



Statistical Analysis and Forecasting of Cause of Death Data: Novel Approaches and Insights

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To the memory of my grandfather, António

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Resume

Substantial improvements in public health resulted in exceptional increases in life expectancy. Nevertheless, mortality related processes have been change differentially at distinctive stages and accordingly with different related processes. To understand and analyze properly the mortality phenomena under a demographic point of view, it is indispensable to study populations as a heterogeneous mixture of individuals. Within this heterogeneous mixture, individuals are frailer than others and there are diverse risks of death competing with each other to be successful. Under this assumption, it is possible to differentiate between the individual pace of mortality increase with age, i.e., the individual rate of aging and the population rate of aging. If the first proves to be a biological constant invariant across humans and over time, besides a great demographic and biological finding, many research disciplines and policymakers may take advantage of previous knowledge and validated information. Insurance companies, e.g., could make use of this previous knowledge to elaborate a more precise and complete evaluation of risks. Thus, coherent and accurate mortality forecasts could be also performed. But if this hypothesis proves to be wrong, recent coherent compositional approaches are available and need to be tested to fulfill the need for detailed forecast discriminated by cause-specific probabilities. Nevertheless, in order to let doubts aside, a distinction between population and individual rate of aging needs to be realized in a clear end complete perspective.

Briefly, in this piece of research we develop a consistent contemporary exploration of mortality patterns, under a heterogeneous perspective, and make use of obtained information to provide not only an interesting demographic and social perspective, but also a tool for decision-makers to evaluate and identify possible points of intervention in what concerns to public health.

Análise Estatística e Métodos de Previsão de Diferentes Causas de Morte: Uma Nova Abordagem

Resumo

De uma forma geral, apesar da evolução extremamente positiva dos comportamentos de mortalidade se traduzir em indicadores-resumo como é o caso da esperança de vida à nascença, ao longo dos anos, as melhorias registadas na saúde pública, encontram-se relacionada com diversos processos distintos. A elaboração de uma análise completa e detalhada do fenómeno da mortalidade sob uma perspectiva demográfica, torna-se assim, unicamente possível se esta análise tiver em conta a composição heterogenia da população. Por entre esta heterogeneidade populacional, existem indivíduos considerados mais frágeis do que outros, onde diversos riscos associados à probabilidade de morte, competem entre si para conseguirem levar a melhor sobre o indivíduo. Deste modo, tendo em conta esta conjectura, torna-se possível diferenciar entre o ritmo de aumento nos níveis de mortalidade por idade, i.e., a velocidade de envelhecimento, tanto associada ao próprio indivíduo, como à população em si. Caso a teoria de que o primeiro indicador será uma constante biológica invariável entre indivíduos e por todo o seu tempo de vida se verificar verdadeira, apesar de ser uma importante descoberta ao nível biológico e demográfico, resultará em informação relevante tanto para diversas áreas de investigação, como para decisores políticos. Companhias de seguros, por exemplo, poderiam utilizar este conhecimento prévio e elaborar uma avaliação de riscos muito mais precisa. Consequentemente, a elaboração de previsões de mortalidade mais precisas e coerentes seria uma realidade. No entanto, se esta teoria não for validada, os resultados obtidos não deixam de ser importantes, pois contribuirão sempre para trabalho futuro, e novas abordagens metodológicas consideradas coerentes para a previsão dos padrões de mortalidade continuam a emergir e a necessitar de serem testadas.

Resumidamente, com este estudo desenvolveu-se uma análise rigorosa e contemporânea dos padrões de mortalidade tendo em conta a heterogeneidade populacional, fazendo uso da informação obtida para elaborar não só uma análise interessante do ponto de vista demográfico, mas também proporcionar informação indispensável para a sociedade e para os decisores políticos.

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CHAPTER 1

INTRODUCTION

1.1. Mortality trajectories and related processes

“How long do we will live?” is one of the most important questions that every individual, as human being, would like to have an answer for. Oeppen and Vaupel (2002) did not answer to this question, but demonstrated that life expectancy is breaking old theorized limits and with time every human can expect to live longer and with improved health.

It is well known that the increase in human longevity occurs due to significant improvements in health and consequent reductions in mortality rates. This improvement can be explained by two main phases, firstly, were massive reductions that occurred in mortality at younger ages, secondly, were the reductions in mortality rates associated to old-age on the second half of the last century, especially after age 65 (see e.g., Oeppen and Vaupel, 2002). Today, the high levels of mortality of the past are being experienced later in life. Nevertheless, observed mortality reductions are not following the same rhythm of decrease across different causes-of-death (from which *circulatory system diseases* and *neoplasms* are assuming the most important role), countries, ages, or even, between sexes (see e.g., Meslé, 2006). Mortality improvements increased, consequently, human longevity and originated considerable negative consequences in what concerns population aging. Nevertheless, individuals are reaching older ages in better physiological conditions (see e.g., Vaupel, 2010).

As acknowledged, mortality rates present different evolutionary patterns across populations, sexes and causes of death. However, it's not sure whether all individuals share the same rate of aging, i.e., the same chance of death across age. Vaupel's hypothesis (2010) suggests that the rate of aging, defined as the relative derivative of the baseline risk of dying, might be a biological constant for every species. Previous studies (Gampe, 2010) found evidence for a leveling-off of human mortality at ages 110-114, speaking in favor of a relative-risk model (Missov and Vaupel, 2015) with a Gompertz-Makeham baseline hazard (Gompertz, 1825;

Makeham, 1860). Unobserved heterogeneity (frailty) can be captured by a gamma distribution with a unit mean and γ variance at the starting age (Vaupel et al., 1979; Missov and Finkelstein, 2011; Missov and Vaupel, 2015). This leads to the gamma-Gompertz-Makeham frailty model (Vaupel et al., 1979) as the model basis to obtain accurate estimates in what concerns the individual rate of aging.

Nevertheless, in opposition with the rate at which age-specific death rates increase, known as the life-table aging rate (LAR) (Horiuchi and Coale, 1990), the individual rate of aging is defined as the relative derivative of the baseline hazard of death if the aging process is captured by a Gompertz curve. If the first is constant across age, the second presents a well-defined and characteristic bell-shaped pattern, widely studied since Gompertz (1825), and consequently finding evidence for a mortality deceleration at older ages. The force of mortality for an entire population is described by the associated age-specific death rates, resulting from a “contribution” of different subpopulations under diverse specific mortality conditions (Beard 1959; Vaupel et al., 1979).

Summarizing, mortality research necessarily requests to analyze population as a heterogeneous group composed by different subpopulations where the individuals are exposed to the hazard of death in very different and random ways, and as a whole, population present distinctive behaviors from the individuals. Thus, every individual in overall population or across different subpopulations is continuously exposed to different risks of death. *“Because death is not a repetitive event and is usually attributed to a single cause, these risks compete with one another for the life of a person. Competing risks must be considered in any cause-specific mortality analysis”* (Chiang, 1991).

A proper evaluation of those risks contributes to a complete and accurate evaluation of probabilities of death, which are important to extrapolate to the future and contribute to decision and policymakers evaluate where to intervene. In this way, accurate forecasts discriminating mortality by cause of death are indispensable. Nevertheless, in a theoretical perspective many authors support that breaking down mortality forecasts by cause results in higher accuracy but practice also proves the opposite (Booth and Tickle, 2008). Studies like the one from Wilmoth (1995) found that decomposing mortality forecasts result not-rarely in higher mortality forecasts. Oeppen (2008) makes use from one of the most known statistical models to forecast

mortality trends over time, the Lee-Carter model (Lee and Carter, 1992), and expressed in a Compositional Data Analysis (CoDa) its equivalent definition, avoiding those issues. In the CoDa equivalent model, the constraint imposed by the sum to the unit of each of the compositional vector, ensures that “*changes in the density by age and cause have to be compensated by changes in other ages and causes*” (Oeppen, 2008).

1.2. Motivation and aim of the study

The main motivation of this study comes from the interest in evaluating Vaupel’s hypothesis about the rate of individual aging. If the findings prove the hypothesis to be correct, its results are not only an important major finding for demography but also for biology. Demographic specific areas as, e.g., mortality forecasts may take advantage of more important and previously known information to elaborate more accurate forecasts. Nevertheless, to explore properly this subject, it is fundamental to understand mortality as a whole, analyzing its evolutionary patterns as an outcome of a heterogeneous mixture of individuals. At the same time, it is indispensable to differentiate between individual and population rate of aging. The knowledge resultant from this research is an indispensable tool to consolidate precise information for decision and policymakers.

Consequently, the overall aim of this study is to contribute to mortality research with important knowledge that comes from investigating mortality trends and its related processes. In this study, we focus essentially on the detailed evaluation of mortality trends discriminating between who and what is contributing the most for the observed increases in human longevity, and which are the leading causes of death across different countries. We also identify how Japan becomes the leading country in what concerns to life expectancy. We do not only focus on longevity measures, as life expectancy, but also on alternative measures of longevity as the median and modal age at death, to give a complete evolutionary perspective. Across entire research, discriminating the analysis by cause of death was a constant.

In the end, we shed light into mortality research by answering questions as: (1) how mortality trends developed across time; (2) what were the registered paces of increase in longevity; (3) are neoplasms consolidating as the cause of death with major impact in mortality, or can we identify one other that needs special attention;

(4) is the reduction in mortality at older ages the most important source of life expectancy increase; (5) is that really true that all individuals age at the same rate; (6) how and why the rate of individual aging is different from the one for individuals; (7) can we identify any relation between the age where the rate of aging for the entire population reaches its maximum with changes in the slope of life expectancy increase; (8) is it really possible to elaborate coherent forecasts of mortality trends when discriminating mortality by different causes of death; (9) if different causes of death present dissimilar rates of aging for individuals, is it still possible to aggregate them based on similar trends; and lastly, (10) which causes of death will play a major role in a near future.

1.3. Outline of the study

Including the present Introduction, this study consists in six different but complementary chapters. In *Chapter 2*, is provided an empirical overview about the evolution of mortality trends and its impact on overall longevity. The study relies on three different measures of longevity (life expectancy at birth, median and modal age at death) in order to evaluate how and why possible positive evolutions in mortality rates contributed to the increasing lifespan registered across time. Here it is also our intention to explore the female/male gap and understand why Japan is a standard model in the last years. To better understand this evolution, the analysis cannot be simply restricted to overall mortality, and we not only discriminate the contribution that the observed reductions in mortality by age had to life expectancy at birth improvement, but also complement those improvements adding information by cause of death. The analysis on the duality between early and old age mortality is a constant explored across the chapter.

In *Chapter 3*, our analysis focused entirely on the most important agent in demography, i.e., in the individual itself. Vaupel (2010) hypothesizes that “*except for individuals with accelerated aging disorders, all other humans have a similar and perhaps, essentially the same, rate of increase in mortality with age*”, i.e., the same rate of aging, which might be a biological constant invariant across humans and over time. Thus, we test Vaupel’s hypothesis by studying major groups of cause of death under a gamma-Gompertz-Makeham framework, seeking evidence to confirm or

refute the stated hypothesis by estimating the individual rate of aging by taking each cause as a subpopulation.

The analysis accomplished in *Chapter 4*, is elaborated once again under a gamma-Gompertz-Makeham framework, refers now to the population rate of aging, or as it is widely known, to the life table aging rate. The analysis explored in this chapter can be summarized in three stages: (1) first, we fit the model to the countries selected based on the qualitatively different evolutionary patterns of life expectancy at birth over time; (2) elaborate on the possible relationship between the population rate of aging and the rate of life expectancy increase in the selected countries, evaluating simultaneously how estimated patterns reflect the age patterns of mortality deceleration for overall and cause-specific mortality; and lastly, (3) we test the “heterogeneity hypothesis” by Horiuchi and Wilmoth (1998), which states that for the characteristic measure’s bell-shaped pattern a) deceleration occurs for the most major CODs, being less pronounced for the ones with lower death rates, and b) mortality deceleration should occur at later ages due to selection effects. The age at which the maximum value for life table aging rate is obtained is called here the age of mortality deceleration, and is used as a new measure of longevity that is used to evaluate how it reacts to changes in life expectancy dynamics.

In *Chapter 5*, we elaborate cause-specific mortality forecasts in order to provide not only an interesting demographic analysis, but also to decision-makers a tool to evaluate and identify possible points of intervention in what concerns to public health. In order to avoid overestimating mortality rates for overall mortality after summing up the obtained results, we make use of Oeppen’s (2008) suggestion of expressing the original Lee-Carter method to forecast mortality trends in compositional form. We elaborate thus, coherent medium-term (30 years horizon) forecasts distinguishable by cause of death for distinctive countries with different cultural backgrounds and characteristic mortality patterns.

Finally, *Chapter 6* concludes, based on a brief, but complete summary, of the most important findings achieved throughout previous chapters. An introspective discussion about the work done and insights resulting from the study is included, as well as the limitations that constrained the approach are identified. Additionally, an outlook for future challenges is also provided.

CHAPTER 2

THE RAISING OF LIFE EXPECTANCY IN EUROPE AND THE BEST-PRACTICE COUNTRY: A CROSS-SECTIONAL COMPARISON

2.1. Introduction

It is well known that, across the years, mortality improvement originated an increase on the average length of live, and consequently, the variance in age at death has decreased. This improvement occurred in two phases, being the first one connected with reductions in the mortality rates at younger ages, and the second one due to improvements in survival after age 65. Therefore, human life expectancy is rising to values thought to be not attainable and is breaking old theorized limits (Oeppen and Vaupel, 2002). The high levels of mortality of the past are now experienced at older ages, suggesting that senescence is being delayed and not stretched (Vaupel, 2010). This brings up the discussion about the concepts of aging and senescence, and if aging is an intrinsic characteristic of life course, senescence becomes more likely as life proceeds (Hamilton, 1966) but is not an inevitable condition (Baudisch, 2008). Nevertheless, if aging can be also connected with some good variation in human condition, as becoming wiser for example, senescence can be described as the decline in physiological functioning with age (Comfort, 1964; Finch, 1990).

In this chapter, we evaluate how and why the registered evolutions in mortality rates contributed positively to life expectancy increase, discriminating different patterns and trying to understand the observed gap between males and females and why Japan is on the leading. Thus, we elaborate single and multiple-decrement life-tables, with the intention to discriminate the impact that each cause of death (COD) has in life expectancy; and applied two distinct decomposition methodologies that allow to evaluate the gains in life expectancy at birth accordingly to different contributes across ages (Arriaga, 1984) and discriminate those contributions also by cause (Shkolnikov et al., 2001).

2.2. The epidemiologic transition and the evolution of mortality rates

Together with the decline in mortality rates, the high rates of mortality are shifting to older ages worldwide. If we break down mortality rates accordingly to different CODs, we can conclude that distinctive patterns of evolution can be identified and the different causes present dissimilar patterns across distinct nations. If in the past were the infectious diseases that strongly affected entire populations, nowadays, neoplasms are leading in almost all of the developed countries. In this way, the elimination of any preventable cause will result in an increasing lifespan, having impact not only in the overall mortality, but as well, in age-specific mortality rates.

