneity and deceleration

Assume that:

- the (unobservable) heterogeneity is expressed by the individual frailty
- the individual frailty remains constant over the whole life span

For a person current age $x$ with frailty level $z$ ($z > 0$) ⇒ (conditional) force of mortality denoted by $\mu_x(z)$

Probability density function (pdf) of the frailty at age $x$: $g_x(z)$

Standard force of mortality:

$$\mu_x = \mu_x(1)$$  \hspace{1cm} (19)

Average force of mortality in the cohort:

$$\bar{\mu}_x = \int_{0}^{+\infty} \mu_x(z) g_x(z) \, dz$$  \hspace{1cm} (20)
Mortality laws: assumptions, relations, implications  \textit{(cont’d)}

Particular models rely on:

1. relation between $\mu_x(z)$ and the standard force of mortality $\mu_x$
2. pdf of the frailty at a given age, e.g. age 0: $g_0(z)$
3. mortality law $\Rightarrow$ model for $\mu_x$

We consider the model proposed by Beard [1959]; see also Vaupel et al. [1979]

1. Multiplicative model for the force of mortality:

$$\mu_x(z) = z \mu_x$$ \hspace{1cm} (21)

2. Gamma distribution
3. Gompertz law or Makeham law
Mortality laws: assumptions, relations, implications (cont’d)

In particular, combining:

1. multiplicative model
2. gamma distribution \( \text{Gamma}(\delta, \theta) \)
3. Gompertz law \( \mu_x = \alpha e^{\beta x} \)

then:

\[
\bar{\mu}_x = \frac{\alpha' e^{\beta x}}{\delta' e^{\beta x} + 1}
\]  

(22)

**Gamma - Gompertz model** \( \Rightarrow \) 1st Perks law, with \( \gamma = 0 \) (i.e. Beard law, see above), and parameters \( \alpha', \delta' \), depending on the parameters \( \delta, \theta \) of the frailty distribution

Deceleration in mortality implied by individual frailty in a cohort

For a formal approach see, for example, Pitacco et al. [2009]
Actuarial research on heterogeneity, frailty and related impacts ⇒ see, for example: Butt and Haberman [2002, 2004], Olivieri [2006], Meyricke and Sherris [2013]

Remark

Logistic model for the average force of mortality, $\bar{\mu}_x$, can be the result of other assumptions, for example the stochastic process of ageing proposed by Le Bras [1976]:

➢ cohort homogeneous at birth ⇒ all its members were in the same state of health
➢ people move from one state of health to another ⇒ heterogeneity then develops during life

See also: Thatcher et al. [1998], Thatcher [1999]
ACTUARIAL ASPECTS

Refer to a portfolio of life annuities, initial age 65

Some numerical results from [Olivieri, 2006]

Assumptions:

- Standard force of mortality (frailty = 1): Gompertz, with \( \alpha = 9.712 \times 10^{-6}, \beta = 0.109 \)

- Homogeneity assumption \( \Rightarrow \) force of mortality \( \mu_x \)

- Heterogeneity assumption \( \Rightarrow \) Gamma - Gompertz model with \( \text{Gamma}(\delta, \theta) \), and \( \mu_x(1) = \mu_x \)

  - \( \theta = \delta \Rightarrow \) at age 0, average frailty = 1
    - low \( \delta \Rightarrow \) strong heterogeneity
    - high \( \delta \Rightarrow \) weak heterogeneity
    - \( \delta \to +\infty \Rightarrow \) homogeneity
    - \( \delta = 30 \) as heterogeneity assumption in some comparisons

- Interest rate 2%
Table 1 - Expected values of individual liabilities

\[ \mathbb{E}(Y^{(j)}_{t}[\text{homog}]) \]

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

\[ \mathbb{E}[Y^{(j)}_{t}[\text{heter}]] = \frac{\mathbb{E}(Y^{(j)}_{t}[\text{heter}])}{\mathbb{E}(Y^{(j)}_{t}[\text{homog}])} - 1 \]

Table 1 - Expected values of individual liabilities

\[ \mathbb{E}[Y^{(j)}_{t}[\text{heter}]] = \text{expected present value at time } t \text{ of individual liabilities for generic annuitant } j \]
### Table 2 - Coefficient of variation of liabilities for some portfolios

<table>
<thead>
<tr>
<th>size ( n_t )</th>
<th>( x = 65 )</th>
<th>( x + 10 = 75 )</th>
<th>( x + 20 = 85 )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>[homog]</td>
<td>[heter]</td>
<td>[homog]</td>
</tr>
<tr>
<td>10</td>
<td>12.757%</td>
<td>14.921%</td>
<td>16.528%</td>
</tr>
<tr>
<td>1000</td>
<td>1.276%</td>
<td>8.090%</td>
<td>1.653%</td>
</tr>
<tr>
<td>10000</td>
<td>0.403%</td>
<td>8.001%</td>
<td>0.523%</td>
</tr>
</tbody>
</table>

From numerical results

- Table 1: disregarding heterogeneity \( \Rightarrow \) underestimation of the expected present values (and hence of the reserves)
- Table 2: disregarding heterogeneity \( \Rightarrow \) underestimation of the (relative) riskiness in the portfolio (and hence of capital requirements)
CONCLUDING REMARKS & OUTLOOK

Various causes of wrong estimation of the age pattern of mortality
A possible cause: heterogeneity, due to:

(a) mixing several cohorts data
(b) heterogeneity among individuals in one cohort, in particular individual frailty

From an actuarial perspective: disregarding (b) ⇒ underestimation of

- expected values (⇒ pricing, reserving)
- risk (⇒ risk margin, capital allocation)
Concluding remarks & outlook (cont’d)

- Individual frailty
- Mixing cohorts in mortality analysis

Heterogeneity (one-cohort population)
Heterogeneity (multiple-cohort population)

Logistic models

Deceleration in the age-pattern of mortality

Higher volatility in:
- number of survivors
- cash flows of life annuity portfolios

Causes
Primary effect
Appropriate modeling
Secondary effects

Heterogeneity, Deceleration, Volatility
Further steps (in collaboration with Annamaria Olivieri, University of Parma)

- **Risk assessment** ⇒ probability distribution of lifetimes, and related typical values (expected value, modal value, variance, etc.) under various mortality assumptions
- **Impact assessment** ⇒ DCF model to quantify liability values under various mortality assumptions
References

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


