# Modeling Middle Mortality: New Insights into Sex specific Patterns of Human Mortality

Oliver Wisser and Jim W. Vaupel

Max Planck Institute for Demographic Research, Rostock, Germany

### Abstract

A great literature gap exists between the frequency of models dealing with infant and senescent mortality and models that try to capture the middle part of mortality in human life course. Diminishing this gap we define midlife mortality  $\mu_m(X)$  as the difference between a Gamma-Gompertz model for old ages, and an exponential decay model for young age mortality. By modeling the ratio of  $\mu_m(X)$  and the total mortality  $\mu(X)$  in the next step we provide a powerful tool (relative Middle Mortality (rMM)) for describing differences in Middle Mortality in time due to sex and region. Applying this tool to several countries in the "Human Mortality Database" we uncover some interesting patterns: The rMM was nearly the same and high for both sexes until the Second World War when it started to decrease. However, since1960s it follows a positive trend for both sexes, but higher and parallel shifted for men.

## Introduction and Model

Middle mortality (MM) can be defined as that part of mortality which is due to a complex interaction between behavioral and environmental risk. It could be seen as extrinsic risk of dying and is often expressed as Makeham's c, but here suggested to have a more complex form. Senescent mortality is a complex interaction between MM and physiological ageing processes (ageing is here defined as intrinsic risk of dying). Although we are not yet sure about the law of this interaction, we provide a method to isolate the extrinsic part of mortality by assuming a dominance of the intrinsic part in high ages.

An example: When the ageing process is highly advanced, people have a high risk to die from falling of a ladder not necessarily because of the event (extrinsic risk), but because of their morbidity (intrinsic risk). This leads to the assumption that the

intrinsic risk is extremely high, whereas extrinsic risk can be mostly ignored. Moreover, the intrinsic risk of dying follows the Gompertz law of mortality (1825) which is expanded by a frailty term z following a  $\gamma$ -distribution (Vaupel et al. 1979). By fitting a  $\gamma$ -Gompertz model for high ages and extrapolating this part over the whole lifespan, we captured the mortality due to intrinsic risks. The parameters a(y) and  $\gamma(y)$  in the  $\gamma$ -Gompertz equation

$$\bar{\mu}_s(x, y) = a(y)e^{bx}\bar{s}_c(x, y - x)^{\gamma(y)}$$
<sup>(1)</sup>

were estimated for periods (y) and for ages (x) 80 to 100 by using Maximum Likelihood, whereas  $\overline{s}_{c}(x,y)$  is the correction for cohort survival. Since the b term is assumed to be negatively correlated with MM we suggest a constant value for b. b estimation is based on the population with the lowest extrinsic risk, which is assumed to be the most recent (Year 2000) female population of the respective country in our study.

The remaining mortality apart from  $\bar{\mu}_{g}(x, y)$  can be seen as MM  $\bar{\mu}_{m}(x, y)$  plus some infant mortality part  $\bar{\mu}_{i}(x, y)$ . Although the extend of infant mortality is to some extent due to extrinsic factors, we define it as a special case in our model, because infant mortality is mostly due to prematurity-related conditions, congenital anomalies and 'Sudden Infant Death Syndrome' (Sowards 1999). These causes are not fully understood and can be seen as a third risk factor due to a strong selection in early years. This factor is well described by the first part of the Heligman-Pollard model (1980):

$$\bar{\mu}_{i}(x,y) = A(y)^{(x+B(y))^{C(y)}}$$
(2)

Parameters were estimated for the ages (x) 0 to 10 for periods (y) and the modeled  $\overline{\mu}_i(x, y)$  was extrapolated over the whole lifespan.

By taking the residuals, the remaining MM can be described as follows:

$$\bar{\mu}_{m}(x, y) = \bar{\mu}(x, y) - (\bar{\mu}_{i}(x, y) + \bar{\mu}_{s}(x, y))$$
(3)



Figure 1. Illustration of mortality curve composition for US males 2007. Total mortality  $\overline{\mu}(x)$  is plotted as dots, the exponentially decreasing line is infant mortality  $\overline{\mu}_i(x)$ , the straight increasing line is the part due to ageing  $\overline{\mu}_x(x)$  and the unsteady line is the Middle Mortality  $\overline{\mu}_m(x)$  also represented by the grey area.

In the next step we calculated the relative amount of MM and called it relative Middle Mortality (rMM) which can take values from 0 to 1.

$$rMM = \frac{\underline{\mu}_m(x_{\mathcal{N}})}{\underline{\mu}(x_{\mathcal{N}})} \tag{4}$$

The shape of the rMM shows a very typical behavior for all studied countries. The first part is characterized by a steep increase starting with puberty at age ~ 10, followed by a peak at age ~20 and then decreases with a long tail (Fig. 2). A similar pattern is true for females.



Figure 2. Relative ammount of middle mortality to the total mortality (rMM) for US males 2007. The maximum of rMM is represented by the vertical dashed line (x = 19).

Since the Peak of rMM can be easily detected for every country in every year we used it for further comparisons.

#### Data

We used available sex specific 1 year period and cohort x 1 year age data from Sweden (1900-2000), Denmark (1936-2000), Belgium (1941-2000) and France (1916-2000) provided by the Human Mortality Database (<u>http://www.mortality.org/</u>).





Figure 3. Relative Middle Mortality Peaks (rMMP) over time for four european countries. Males = blue lines. Females = red lines. The dotted line represents the year 1945.

Swedish and French data imply that the rMMPs are almost similar for males and females until 1945 with a male bias during both World Wars. After the Second World War some change occurred. The rMMP drops down for both sexes. This trend could

be due to an increasing social equality which lead to a decrease in infectious diseases, a leading cause of death in adolescents before 1945 and still in the early 1950's (WHO database, <u>http://www.who.int/</u>). Nevertheless females rMMPs drop to a lower level compared to males. One factor could be the usage of passenger cars by a growing amount of the population. Since males used cars more frequently and behaved more recklessness, the death rate caused by traffic accidents is higher in men (WHO database, <u>http://www.who.int/</u>) and can explain the higher rMMPs. Nevertheless in the 1970s women's rMMP started to increase due to a social transition: In 1974 Sweden became the first country to give parents the right to paid leave from work at childbirth, this allowed women to participate in the labour force which increased the frequency of car usage and their environmental risk.

#### Conclusion

We showed that the rMM model provides a good tool to observe changes in external risk factors in adolescents. The next step is to develop a formal description for a better understanding and quantification of the rMM/MM shape. The interaction of extrinsic and intrinsic processes is not fully understood yet and the discussed method depends on measures and selection of mortality models for old ages as well as on rough assumptions in the way that the intensity of extrinsic risk among the elderly is very low. Nevertheless extrinsic risk and therefore MM is an important factor in testing social as well as evolutionary and ecological theories not just for humans but also for a broad range of species.

#### Literature

- 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,* Vol. 115, pp. 513-583.
- Heligman, L., Pollard, J.H. (1980). The age pattern of mortality. *Journal of the Institute of Actuaries,* Vol 107, pp. 49-80.
- Sowards, K.A. (1999). What is the leading cause of infant mortality? A note on the interpretation of official statistics. *American Journal of Public Health*, Vol. 89, pp. 1752-1754.
- Vaupel, J.W., Manton, K.G., Stallard, E. (1979). The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality. *Demography*, Vol. 16, No. 3, pp. 439-454.