All these developments suggest that mortality advances are closely related to health improvements, mainly due to population prosperity and medicine development. Preventive medicine diffusion among medical doctors played a major important role in medicine development, together with the identification of the microbiological agents from infectious diseases (Morais, 2002). If prosperity represents better opportunities to live a healthier life due to better chances in satisfying some basic needs (at least in most developed countries), advances in medicine provide a better approach to compete with most severe diseases and allows the population to have more opportunities to access better treatments. Nevertheless, measuring health is not an easy task due to its subjectivity, so, “*mortality is by far the most important readily and reliably measured index of health*” (Vaupel, 2010), also agreed by Santana (2005).

The theory of epidemiological transition presented by Omran in 1971, was the first relating the nature of the connection between the observed CODs with the age patterns of mortality for one population, pointing out that mortality declines were related with the reductions observed in the deaths caused by infectious diseases and continuous shift of degenerative diseases to older ages. In this situation, children and young women were the ones with more benefits with increases in life expectancy (Omran, 1971). The theory of epidemiologic transition try to combine the different factors that play an important role in the past mortality trends (Meslé and Vallin, 2006), presenting three main stages, or like it was called by Omran, three epidemiologic ages. The first age, is denominated as the age of pestilence and famine,

which was characterized by high rates of mortality and low life expectancy (around 30 years of average life), marked by the predominance of infectious diseases, endemics, epidemics and famine. The second stage, or age, is documented as the age of receding pandemics, where a strong rise in life expectancy was observed, mainly due to the lower frequency of epidemics and fewer registered cases of endemic infectious diseases. Finally, during the third age, the age of degenerative and man-made diseases, a continuous decline and consequent stabilization of mortality at very low rates was presented. In this last stage, the pace of increase in life expectancy starts to decelerate and seems to reach a threshold.

Nonetheless, the mortality progress is being continuous and the epidemiological transition theory proposed by Omran is now considered, as discussed by Meslé and Vallin in 2006, as becoming outdated, despite that the three-presented ages reflected accurately the reality until the end of the 1960s. Following the authors, a massive decline in cardiovascular diseases in the western countries resulted in a new acceleration in the pace of increase of life expectancy in the early 1970s. Still, Meslé and Vallin, find it preferable to adopt the concept of health transition, once that it is a wider concept and if in a first phase includes the gains in life expectancy related with the reductions in the levels of mortality due to infectious diseases, a second phase is advanced, marked by the decline in cardiovascular diseases. The concept of health transition also opens space for the possibility of future changes originated by reductions in mortality rates due to different causes, like the example of cancer, or due to delays in senescence, already expressed by Vaupel in 2010.

As expected, the presented stages that characterize the mortality progress did not occur at the same precise time across different countries, presenting major disparities when mortality is focused on COD patterns. Even within the same country, it is possible to identify diverse evolutionary stages at the regional level possibly closely related to the socioeconomic development stage of a region. Oliveira et al. in 1994, found a clear and distinctive level of socioeconomic development between the interior north and the south coast of Portugal. In this study, the authors also identified a strong evidence of deaths caused by malignant neoplasms, ischaemic heart diseases and tuberculosis. On late 20th century, infectious and parasitic diseases and diseases of the circulatory system declined and circulatory system diseases were negatively influencing health of males above age 45 (Morais, 2002). Oliveira and Mendes (2010)

also found in their study, that in Portugal, the life expectancy gap between sexes was increasing till middle 1990s, only declining afterwards.

Comparing Portugal and Spain, Canudas-Romo et al. (2008), concluded that the first was ahead in what concerns to positive life expectancy improvements, diminishing the registered gap between both countries, however, is still behind its south European neighbors (Fernandes, 2007).

Following the COD statistics published by the EUROSTAT (2014), we can realize that in a general way, between 2004 and 2010 cancer related death rates diminished 8.4 % and 4.8 % from females and males, respectively, across the European Union countries. Deaths caused by ischaemic heart diseases and transport accidents registered were reduced in more than 20 %. These results correspond to a very positive evolution, but neoplasms (principal cause) and diseases of the circulatory system are still the leading CODs. The most common causes related to the circulatory system diseases are the ischaemic heart diseases, affecting mainly, e.g., countries like Slovakia, Hungary and Estonia. Portugal, France and Spain are among the countries with lower death rates related to ischaemic heart diseases. Identified as the third most common cause across the European Union, respiratory system diseases presented the highest death rates in the United Kingdom, Denmark, Ireland and Portugal. In Japan, the results presented in the Japan Statistical Yearbook of 2014, showed a very identical pattern, once that neoplasms, circulatory and respiratory system diseases are the most recorded.

2.3. Methods

2.3.1. Measuring longevity: life expectancy, median and mode

Population longevity is often measured in terms of life expectancy. However, other measures can provide different insights about longevity trends and better understanding about possible differences observed across countries. This is why we choose to include in this analysis, the median and modal ages at death.

If life expectancy can be calculated as the mean age at death, taking the single decrement life-table methodology we have:

$$e(x, t) = \frac{T(x, t)}{l(x, t)} \quad (2.1)$$

where, $e(x, t)$ is the life expectancy at age x and time t , $l(x, t)$ is the proportion of people alive at age x , and $T(x, t)$ the person-years lived above age x .

The median age at death (Md, t) is the age by which half of the population at time t is dead, i.e., when the survival function equals 0.5: $l(Md, t)=0.5$. So, letting x and $x+1$ be the ages of the interval in which $l(Md, t)=0.5$ is located, the median age at death is given by:

$$Md(t) = x + \frac{[0.5 - l(x, t)]}{[l(x + 1, t) - l(x, t)]} \quad (2.2)$$

Lastly, the modal age at death is the age when most of the deaths occur: $M(t) = \{x \mid \max[d(x, t)]\}$. Here, $d(x, t)$ is the life-table density function for the distribution of deaths. Nevertheless, and taking into account the age distribution of deaths, two modes should be distinguished (Canudas-Romo, 2010): one at age 0, where people die soon after birth, and a late mode found at older ages. In this essay, our focus is in the second one, i.e., the late modal age at death, and for this only deaths occurred after age 5 are considered:

$$M(t) = \{x \mid \max[d(x, t)] \text{ for } x > 5\} \quad (2.3)$$

To obtain a precise result, Kannisto (2001) proposed an approach that allows obtaining a modal age at death with decimal precision. In this way, taking x as the age with the highest number of deaths in life the table at time t , we have:

$$M(t) = x + \frac{[d(x, t) - d(x - 1, t)]}{[d(x, t) - d(x - 1, t)] + [d(x, t) - d(x + 1, t)]} \quad (2.4)$$

2.3.2. Multiple decrement life-tables

If the conventional construction of a life-table is described as a singular decrement process in which individuals have only one mode of exit from a defined state,

multiple decrement processes, like the name suggests, are the ones in which the individuals have more than one mode to exit. These processes can be used in a set of different approaches, e.g., in the analysis of fertility, migration or nuptiality. Nevertheless, and if one of our main goals is to interpret the impact that each COD group has in population life expectancy, multiple decrement life-tables, period based, will be constructed. Here, the intention is to estimate which would be the expected life expectancy in the absence of a certain cause. However, many critics are raised against this approach because there are significant competing risks to which all individuals of a population are exposed, and if an individual doesn't die from a certain COD, it is possible that another cause attempts to be successful.

In this way, if the conventional life expectancy is given by equation (2.1), the life expectancy that a population can expect in the absence of a certain disease is given by:

$$e^{-i}(x, t) = \frac{T^{-i}(x, t)}{l^{-i}(x, t)} \quad (2.5)$$

where $-i$ represents the absence of a certain cause.

2.3.3. Decomposing differences in life expectancies

The simple measure of changes in life expectancy at birth between two different periods, populations or CODs, is very useful to calculate the rate of increase in life expectancy across time or countries, or even simply to quantify the increase itself. However, it is also very useful to estimate how mortality differences in a specific age group can contribute to the total difference in life expectancy. In this chapter, one of our goals is also to define the importance of older ages in life expectancy increment. To reach our goal, two different methodologies were chosen: firstly, the one developed by Arriaga in 1984 (and explained by Preston et. al. in 2001, for example), for the decomposition of total increases in life expectancy; and, secondly, the methodology developed by Shkolnikov et. al. in 2001, that will also allow to quantify the contribution of different age groups, but, identifying at the same time, the contributions of different CODs.

Starting by the discrete approach proposed by Arriaga (1984), we have that the total effect, ${}_n\Delta_x$, of a difference in mortality rates between ages x and $x + n$, is given by:

$${}_n\Delta_x = \frac{l_x^1}{l_0^1} \left(\frac{nL_x^2}{l_x^2} - \frac{nL_x^1}{l_x^1} \right) + \frac{T_{x+n}^2}{l_0^1} \left(\frac{l_x^1}{l_x^2} - \frac{l_{x+n}^1}{l_{x+n}^2} \right) \quad (2.6)$$

where:

$$e_0^2 - e_0^1 = \sum_0^\infty {}_n\Delta_x \quad (2.7)$$

If the first term of equation (2.6) corresponds to the direct effect of a change in mortality rates, the second term, corresponds to the sum of the indirect and interaction effects, i.e., the contribution of the exposition to new mortality conditions. For the open-ended age interval, there will be only a direct effect, resulting in the following equation:

$${}_\infty\Delta_x = \frac{l_x^1}{l_0^1} \left(\frac{T_x^2}{l_x^2} - \frac{T_x^1}{l_x^1} \right) \quad (2.8)$$

Nevertheless, in presence of cause-specific mortality data, this approach can take a supplementary step and a further decomposition according different CODs can be performed. That was the intention of Shkolnikov et. al. in 2001, with the presentation of a new method that allows to decompose the differences between life expectancy by age and CODs. Here, the difference between the observed life expectancy for two populations between ages x and $x + n$ and CODs j , is given by:

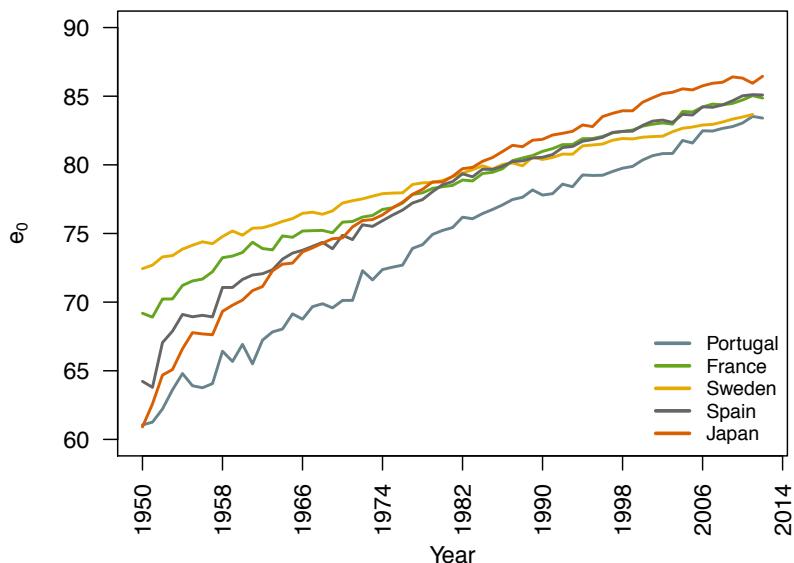
$${}_n e_{x,j} = \frac{{}_n M_{x,j}^1 - {}_n M_{x,j}^2}{{}_n M_x^1 - {}_n M_x^2} \Delta_x \quad (2.9)$$

where, ${}_n M_{x,j}^1$ and ${}_n M_{x,j}^2$ are the central death rates for two different populations between ages x and $x + n$ and cause j . Additionally, ${}_n M_x^1$ and ${}_n M_x^2$ also correspond to the central death rates for the same two populations, but now, for all causes combined.

2.4. Data

Country selection was based on the qualitatively different patterns of life expectancy evolution over time, where France and Sweden, registering a high life expectancy at birth already in the 1950s, experienced a drop in the life expectancy rate of increase in the following decades; Spain, Portugal and Japan experienced a low life expectancy at birth in the 1950s, but the rates of increase surpassed the life expectancy leaders at the time, and the three countries caught up and the values of life expectancy in Spain even surpassed the ones registered in Sweden. Japan, registers today the highest life expectancy in the world (Figure 2.1).

Figure 2.1: Female life expectancy for selected countries, 1950-2012



Source: HMD 2014, own elaboration

Data on *overall death counts* $D(x, y)$ and *exposures* $E(x, y)$ derived from the *Human Mortality Database* (HMD 2014: www.mortality.org) and *deaths by cause* $D_i(x, y)$ from the *World Health Organization Mortality Database* (WHOMD 2014).

WHOMD COD data are only available by five-year age groups and once that we extract death counts according to the 10th International Classification of Diseases (ICD), our timeline in what concerns to COD is restricted essentially to the last decade (depending on the country data availability). Data codification corresponds to the ICD 10 detailed 3rd and 4th character list classification and was rearranged in 8 main COD groups: 1) Neoplasms, 2) Ischaemic Heart Diseases; 3) Cerebrovascular

Diseases; 4) Remaining Diseases of the Circulatory System; 5) Diseases of the Respiratory System; 6) Diseases of the Digestive System; 7) External Causes of Death; and 8) Remaining Causes of Death. Following the WHO recommendation (1977) to avoid uncertainty that comes from changes and updates in the classification system, we base our research on the *underlying cause-of-death*, *i.e.*, the disease or injury, which initiated the series of events that lead to death.

2.5. Results

2.5.1. Life expectancy patterns and the best-practice country

As discussed before, life expectancy is increasing with time due to general improvements in public health. Nevertheless, different countries present different evolutionary patterns and rates of increase in life expectancy.

Table 2.1 not only presents information about life expectancy values observed in 1950 and 2012 (due to data availability, 2011 for Sweden) for males and females, but also ranks the countries from higher to lower values. In the slightly more than 60 years of analysis, we can realize that major increases in life expectancy values were verified. In 1950, Japan, independently of the sex, was on the bottom, with lower life expectancy at birth. Sweden, was at the time, leading, with a life expectancy at birth of 72.44 and 69.83, for females and males, respectively. Thus, between those two countries existed a gap of 11.53 years for females and 14.04 years for males.

Recently, for the latest available year, Japan is on the “*pole position*”: a Japanese female newborn in the year 2012 could expect to live on average 86.45 years and a newborn male 79.96. If in the female case, Sweden is now presenting very similar values to the Portuguese case (Sweden: 83.67, Portugal: 83.41), expecting to live on average almost 3 years less (2.78) than a Japanese one, males are ranked on the second position with a gap lower than one year (0.16).

Despite the changes on the life expectancy ranking for the selected countries, we can denote a higher proximity between the presented values in more recent periods. For the most recent available year, the registered gap between countries for both sexes separately, is roughly 3. Nevertheless, the gap between males and females life expectancy at birth is still high. For the observed countries, the average difference between sexes in the year of 1950 was 4.37 and in 2012 increases to 5.73. Further,

breaking down the values of life expectancy by decade will give a better understanding about why the average difference between sexes increased.

Table 2.1: Ranking Countries by Life Expectancy

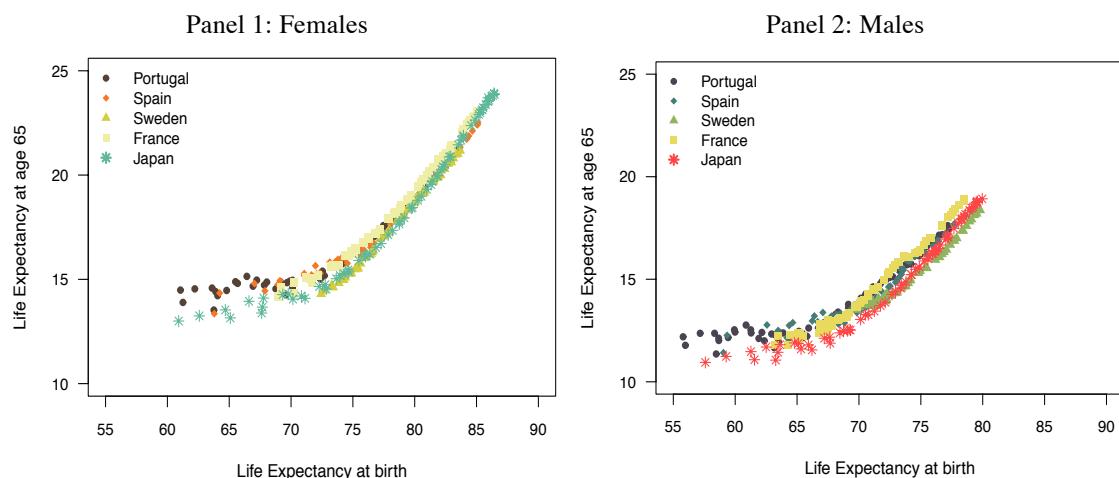
L. E. Ranking	Females				Males			
	1950		2012		1950		2012	
	Country	e_0	Country	e_0	Country	e_0	Country	e_0
1	Sweden	72.44	Japan	86.45	Sweden	69.83	Japan	79.96
2	France	69.19	Spain	85.09	France	63.43	Sweden*	79.80
3	Spain	64.23	France	84.87	Spain	59.35	Spain	79.33
4	Portugal	61.06	Sweden*	83.67	Japan	57.59	France	78.51
5	Japan	60.91	Portugal	83.41	Portugal	55.79	Portugal	77.26

* Due to data availability refers to 2011

Source: HMD 2014, own elaboration

Figure 2.2 represents both, life expectancy at birth and at age 65, for males and females from 1950 to the last available year, registered on the five selected countries. As expected, the recorded values present a clear and positive evolution of both longevity indicators, but if we imagine a two-break segmented regression line over the dots, a distinctive difference in the slope of life expectancy increase can be recognized. Plus, it was only when life expectancy at birth reached values around 75 for females and 70 for males, that life expectancy at age 65 started to increase significantly.

Figure 2.2: The evolution of Life Expectancy between 1950 and 2011/12



Breaking down the values of life expectancy at birth increase by decade (Table 2.2¹) demonstrates that for all countries and both sexes, but Swedish males, were the 1950s that recorded the highest increase of the analysis. On the other hand, after the year 2000, Portugal was the country that recorded the highest increase in opposition with Japan and Swedish females.

Generally, it can be also said that in the last 3 decades, Japan is the country that presents lower life expectancy increase and Swedish males are the ones that, in the same timeline, present higher improvements when compared with the first 3 previous decades. Males, when compared with females, present higher improvements in the last 3 decades, and the Portuguese ones recorded the highest observed improvement between the year 2000 and 2012. Thus, we can conclude, that despite the average gap between sexes have increased between 1950 and 2012, in the last decades, males are catching up and becoming nearer to female values of life expectancy.

Table 2.2: Quantifying the increase of life expectancy at birth by decade

Period	Portugal		France		Sweden*		Spain		Japan	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
1950 - 1960	5.49	5.87	3.60	4.43	1.41	2.44	7.32	7.42	7.73	9.24
1960 - 1970	2.60	3.20	1.35	2.21	1.01	2.34	2.65	3.22	4.00	4.53
1970 - 1980	4.22	5.10	1.78	2.58	0.54	1.63	3.07	3.68	4.06	4.08
1980 - 1990	2.66	2.57	2.57	2.58	2.03	1.54	1.03	2.00	2.55	3.11
1990 - 2000	2.49	2.55	2.52	1.84	2.57	1.62	2.57	2.32	1.75	2.70
2000 - 2012	4.00	3.06	3.26	2.05	2.42	1.66	3.36	2.22	2.29	1.89

* Due to data availability last year is 2011

Source: HMD 2014, own elaboration

Notwithstanding life expectancy as the most common used measure to evaluate human longevity, it is important, not only to policymakers specifically, but also for decision-makers in general, to identify when is half of the population of a country dying or when are occurring most observations.

Tables 2.3 and 2.4 present two alternative measures of longevity to life expectancy: the median and the modal age at death. Starting with the analysis of Table 2.3, one can realize that, normally, 50 % of the deceases occur later than the

¹ Whenever tables are presented with color graduation, it means that red colors recognize higher values

average (life expectancy), and the age where the highest number of deaths is recorded, is even later. In the year of 1950, for the five countries, 50 % of females deceased, approximately around age 74 (on average) and on last decade the value increased to about 88 years. From an average modal age at death of 80.50 in 1950, the female modal age at death in 2012 increased to an average of 90.24.

Table 2.3: Life Expectancy at birth, Median and Modal age at death for Females

	Year	1950	1960	1970	1980	1990	2000	2012	Tot. Inc.	Inc. Rate ¹
Portugal	e_0	61.06	66.92	70.12	75.22	77.80	80.35	83.41	22.35	4.26
	Median	72.59	75.80	76.79	79.34	81.24	83.35	86.04	13.44	2.56
	Mode	80.47	78.49	78.55	82.87	83.86	85.82	89.17	8.70	1.66
France	e_0	69.19	73.62	75.82	78.41	80.98	82.82	84.87	15.68	2.99
	Median	75.65	78.21	79.90	82.08	84.42	86.09	88.05	12.40	2.36
	Mode	80.63	81.86	82.77	85.12	86.92	88.64	90.55	9.91	1.89
Sweden²	e_0	72.44	74.88	77.22	78.85	80.39	82.01	83.67	11.23	2.17
	Median	76.51	78.30	80.45	81.87	83.35	84.70	86.36	9.85	1.91
	Mode	80.45	81.75	82.15	85.22	86.48	88.35	89.36	8.90	1.72
Spain	e_0	64.23	71.65	74.87	78.55	80.55	82.87	85.09	20.86	3.97
	Median	73.43	77.20	79.05	81.78	83.68	85.60	87.65	14.22	2.71
	Mode	80.47	80.36	82.75	84.63	86.59	88.11	89.95	9.48	1.80
Japan	e_0	60.91	70.15	74.68	78.75	81.86	84.56	86.45	25.54	4.86
	Median	69.76	75.45	78.25	81.78	84.70	87.42	89.26	19.50	3.71
	Mode	80.54	79.20	82.25	84.65	87.45	91.05	92.18	11.65	2.22

¹Rate of increase per year in months; ²due to data availability last year is 2011

Source: HMD 2014, own calculation

Compared with females, and as expected, 50 % of male's deaths (Table 2.4) are recorded earlier, occurring on average at age 69 in 1950 and 82.23 in 2012. Similar situation can be observed for the modal age at death, even presenting higher values than life expectancy at birth and median age at death, is still lower in average than for females (1950: 76.38; 2012: 86.20).

Nevertheless, from Tables 2.3 and 2.4, we also can access information about the total life expectancy at birth increase and the increase rate per year in months. Japan, as the best-practice country in what concerns to life expectancy at birth, present the maximum observed increase since 1950 being closely followed by Portugal, the country with lower life expectancy at birth in the analysis. Both

countries were even the only two recording an average monthly increase in life expectancy at birth of more than 4 months.

Table 2.4: Life Expectancy at birth, Median and Modal age at death for Males

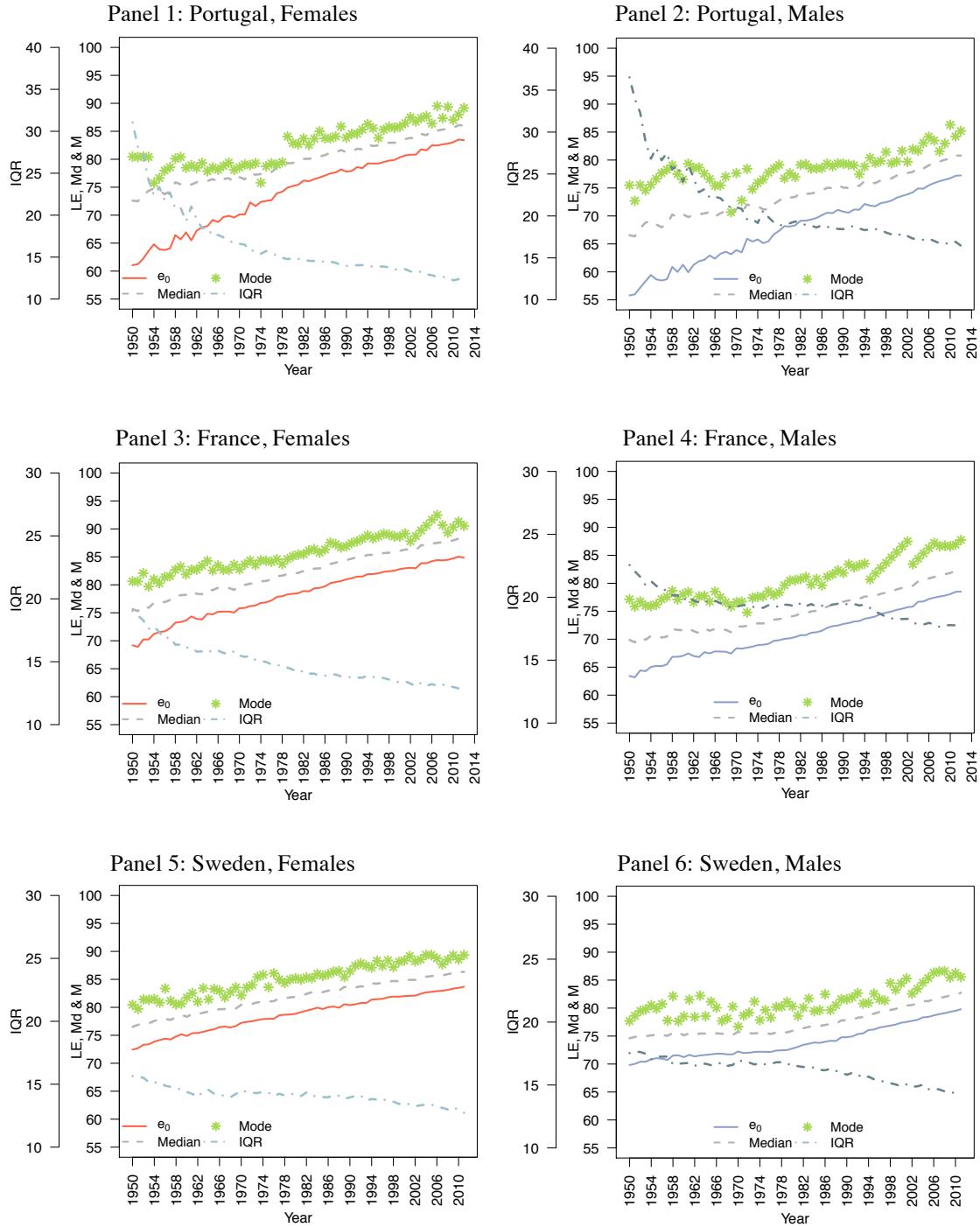
	Year	1950	1960	1970	1980	1990	2000	2012	Tot. Inc.	Inc. Rate ¹
Portugal	e_0	55.79	61.28	63.88	68.11	70.76	73.26	77.26	21.47	4.09
	Median	66.55	70.29	71.22	73.28	75.07	77.21	80.78	14.23	2.71
	Mode	75.49	76.51	77.65	77.65	79.34	79.75	85.20	9.71	1.85
France	e_0	63.43	67.03	68.38	70.16	72.73	75.25	78.51	15.08	2.87
	Median	69.88	71.58	72.32	74.02	76.71	78.90	82.31	12.43	2.37
	Mode	77.14	78.01	76.63	80.48	81.85	85.58	87.74	10.60	2.02
Sweden²	e_0	69.83	71.23	72.24	72.78	74.80	77.37	79.80	9.97	1.93
	Median	74.61	75.11	75.79	75.93	77.81	80.22	82.74	8.13	1.57
	Mode	77.69	78.52	76.69	80.30	81.57	84.43	85.56	7.87	1.52
Spain	e_0	59.35	66.66	69.32	72.39	73.42	75.97	79.33	19.98	3.81
	Median	68.19	72.71	74.03	76.07	77.20	79.25	82.34	14.15	2.70
	Mode	78.31	78.45	78.54	80.67	82.29	82.75	85.69	7.38	1.41
Japan	e_0	57.59	65.32	69.32	73.38	75.93	77.68	79.96	22.37	4.26
	Median	65.79	70.71	73.13	76.75	79.14	80.72	82.97	17.18	3.27
	Mode	73.28	75.54	77.42	80.42	83.28	84.41	86.80	13.52	2.58

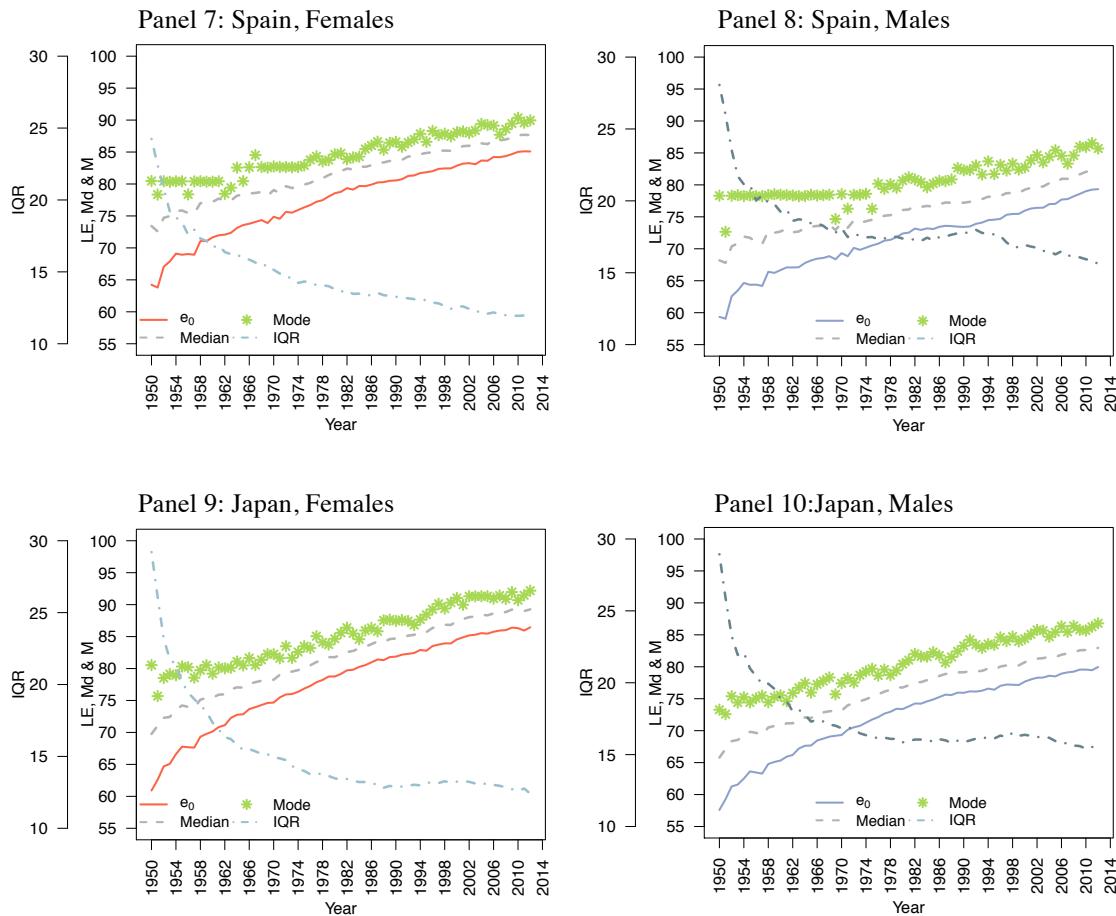
¹ Rate of increase per year in months; ²due to data availability last year is 2011

Source: HMD 2014, own calculation

Representing graphically information presented in Tables 2.3 and 2.4 (Figure 2.3), and adding the information about the interquartile range, one has a better idea about what was previously explained. We can also realize that besides the differences between the 3 present measures, always with positive patterns evolution, the interquartile range is diminishing. As a result, it indicates that high rates of mortality are becoming concentrated in a shorter age range, more specifically it can be concluded that the concentration of deaths occurs at older ages.

Figure 2.3: Life expectancy at birth, median and modal age at death



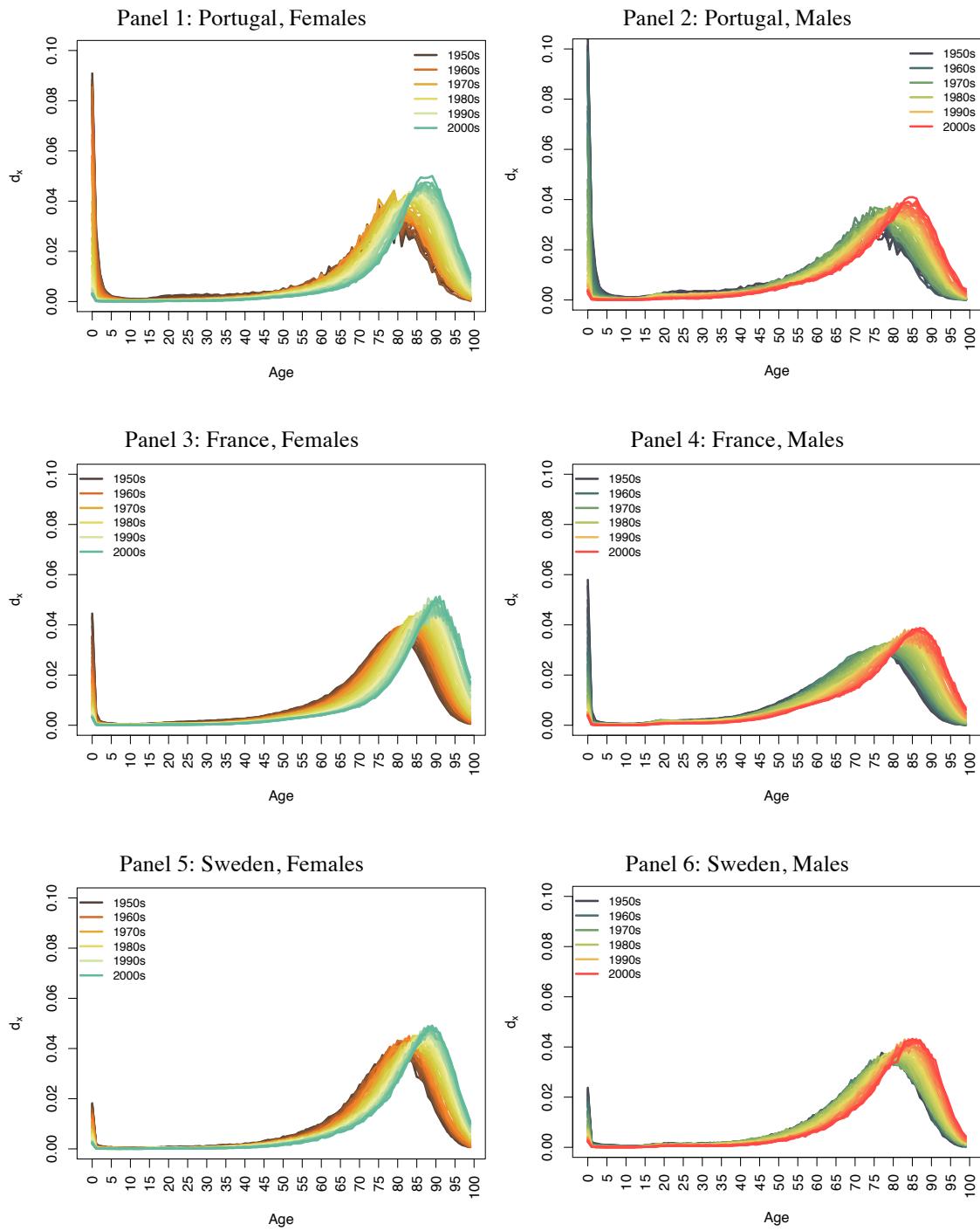


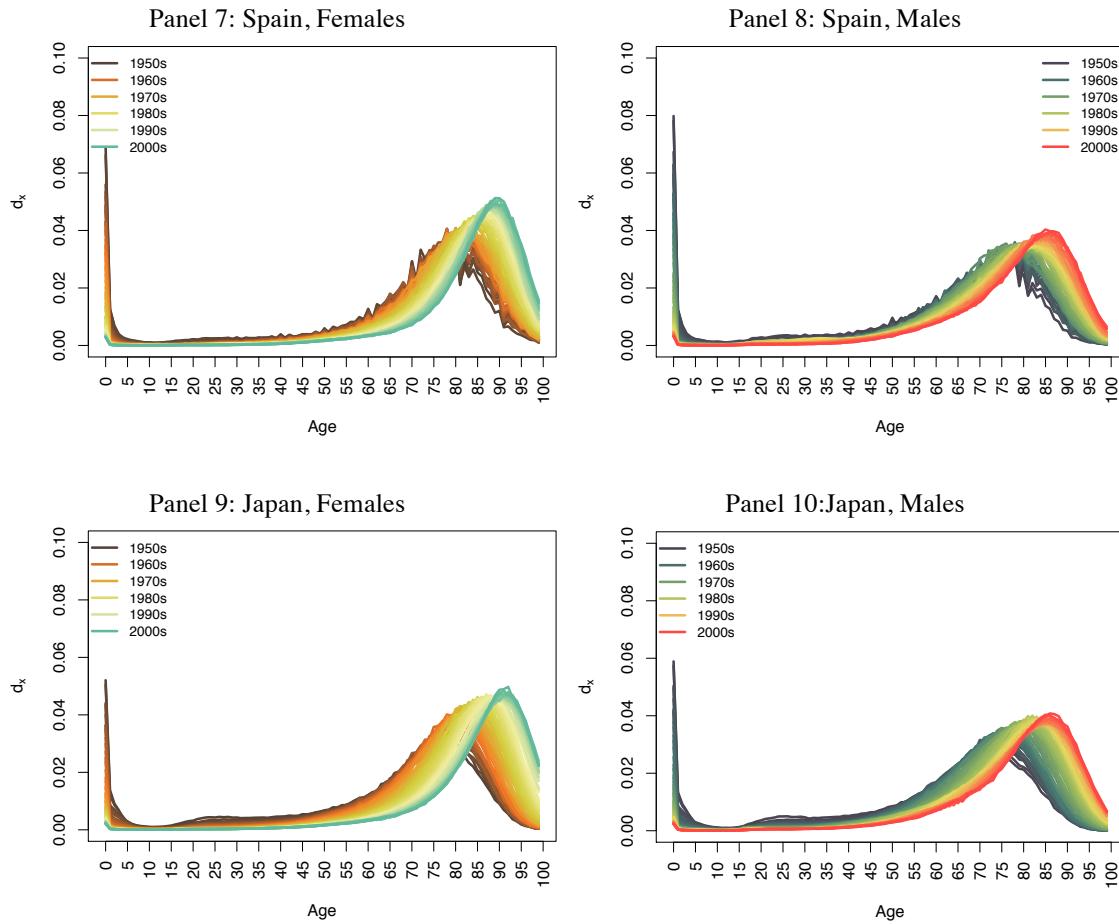
Source: HMD 2014, own elaboration

Figure 2.4 clearly confirms our previous deduction with graphical representation of the life-table age distribution of deaths d_x , for all five countries and both sexes. The colored patterns consent the identification of the different evolutions by decades, and as time proceeds, less and less deaths are being recorded at early ages, independently of country and sex, and most deaths are being recorded later in life. Despite the clear observed shift in deaths to older ages, the peak is becoming more and more pronounced, especially in the female case. As a result, it results in a rectangularization of the life-table survival curve, already widely studied.

Concluding, as expected, the observed life-table age distribution of deaths d_x , reflect unquestionably, the evolutionary patterns observed before for life expectancy at birth, median and modal ages at death.

Figure 2.4: Life-table age distribution of deaths d_x





Source: HMD 2014, own elaboration

2.5.2. Decomposing life expectancy improvements

Previous findings explained in the introductory part of this chapter, distinguished two main phases that contributed to the increase of life expectancy at birth: 1) massive reductions in mortality rates at younger ages, and 2) decrease in mortality rates beyond age 65. Taking advantage of this information, we re-calculated life expectancy at birth assuming that mortality rates after age 65 were maintained unchanged since 1950. Table 2.5 presents the obtained results and demonstrates that the consolidation of Japan as best-practice country was only possible due to improvements registered after age 65. If mortality rates for those ages become unchanged since 1950, Japan would be at the bottom of the “classification” in 2012 with 76.26 years of life expectancy at birth for females and 72.96 for males (nearby France and Portugal).

Excluding Japan, in the female case, life expectancy at birth would be around 77 years and for males would present higher disparity between registered values besides (Sweden: 75.49; Spain: 73.95).

Table 2.5: Life expectancy at birth if mortality rates kept constant after age 65 since 1950

L. E. Ranking	Females 2012				Males 2012			
	Country	e ₀	e ₀ [*]	Diff.	Country	e ₀	e ₀ [*]	Diff.
1	Japan	86.45	76.26	<u>10.19</u>	Japan	79.96	72.96	<u>7.00</u>
2	Spain	85.09	77.52	<u>7.57</u>	Sweden*	79.80	75.49	<u>4.31</u>
3	France	84.87	77.31	<u>7.56</u>	Spain	79.33	73.95	<u>5.38</u>
4	Sweden*	83.67	77.30	<u>6.37</u>	France	78.51	72.98	<u>5.53</u>
5	Portugal	83.41	77.29	<u>6.12</u>	Portugal	77.26	72.88	<u>4.38</u>

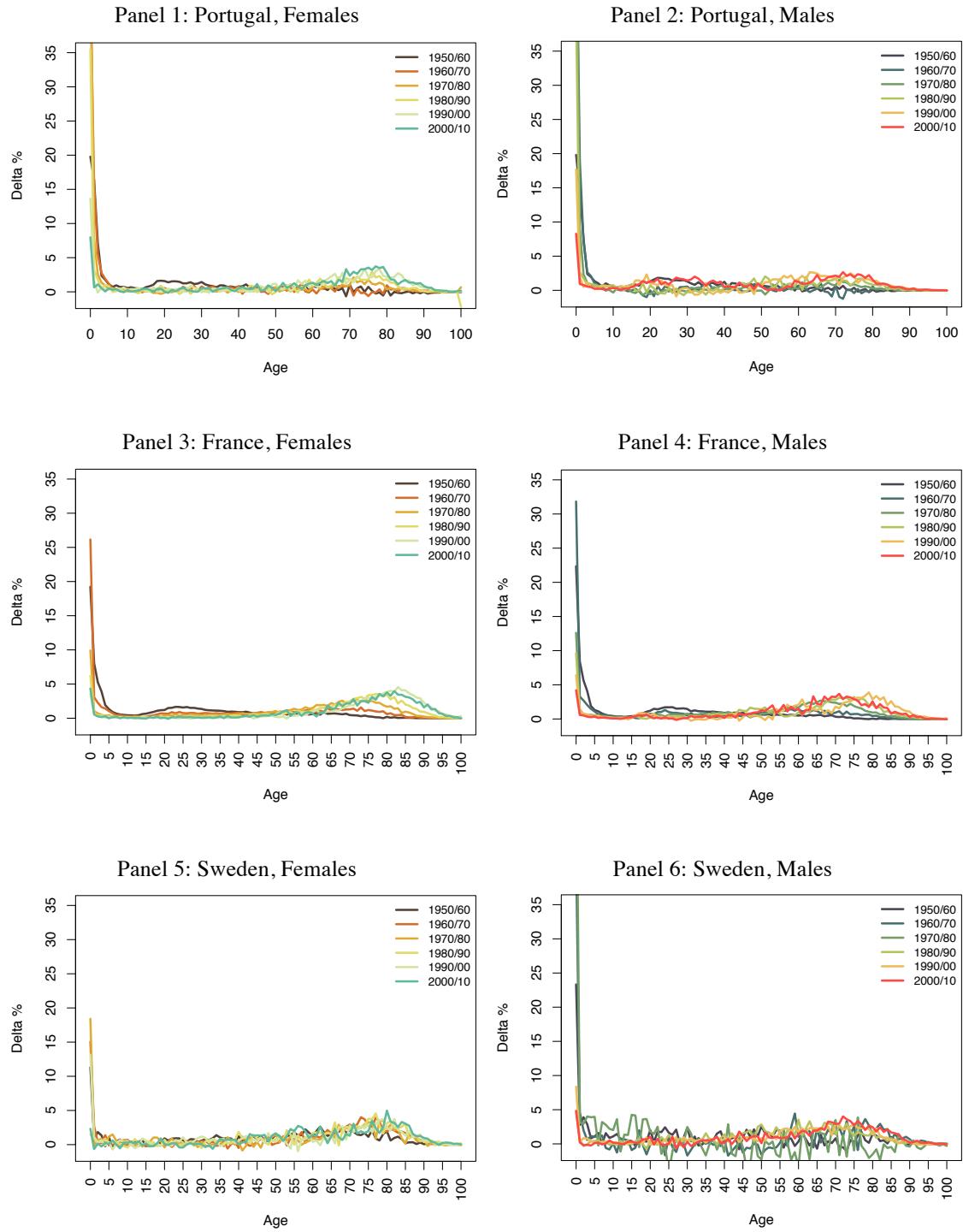
* Due to data availability last year is 2011

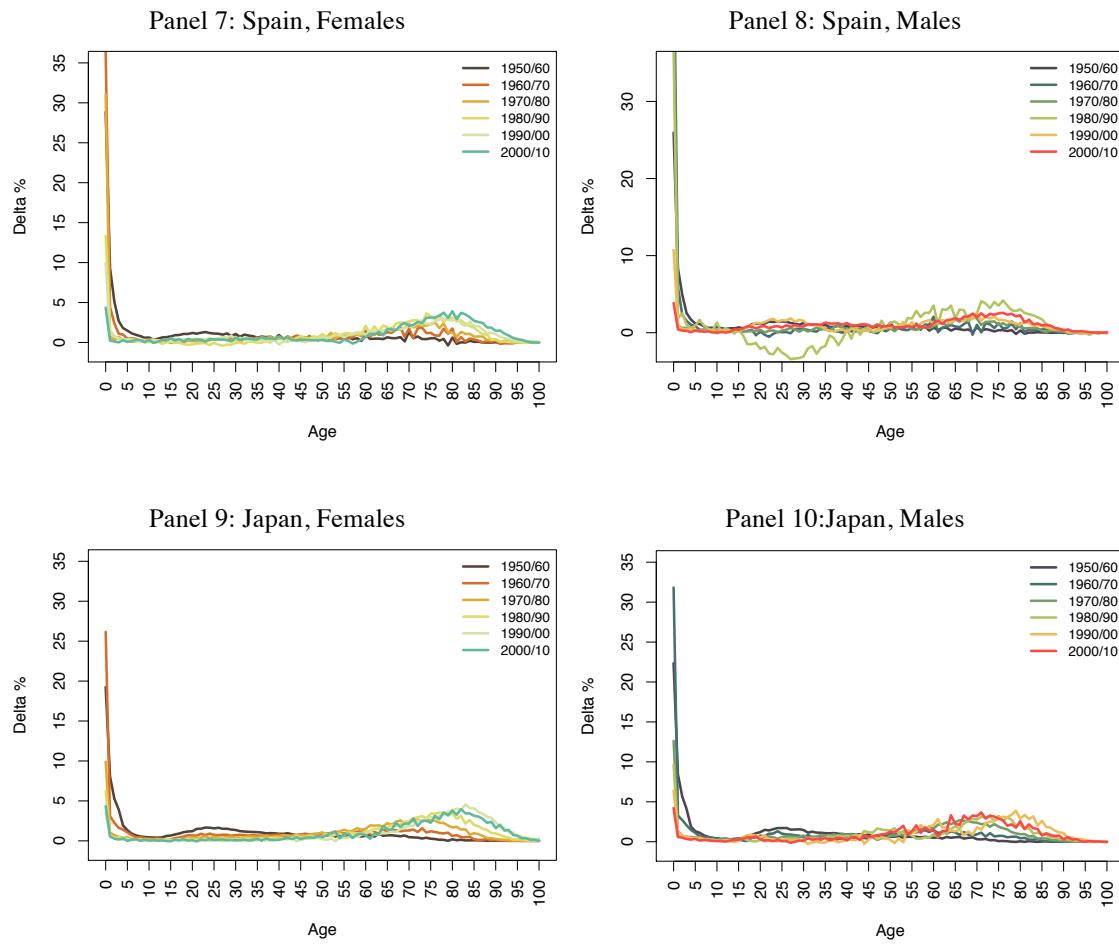
Source: HMD 2014, own elaboration

Complementary information can be extracted by the methodology developed by Arriaga (1984), once that allows identifying contributes by age (the obtained results are presented in Figure 2.5).

Generally, we can confirm that in the beginning of the period were the improvements at younger ages that contribute the most to life expectancy improvement. However, the positive contributions observed here are highly connected with older ages. If for females we can expect that for the future, improvements may be especially connected with older ages, in the male case, there is still a larger flexibility to improve at younger ages, essentially between age 15 and 35. As a result of previous studies, we can assume that contributes associated with those ages are mainly connected with behavioral decisions, i.e., external causes-of-death (essentially transport accidents in the Portuguese case, for example).

Figure 2.5: Contributions, in percentage, for e_0 increase



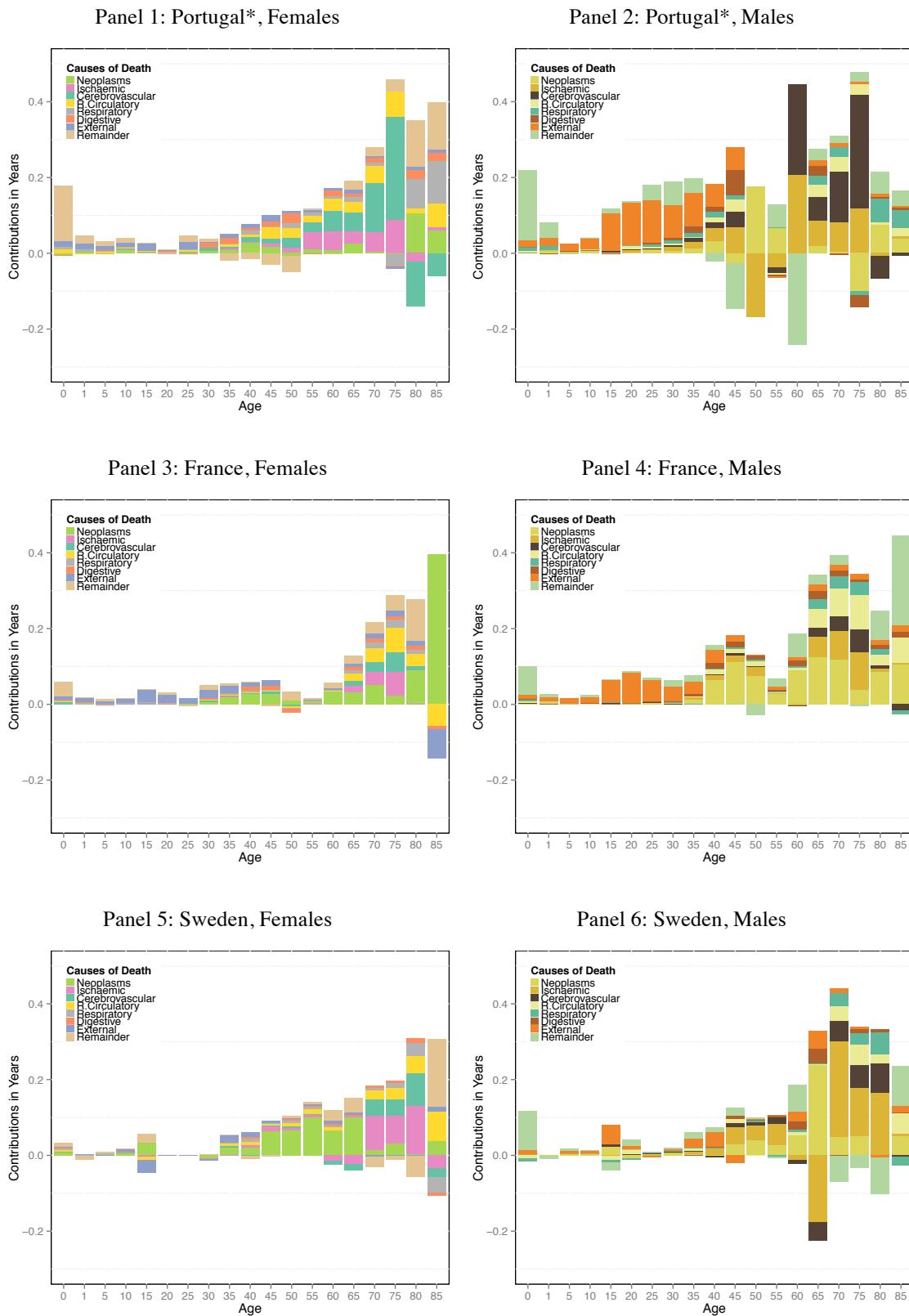


Source: HMD 2014, own elaboration

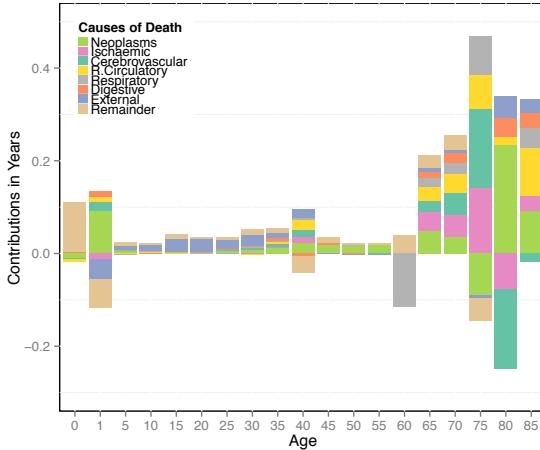
Despite the possible identification of positive or negative contributions to the increase of life expectancy applying the decomposition methodology developed by Arriaga (1984), the computation of the decomposition approach developed by Shkolnikov et al. (2001) allows to break down this contributions by COD.

Intending to focus on recent years, Figure 2.6 presents the obtained results for the last decade and we can already identify what was said before. Nonetheless, for this last decade, in the male case, some important contributions were given by young adult ages, especially related to deaths originated by external causes of death. Besides external causes for males, in a broad perspective, were the positive impacts of neoplasms, ischaemic, cerebrovascular and other circulatory system diseases that most “affected” life expectancy at birth. Lastly, especially at older ages, diseases of the respiratory system can also be identified as important.

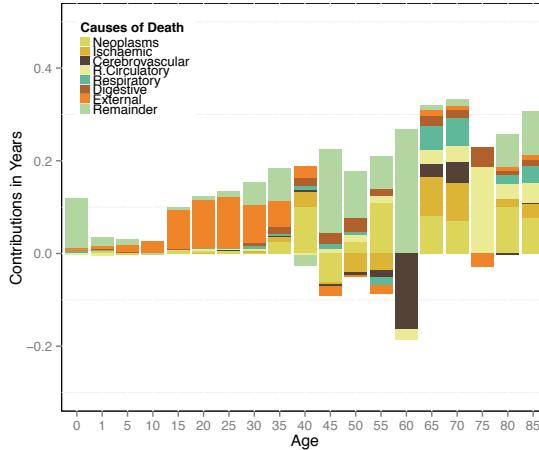
Figure 2.6: Contributions, in years, for e_0 increase by COD (2000 and 2010)



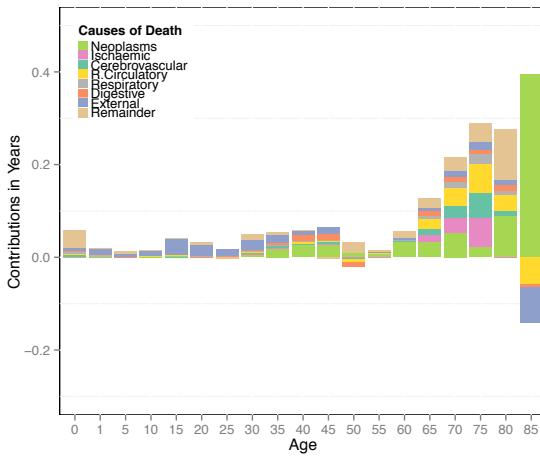
Panel 7: Spain, Females



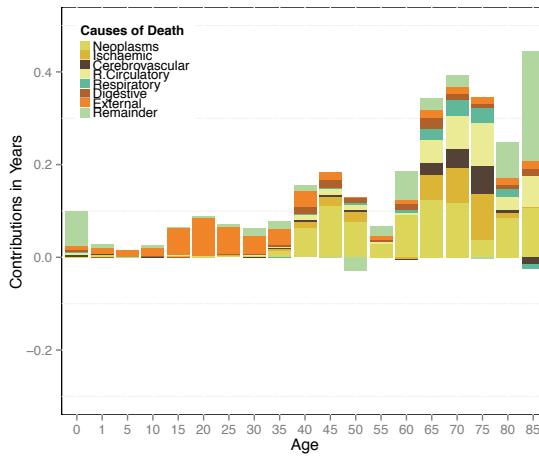
Panel 8: Spain, Males



Panel 9: Japan, Females



Panel 10: Japan, Males



* Due to data availability first year is 2003

Source: HMD 2014, own elaboration

With the previous analysis elaborated in this chapter, we can conclude that are (still in this decade) mainly the positive contributes given by older ages and reductions in mortality rates connected with neoplasms and diseases of the circulatory system in general, and external causes predominantly for males, that contribute the most for the registered life expectancy at birth in 2012. Nonetheless, if we calculate the associated single decrement life-table for other than a specific COD, we would be able to evaluate how much is the impact of a certain cause in overall life expectancy.

Thus, based on that approach, we calculated correspondent life expectancy at birth in the absence of each considered COD. Due to competing risks theory, which theorizes that if an individual doesn't die from a certain and specific COD, there are many different ones to which he is exposed, competing to contribute to his dead. We

need thus, to be aware that the real impact of the absence of a certain COD in life expectancy would be slightly lower than the obtained by this approach.

Our intention is simply to identify which COD influences the most, negatively, life expectancy at birth, and the obtained results for the year 2000 presented in Table 2.6 show that are the death related with neoplasms that had higher negative influence across all countries and sexes. In Sweden, again for both sexes, it can also be identified a negative influence caused by ischaemic heart diseases. Generally, as expected, were the diseases related with the circulatory system, together with neoplasms, the ones presenting a major negative impact. On the other side, were the deaths caused by digestive system diseases that less influence negatively life expectancy at birth.

The group that includes all the remaining CODs also records significant values to be neglected, however, this shows that there are still an important group of all other CODs not specified here that need intervention. It is not our intention in this monograph, but the identification of the different CODs correspondent to that last group, will certainly produce important information for the health public system

Table 2.6: Life expectancy at birth in the absence of a certain COD, 2000

COD	Portugal*		France		Sweden		Spain		Japan	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Neoplasms	3.44	2.67	4.89	3.48	3.25	3.44	4.47	2.96	4.74	3.88
Ischaemic	1.23	0.93	1.17	0.86	2.86	2.16	1.51	1.09	0.93	0.98
Cerebrovascular	1.74	2.54	0.71	0.98	0.97	1.31	0.94	1.42	1.56	2.31
R. Circulatory	1.06	1.52	1.54	2.03	1.34	1.61	1.27	2.13	1.12	1.72
Respiratory	1.11	0.89	0.80	0.75	0.76	0.79	1.53	1.11	2.01	2.04
Digestive	0.81	0.51	0.70	0.58	0.44	0.42	0.76	0.61	0.60	0.53
External	1.88	0.66	1.88	1.06	1.32	0.65	1.47	0.57	1.71	0.94
Remaining	3.24	3.36	2.98	3.77	1.91	2.27	2.26	3.03	1.52	2.30

* Due to data availability, corresponds to year 2003

Source: HMD 2014, own elaboration

Likewise as shown for the year 2000, in 2010 are still neoplasms that influence the most overall life expectancy at birth and diseases of the digestive system the least. Respiratory system diseases also present a significant impact when compared with the obtained values in the year 2000. Nevertheless, in these 10 years of observation the group of CODs that includes all than the specified here, is

increasing its negative impact. This situation reinforces the need to identify “emerging” diseases that may influence negatively overall life expectancy.

Table 2.7: Life expectancy at birth in the absence of a certain COD, 2010

COD	Portugal		France		Sweden		Spain		Japan	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Neoplasms	3.98	2.83	5.13	3.85	3.39	3.43	4.82	3.23	4.59	3.90
Ischaemic	0.91	0.75	1.00	0.72	2.16	1.61	1.30	0.96	0.87	0.79
Cerebrovascular	1.28	1.79	0.63	0.86	0.77	1.01	0.78	1.11	1.19	1.53
R. Circulatory	1.01	1.52	1.59	2.16	1.45	1.77	1.37	2.23	1.23	2.05
Respiratory	1.28	1.19	0.82	0.76	0.66	0.64	1.45	1.09	2.19	2.09
Digestive	0.74	0.50	0.69	0.57	0.45	0.38	0.72	0.60	0.56	0.52
External	1.20	0.55	1.65	0.91	1.31	0.66	0.93	0.45	1.56	0.91
Remaining	3.44	3.73	3.55	4.88	2.53	3.25	2.61	3.85	1.76	3.03

Source: HMD 2014, own elaboration

2.6. Discussion

As a result, obtained results in this chapter confirm what has been concluded in many other previous studies: neoplasms, diseases of the circulatory and respiratory system are the principal CODs and the ones that influence the most, negatively, life expectancy for populations. However, in this chapter, with the calculation of alternative measures of longevity and presentation of different points of view, we perspective that if the rate of increase in life expectancy at birth keeps recent evolutionary patterns, it is possible that the male/female gap keep diminishing and all analyzed countries can aim for Japanese values.

Usually, less attention is given to causes that are less expressive in overall mortality, however, in our study we identify that this group as being one of the most important in influencing negatively life expectancy.

Likewise Oliveira and Mendes acknowledged (2010) the emergent importance associated with this wide-ranging group of CODs might be intimately related with shift of high rates of mortality to older ages.

2.7. Conclusions

Together with the shift of higher rates of mortality to older ages with time, changes in the CODs that are influencing those deaths are also being registered. Decision makers should not neglect less representative CODs and be prepared to, not new, but emergent CODs that were not related with mortality rates registered at younger ages.

Nonetheless, it is also very important to identify how different CODs may influence overall slopes of mortality, either for individuals and populations, evaluating how those can be differentiated and if they contribute to each other. That will be done on the next two chapters.

CHAPTER 3

THE INDIVIDUAL RATE OF AGING BY CAUSES OF DEATH: TESTING VAUPEL'S HYPOTHESIS

3.1. Introduction

Population's longevity is being affected, across history, by changes in the curve of age-specific mortality. Changes in the slope are often designated by *rate of aging* (e.g.: Vaupel, 2010; Zarulli, 2012) or *rate of senescence* (e.g.: Comfort, 1964; Rozing and Westendorp, 2008). In both, the general concept is rather similar. In this research, the term *rate of aging* is the choice over rate of senescence.

Vaupel (2010) hypothesizes that “*except for individuals with accelerated aging disorders, all other humans have a similar and perhaps, essentially the same, rate of increase in mortality with age*”, i.e., the same rate of aging, which might be a biological constant invariant across humans and over time.

In opposition with the rate at which age-specific death rates increase, known as the life-table aging rate (LAR) (Horiuchi and Coale, 1990), the individual rate of aging is defined as the relative derivative of the baseline hazard of death if the aging process is captured by a Gompertz curve. If the first is constant across age, the second presents a well-defined and characteristic bell-shaped pattern, widely studied since Gompertz (1825), and consequently finding evidence for a mortality deceleration at older ages. The force of mortality for an entire population is described by the associated age-specific death rates, resulting from a “contribution” of different subpopulations under diverse specific mortality conditions (Beard, 1959; Vaupel et al., 1979). Like it was suggested by Vaupel and Yashin in 1985, and well illustrated by the gamma-distributed frailty model with exponentially increasing individual mortality, i.e., the widely known gamma-Gompertz or gamma-Gompertz-Makeham model, individual and population mortality patterns might be qualitatively distinct. In their study, Vaupel and Yashin (1985) explored in a variety ways, that the pattern of attrition for the population as a whole can differ from the patterns for subpopulations or individuals, and in 2006, the same authors, pointed out 3 key reasons to justify the application of frailty models: (1) Cox regression yields coefficient estimations that

tend to be biased towards zero; (2) frailty models permit the estimation of underlying (baseline) hazards (i.e., the hazards that control the trajectory of risks at the individual level); and, (3) the possibility of using supplementary vital statistics in the analysis. Within this framework, individual mortality is characterized by different starting levels, which combined, result in a S-shaped logistic mortality curve for the population (Beard, 1959).

If Vaupel's hypothesis is confirmed, the individual force of mortality, from senescent causes, is characterized by 1) a constant and essentially identical rate of aging across individuals that share the same subpopulation, in a given cohort; and 2) for all subpopulations combined across different cohorts.

Testing this hypothesis is not easy, though. Data for complete cohorts becomes only available when all the individuals that share the same birth cohort leave the population by death. Still, data available for complete cohorts correspond to beginning of the 20th century, while for more ancient cohorts data quality is questionable (HMD, 2014; Wilmoth et al., 2007). Besides the issues about data convenience and quality, constant reductions in the age-specific mortality rates across time for countries considered to provide high-quality data, mainly after World War II (Tuljapurkar et al., 2000; Rau et al., 2008), subject individuals born in different cohorts with age x in year y to lower mortality rates than other individuals that attained the same age on previous years. This results in the underestimation of the rate of individual aging due to the rate of mortality improvements, making challenging to test point 2). Salinari and Santis (2014), based on Human Mortality Database data (HMD, 2014), find no evidence of a common rate of individual aging, but the direct association of mortality improvements and the rate of individual aging might be an explanation.

If in modeling non-human organisms there are several examples of scientific experiments where analyses of the effects of environmental and genetic experiments are conducted, in the case of humans this is rather difficult and profoundly censured. To avoid this, a way to analyze the validity of Vaupel's hypothesis is to analyze “natural” mortality shocks (occurred in different subpopulations) to achieve a better understanding about the changes in human mortality curves (Zarulli, 2012). Till now, the only piece of research addressed to this context comes from Zarulli (2012). The author took advantage of “natural mortality experiments” and achieved the same

mortality shifts in the age mortality trajectories of humans, finding that among Australian prisoners-of-war (in Java, during WWII) obtained estimates speak in favor of a constant rate of individual aging, but not in the case of Ukrainian famine. Nevertheless, in some cases, the conduction of experimental analyses with diverse non-organisms resulted in changes in the slope of the mortality trajectory (de Magalhães et al., 2005; Johnson, 1990) and others have found a parallel shift in the age of mortality trajectories when compared with the control group (Lin et al., 1998; Flurkey et al., 2001; Mair et al., 2003).

In this chapter, Vaupel's hypothesis is tested by studying major groups of cause of death (COD), addressed here as subpopulations. Data aggregation in major groups of CODs provides subpopulation sizes large enough to produce a significant statistical analysis. Period data, distinguishable by country and age is used to estimate the individual rate of aging, by fitting a gamma-Gompertz-Makeham [GGM] model, and seeking evidence to confirm or refute the existence of a constant rate of individual aging over time, and if, the obtained estimates accordingly each subpopulation, i.e., COD group, presents essentially the same rate of individual aging.

3.2. Methods

3.2.1. The gamma-Gompertz-Makeham frailty model

Generally, demographers view populations as a heterogeneous mixture of individuals, sharing, at adult ages, the same baseline hazard of death. This means that each population is composed by different subpopulations where, despite sharing the same hazard of death, all individuals are susceptible to in a different and random way. To estimate cause-specific individual rates of aging we fit a gamma-Gompertz-Makeham (GGM) frailty model, where the baseline hazard of death $\mu(x)$ at age x , is assumed to follow a Gompertz-Makeham mortality pattern (Gompertz, 1825; Makeham, 1860):

$$\mu(x) = ae^{bx} + c \quad (3.1)$$

where the aging process is captured by b , obtained by the relative derivative of ae^{bx} , i.e., the Gompertz part of the equation, a refers to the starting level of mortality for a

given adult age (where $x = 0$) and c corresponds to non-senescent mortality. Previous research revealed that individual susceptibility could be reflected in both parameters of the model presented in (3.1) at the same time; only on the mortality level at the starting age given by parameter a , applying relative-risk models; or on the individual rate of mortality increase, here given by b , applying accelerated-life models. Nevertheless, Gampe (2010), in her research, found evidence for a human mortality plateau at older age mortality when realized that mortality rates are leveling off beyond age 110, being favorable to the relative risks (Missov and Vaupel, 2015), while that accelerated-life mortality models would result in an eventual vanishing hazard of death (Finkelstein and Esaulova, 2006).

So, letting a random variable *frailty* capture individual susceptibility to death, in a relative-risk model framework with Gompertz-Makeham baseline mortality, the force of mortality $\mu(x | z)$ for individuals with frailty z is given by:

$$\mu(x | z) = z ae^{bx} + c. \quad (3.2)$$

If we assume frailty as being gamma-distributed with a unit mean and γ variance at the starting age of analysis, which is an additional standard (Beard, 1959; Vaupel et al., 1979) already proved theoretically (Steinsaltz and Wachter, 2006; Missov and Finkelstein, 2011), we end-up with a *gamma-Gompertz-Makeham* (Γ GM) *frailty model*. Thus, within a Γ GM theoretical framework, the hazard for the population is given by (Vaupel et al., 1979; Vaupel, 2002; Vaupel and Missov, 2014):

$$\bar{\mu}(x) = ae^{bx}[\bar{s}(x)]^\gamma + c. \quad (3.3)$$

where, a , b and c maintain the same meaning explained before, \bar{s} corresponds to the population survivorship at age x and γ is the frailty variance at the initial age of analysis. The inclusion of the Makeham term c is fundamental to obtain non-biased estimates since it is statistically significant and its exclusion of (3.3) may interfere negatively with the obtained estimates for the other parameters in the model (Missov and Nemeth, 2014).

Like it was stated before, considering the model in (3.3), the individual rate of aging is captured by the relative derivative of the Gompertz part ae^{bx} , but in

opposition, the relative derivative of $\bar{\mu}(x)$ results in the obtainment of the population rate of aging. If the first is constant across age, corresponding to b in (3.3), the second, proposed by Horiuchi and Coale in 1990 and addressed later as the *life-table aging rate* (LAR), varies age-wise presenting a characteristic bell-shaped pattern (Horiuchi and Coale, 1990; Horiuchi and Wilmoth, 1997, 1998; Vaupel and Zhang, 2010). However, b is only well captured under this theoretical framework, if and only if the baseline hazard $\mu(x)$ follows a Gompertz pattern. In opposition to the individual's rate of aging, LAR has been widely studied, either for human populations (e.g., Horiuchi and Wilmoth, 1997, 1998) or non-human (e.g., Carey and Liedo, 1998), which might have caused some misperception about the relationship between both measures. In 2010, Vaupel and Zhang, explored an explicit relationship between the individual rate of mortality increase and the population rate of aging, proving that, not only b differs from LAR, but also contributes to the estimation of the second.

3.2.2. Model fitting

Despite model (3.3) is proposed for cohorts, in this chapter, we use data collected for different CODs by single years y . Consequently, due to data availability, to capture overall mortality (Vaupel, 2002; Vaupel and Missov, 2014) we apply:

$$\bar{\mu}(x, y) = a(y)e^{b(y)x}[\bar{s}^{(c)}(x)]^{\gamma(y)} + c(y) \quad (3.4)$$

where $a(y)$, $b(y)$ and $c(y)$ correspond to estimates at the period level and $\bar{s}^{(c)}$ to survivorship for the subpopulation born in year $y - x$.

Every individual in overall population or across different subpopulations is continuously exposed to different risks of death. “*Because death is not a repetitive event and is usually attributed to a single cause, these risks compete with one another for the life of a person. Competing risks must be considered in any cause-specific mortality analysis*” (Chiang, 1991). In our approach, we incorporate this statement, assuming that the individuals exposed to the risk of death by COD are the same that are exposed to overall mortality: $E_i(x, y) = E(x, y)$. Model (3.4), when applied to different COD_i is characterized by a unique starting level of mortality $a_i(y)$, $b_i(y)$,

$\gamma_i(y)$, $c_i(y)$ and a common survivorship by cause $\bar{s}_i^{(c)}(x)$ and cohort, however, due to data availability, period survival $\bar{s}_i(x, y)$ is used instead:

$$\bar{\mu}_i(x, y) = a_i(y)e^{b_i(y)x}[\bar{s}_i(x, y)]^{\gamma_i(y)} + c_i(y) \quad (3.5)$$

Looking at each COD_i separately may influence obtained results, mainly due to the competing risks to which individuals are exposed. Thus, if we discriminate cohort survivorship by COD_i , it may still exclude competing risks from our analysis. However, assuming that $\bar{s}_i^{(c)}(x) = \bar{s}^{(c)}(x)$, additionally to common exposures and a single frailty term with variance $\gamma(y)$ at the starting age of analysis, we can incorporate again the competing risks into the model:

$$\bar{\mu}_i(x, y) = a_i(y)e^{b_i(y)x}[\bar{s}^{(c)}(x)]^{\gamma(y)} + c_i(y). \quad (3.6)$$

Applying model in (3.5) to our data results in the estimation of 4 parameters at a time by period y . In opposition, the outcome from the model presented in (3.6) results in 25 parameters at a time y , leading to a very unstable optimization procedure highly manipulated by chosen starting values. As a result, and due to very complete information about cohort survival concerning overall mortality available at HMD, a model with differentiated γ_i is also fitted:

$$\bar{\mu}_i(x, y) = a_i(y)e^{b_i(y)x}[\bar{s}^{(c)}(x)]^{\gamma_i(y)} + c_i(y). \quad (3.7)$$

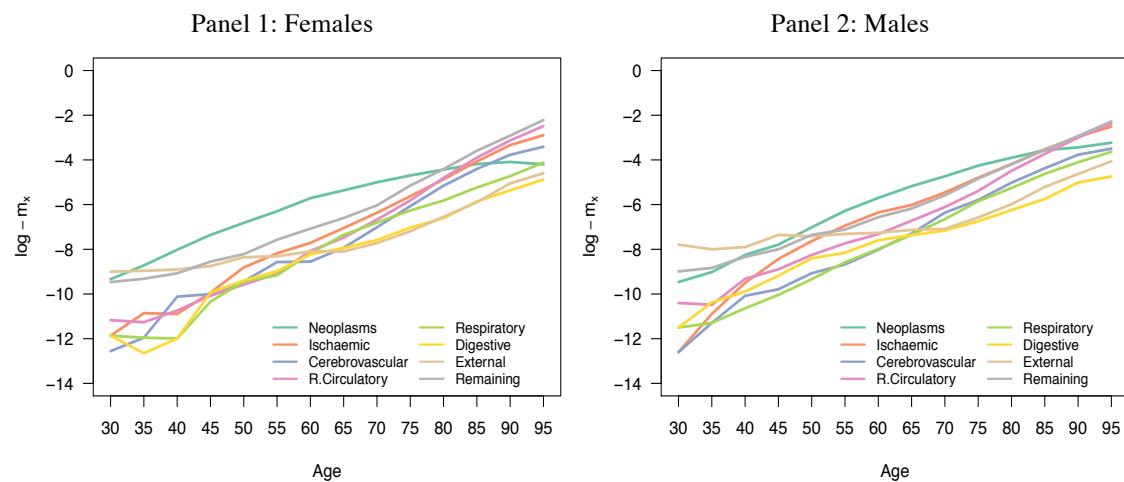
Comparing obtained results in (3.5) with the obtained in (3.7) leads to almost identical parameter estimates, thus, as a results, only the estimates from (3.7) will be seen in the *results* section when the obtained estimates for rate of individual aging are presented.

Introduced the model framework, fitting procedure holds on the assumption that $D_i(x, y) \sim Poisson(E(x, y) * \bar{\mu}_i(x, y))$ (Brillinger, 1986), where $D_i(x, y)$ denote the cause-specific death counts i , and $E(x, y)$ denote the age specific exposures, both in year y . Thus, for each COD_i separately in year y we maximize a Poisson log-likelihood:

$$\ln L(a_i(y), b_i(y), \gamma_i(y), c_i(y)) = \sum_x [D_i(x, y) * \ln \bar{\mu}_i(x, y) - E(x, y) * \bar{\mu}_i(x, y)] \quad (3.8)$$

The same procedure is adopted to estimate the parameters for all causes combined (when $i = \emptyset$, we refer to (3.4)). Analyzing Figure 3.3, where are presented the observed age-specific mortality rates for Sweden (2010) by COD_i , we can conclude that after age 65, i.e., after age group 65-69, that mortality patterns associated with different major CODs register an approximated exponential increase, followed, in some cases, by an eventual deceleration pattern. Thus, all mentioned models are fitted starting at age group 65-69, because fitting a GGM model at earlier ages will result in meaningless $b_i(y)$ -estimates for CODs that do not seem to follow a GGM curve. Nevertheless, for some specific causes, like e.g. neoplasms, the fitting procedure might need to start earlier once that major incidence for cancer is concentrated at younger ages, being reflected in the age-specific mortality pattern after age 65. However, that strategy needs to be well defined because each cause is singular, so, in this chapter, and for comparison proposes, all estimates are performed starting at age 65.

Figure 3.3: Observed age-specific mortality rates after age 30 in Sweden, 2010



Source: WHOMD and HMD 2014, own elaboration

3.3. Data

3.3.1. Source, classification and concepts of cause of death data

The *International Classification of Diseases* (ICD) regulates the classification of COD data, which is revised periodically to allow the inclusion of new diseases and/or the refinement in the identification of diseases and promotes the international comparability of COD statistics. However, new classifications that result from ICD revisions are inevitably followed by the impossibility (for less aggregated groups of causes, e.g., *lung cancer* or *poisoning*) of making comparisons over time and across countries since different countries may adopt the new classifications at different times. Still, more refined the classification becomes, the greater the need for expert clinical diagnosis of cause of death.

National mortality data derives essentially from death certificates, from which diverse information can be used. In death certificates it can be found information about the *underlying cause of death*, i.e., the disease or injury that initiated the train of events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury (WHO, 1977). Death certificates also provide information that gives the possibility to elaborate a more profound and complete analysis, containing information about other contributing causes or identifying what was the *immediate cause of death*, which is the final disease or condition that results in death. This means that an underlying COD can trigger a different disease and only after, death, being this last disease the immediate cause. Exemplifying, imagine a deceased that died from a coronary occlusion. Despite the immediate COD be the coronary occlusion, the underlying COD can be different, like diabetes.

Regardless to the underlying or immediate causes of death, it is also important to define the *external cause of death* category, where the recorded cause is the external cause that resulted in death, not the nature of the injury or the antecedent condition of the deceased. Death is frequently produced by more than one cause and it is also possible, for a restrict number of countries, to have access to statistics on *multiple causes of death*, presenting more detailed information recorded in the death certificates, that can be seen as a recognition about the involvement of combinations of diseases and conditions in many deaths.

3.3.2. Validity and viability of data

COD data is very often facing reliability problems, produced mainly by 1) the lack of accuracy in death certificates, or 2) by changes in the classification system (Meslé, 2006), and to avoid this uncertainty, the World Health Organization (WHO) recommends to base COD statistics on the *underlying* cause of death category.

Some studies were conducted with the intention of comparing death certificates and *post-mortem* diagnosis and the most part of the results showed that exists a significant disagreement between them (Modelmog et. al., 1992). This situation can occur, for example, when physicians or other medical professionals are unfamiliar with the true cause of death (Lilienfeld et. al., 1994). Some interrelated illness can be also the explanation for this uncertainty, such as diabetes together with coronary disease, which are two diseases feasible to be fatal. On the other hand, some diseases can be only the contributing COD, what can difficult to identify the real cause. From the combination of all these difficulties can result an impossibility to identify the COD of a deceased and the best option is to consider autopsy results combined with clinical data (Kircher et. al., 1985), however not every deceased is autopsied.

In what concerns to cancer more precisely, different studies where information from death certificates was crossed with autopsy information, presented really positive results when this disease is the underlying COD. It was shown that the sensitivity of death certificates varies between 87 % and 93 % and the predictive capacity between 85 % and 96 % (Kircher et. al., 1985; Engel et. al., 1980; Schottenfeld et. al., 1982).

German et. al. (2011) evaluated the accuracy of cancer statistics in the U.S.A., based on the ICD 9 and ICD 10 classifications, and with a 95 % confidence interval, they achieved a confirmation rate that varies between 80.4 % and 81.6 % for the number of deaths classified under the 10th International Classification of Diseases. The confirmation rate for the deaths recorded under the 9th International Classification of Diseases was even a little higher, varying between 84.8 % and 85.2 %.

Nevertheless, once that is difficult to have access to *microdata* like the *post-mortem* diagnosis for each individual, one option that is often used in this kind of analysis is the calculation of the proportion of death coded in the *ill-defined and unspecified* causes of death category (Meslé, 2006) in order to evaluate the data.

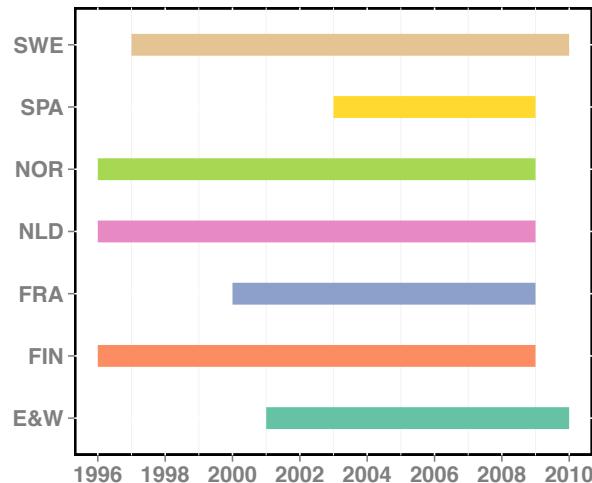
3.3.3. Data selection and availability

As already exposed, data collection can face many problems that could influence their validity and viability, however, and besides some of the enumerated problems that COD classification may originate, data availability is also a concerning issue. In some countries, the distribution of data categorized by COD can be considered an ethical problem, and only major causes of death are available. Besides that, it is also almost impossible to find data disaggregated by single-year age groups, so it is very common to deal with data aggregated in five-year age groups, in which the highest open age group starts mainly at age 85. Sometimes, though, it can be also possible to find data where the open age interval starts at age 95 or 100.

Likewise it was done on Chapter 2, data on *overall death counts* $D(x, y)$ and *exposures* $E(x, y)$ derived from the *Human Mortality Database* (HMD, 2014: www.mortality.org) and *deaths by COD* $D_i(x, y)$ from the *World Health Organization Mortality Database* (WHOMD, 2014).

Again, WHOMD cause of death (COD) data are only available by five-year age groups and once that we extract death counts according to ICD10, our timeline in what concerns to COD is restricted essentially to the last available decade (depending on the country; see Figure 3.1). Data codification corresponds to the ICD 10 detailed 3rd and 4th character list classification and was rearranged in 8 main COD groups: 1) Neoplasms, 2) Ischaemic Heart Diseases; 3) Cerebrovascular Diseases; 4) Remaining Diseases of the Circulatory System; 5) Diseases of the Respiratory System; 6) Diseases of the Digestive System; 7) External Causes of death; and 8) Remaining Causes of death. Following the WHO recommendation (1977) to avoid uncertainty that comes from changes and updates in the classification system, we base our research on the *underlying cause of death*, i.e., the disease or injury, which initiated the series of events that lead to death. It also should be said that, for all selected countries, the ill-defined COD group corresponds to less than 3.5 % of all deaths after age 65 and across the study period. Following Meslé (2006), this is an indicator of good overall data quality.

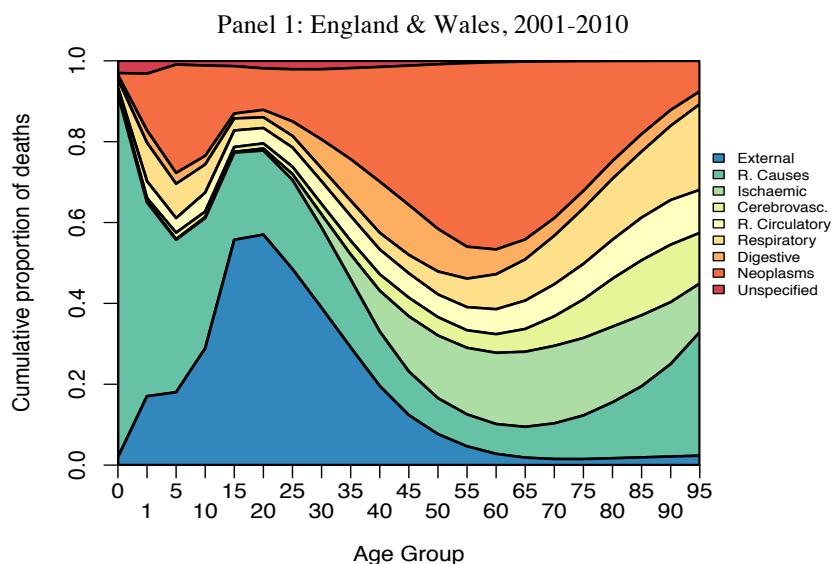
Figure 3.1: List of countries and years available for ICD10 cause of death classification

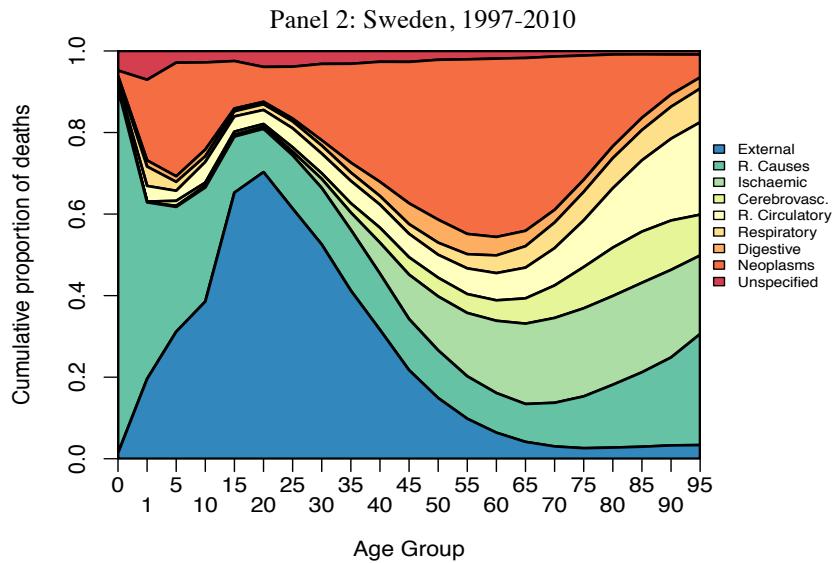


Source: WHOMD 2014

Figure 3.2, exemplifies the typical distribution of age-specific deaths by COD, from which we can realize that external causes of death, as well as diseases of the digestive system contribute the least beyond age 65. This situation results in relatively small corresponding $D_i(x, y)$, especially for Sweden, Finland, Norway and the Netherlands.

Figure 3.2: Proportion of age-specific deaths by cause





Source: WHOMD 2014, own elaboration

3.3.4. Data limitations

When we decide to look to CODs as subpopulations and estimate the rate of individual aging, two major difficulties emerge. First of all, COD information desegregated by cohort is not available, restricting our analysis by period. However, previous research found evidence that the actual mortality experience of a preceding cohort is well corresponded by period mortality, as it was discussed, e.g., in Bongaarts and Feeney (2003) or in Rodriguez (2006). This means that with our approach we are also estimating the rate of individual aging for some earlier cohorts.

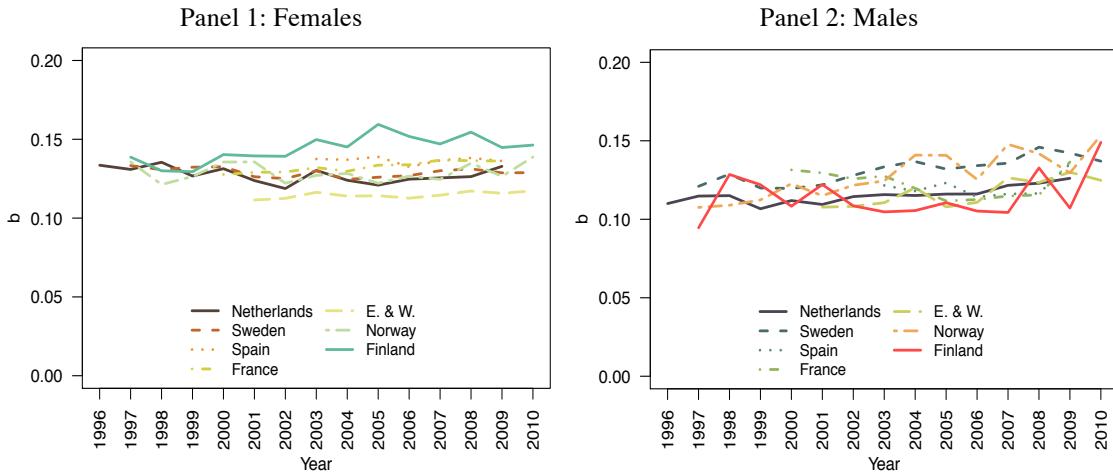
Secondly, changes in the ICD classification system and the necessity of cohort survivorship reconstructed by a given year results in the analysis of a shorter period of time. Nevertheless, when longer periods become available under the same classification, the adopted methodological approach can be still extended.

3.4. Results

The rate of individual aging, following what was explained in previous sections, was estimated for all the seven selected countries (Sweden, Spain, France, Norway, Netherlands, Finland and England and Wales), males and females separately, in each

available year (Figure 3.1), by fitting a Γ GM model starting at the age group 65-69. The obtained results corresponding to the overall mortality $\hat{b}(y)$ are presented in Figure 3.4, and, as it can be seen, especially in the female case, estimates seem relatively stable across the different analyzed countries.

Figure 3.4: Estimated $\hat{b}(y)$ by country and gender for major CODs group



Source: WHOMD and HMD 2014, own elaboration

A closer look, given by Table 3.1, where the obtained results for England and Wales are presented, clearly exemplifies what was just been said. Obtained estimates show that in ten singular years of observation, $\hat{b}(y)$ only increased 0.006 (between 2001 and 2010) in the female case and 0.017 for males, being the associated standard errors smaller in the female case than for males. However, despite the observed differences, a narrow gap for confidence intervals is obtained, and that might be explained by the fact that England and Wales provide a larger subpopulation of analysis. Still, the known fact that females live longer than males, can also affect the subpopulation size and correspondent estimates.

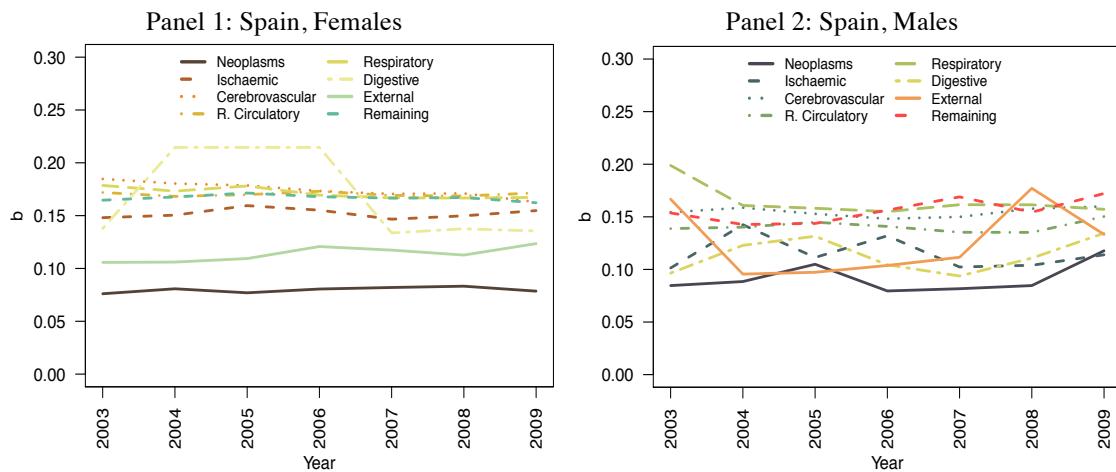
Table 3.1: Estimates of $\hat{b}(y)$ for overall mortality for England and Wales with associated standard errors

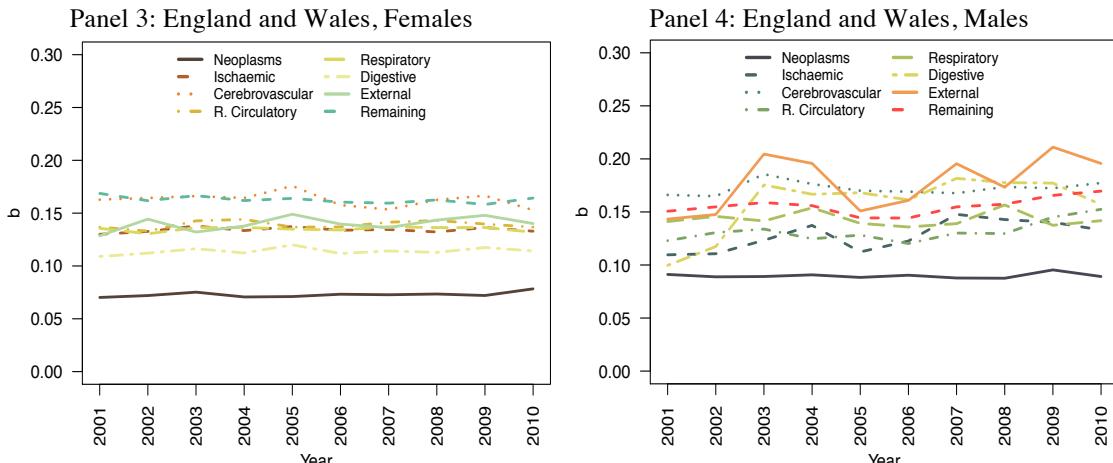
Year	Females		Males	
	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$
2001	0.111	0.0019	0.108	0.0033
2002	0.113	0.0018	0.108	0.0035
2003	0.116	0.0018	0.111	0.0038
2004	0.114	0.0018	0.120	0.0040
2005	0.114	0.0017	0.108	0.0041
2006	0.113	0.0016	0.111	0.0036
2007	0.115	0.0016	0.126	0.0034
2008	0.117	0.0015	0.123	0.0034
2009	0.116	0.0015	0.130	0.0033
2010	0.117	0.0014	0.125	0.0034

Source: WHOMD and HMD 2014, own elaboration

Figure 3.5, reveals that the $\hat{b}_i(y)$ estimates for the rate of individual aging differentiated by COD_i follow a similar pattern across time, with exception for some cases with lower subpopulation size, like the *digestive system diseases* in Spain for females, or in the overall male case. Here we already can realize that, despite the existence between the obtained results, of dissimilar estimates, they seem to be reasonably constant over time.

Figure 3.5: Estimated $\hat{b}_i(y)$ by COD_i for Spain and England and Wales





Source: WHOMD and HMD 2014, own elaboration

Despite Figure 3.5 displays a very similar level across the presented estimates, in Table 3.2, we can realize that the gender differences calculated based on the average rates of individual aging over the country-specific available periods seem to be negligible in some cases (countries and CODs). In some other situations, the difference is at least 0.01, what at this magnitude, is considered to be significant.

Generally, the obtained estimates vary from an average of 0.115, registered in Finland and Netherlands, to the Swedish 0.131, for males; while for females varies from the same 0.115, registered in England and Wales, to 0.144 from Finland. From these results, can be also recognized that $\hat{b}(y)$ is constantly overestimated for females in comparison with males, but a possible explanation can be advanced by the gender-gap in the associated mortality rates for both sexes, that result, inevitably, in different estimates for the other corresponding parameters $\hat{a}(y)$, $\hat{c}(y)$ and $\hat{\gamma}(y)$.

Table 3.2: Average country-specific individual rate of aging

	Sex	All	Neo.	Isc.	Cer.	R.Cir.	Resp.	Dig.	Ext.	Rem.
SWE	F	0.129	0.069	0.148	0.174	0.179	0.131	0.132	0.152	0.162
	M	0.130	0.095	0.137	0.171	0.182	0.156	0.172	0.223	0.168
SPA	F	0.137	0.080	0.152	0.175	0.170	0.171	0.170	0.114	0.167
	M	0.121	0.092	0.115	0.155	0.141	0.165	0.113	0.126	0.156
FRA	F	0.132	0.067	0.147	0.150	0.161	0.156	0.125	0.145	0.152
	M	0.123	0.069	0.131	0.156	0.155	0.140	0.136	0.152	0.165
NED	F	0.128	0.065	0.136	0.175	0.156	0.146	0.149	0.162	0.162
	M	0.115	0.091	0.123	0.167	0.133	0.179	0.152	0.196	0.155
FIN	F	0.144	0.080	0.178	0.179	0.169	0.160	0.161	0.175	0.170
	M	0.115	0.093	0.128	0.156	0.136	0.147	0.207	0.254	0.177
NOR	F	0.129	0.065	0.157	0.184	0.178	0.154	0.147	0.171	0.152
	M	0.128	0.095	0.145	0.180	0.169	0.163	0.150	0.191	0.156
E & W	F	0.115	0.073	0.134	0.163	0.139	0.135	0.114	0.140	0.163
	M	0.117	0.090	0.124	0.172	0.132	0.143	0.158	0.178	0.156

Source: WHOMD and HMD 2014, own calculation

What has just been exposed is clearly exemplified in Table 3.3, where the Spanish example for overall mortality is presented.

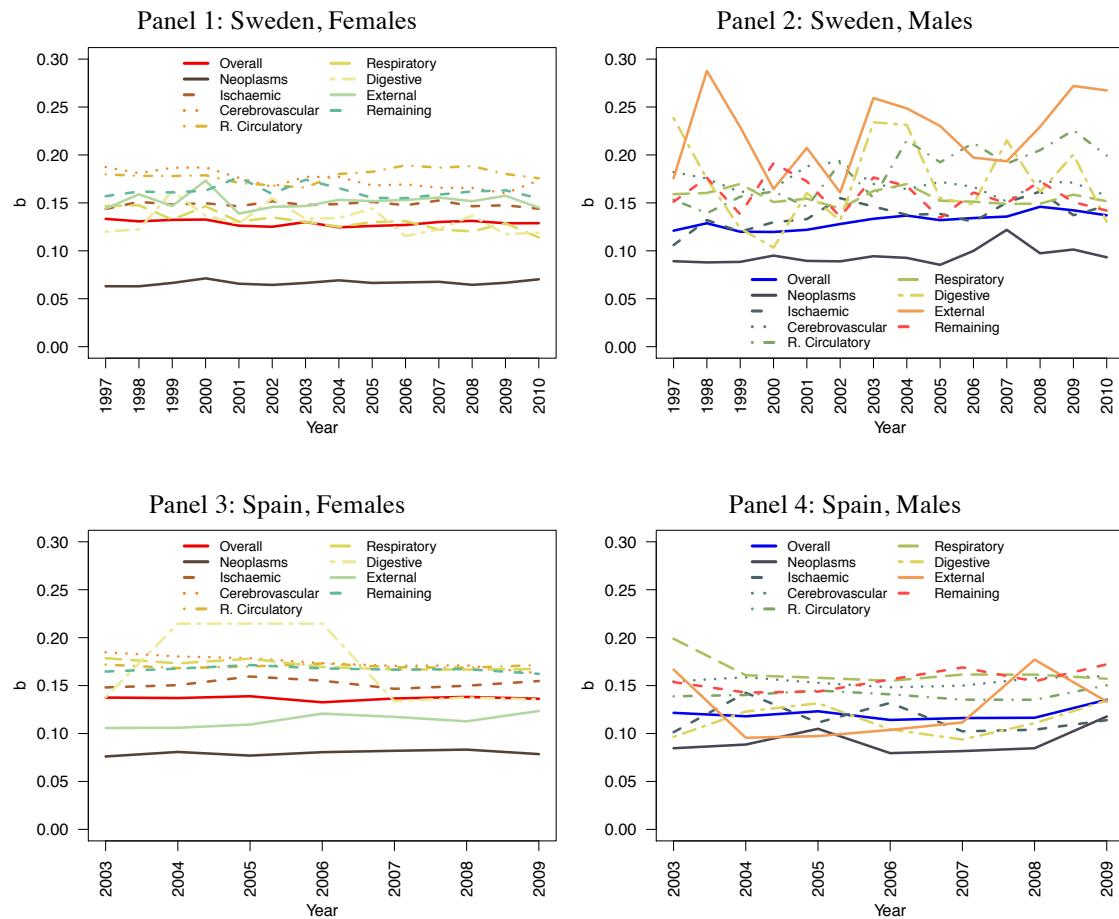
Table 3.3: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for overall mortality in Spain

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0.00757	0.00021	0.137	0.0021	0.132	0.0231	0.00000012	0.00050474
2004	0.00714	0.00020	0.137	0.0021	0.147	0.0235	0.00000021	0.00037330
2005	0.00707	0.00019	0.139	0.0020	0.141	0.0225	0.00000000	0.00263809
2006	0.00687	0.00018	0.133	0.0019	0.047	0.0214	0.00000001	0.00167468
2007	0.00664	0.00016	0.137	0.0017	0.096	0.0208	0.00000006	0.00055173
2008	0.00636	0.00015	0.138	0.0016	0.102	0.0206	0.00000007	0.00048874
2009	0.00623	0.00014	0.136	0.0015	0.064	0.0192	0.00000003	0.00073094
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0.01492	0.00077	0.122	0.0040	0.140	0.0291	0.00469955	0.00084663
2004	0.01500	0.00078	0.118	0.0040	0.130	0.0289	0.00338730	0.00084960
2005	0.01427	0.00071	0.123	0.0038	0.165	0.0283	0.00415971	0.00078600
2006	0.01448	0.00083	0.114	0.0042	0.088	0.0311	0.00285396	0.00089437
2007	0.01478	0.00065	0.116	0.0033	0.123	0.0254	0.00215482	0.00070332
2008	0.01419	0.00063	0.116	0.0033	0.117	0.0261	0.00190371	0.00068821
2009	0.01080	0.00053	0.135	0.0038	0.269	0.0314	0.00557874	0.00059131

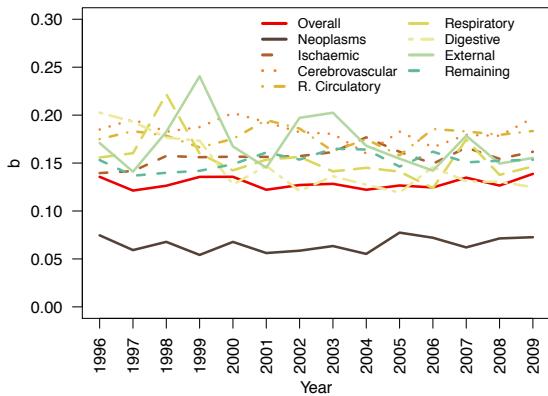
Source: WHOMD and HMD 2014, own elaboration

Focusing now again on the estimated rates of individual aging by COD (Figure 3.6), we can distinguish three main patterns, 1) the obtained estimates for neoplasms $\hat{b}_i(y)$ seem to be always lower than the registered for overall mortality $\hat{b}(y)$; 2) the estimates for overall mortality are in agreement with the ones obtained for ischaemic heart diseases, being very similar; and lastly, 3) all other CODs show an associated $\hat{b}_i(y)$ -estimate higher than $\hat{b}(y)$. Thus, we can conclude that neoplasms influence “negatively” the rate of individual aging estimated for overall mortality, once that the still observed high mortality rates at age 65 contributes to a lower overall $\hat{b}(y)$.

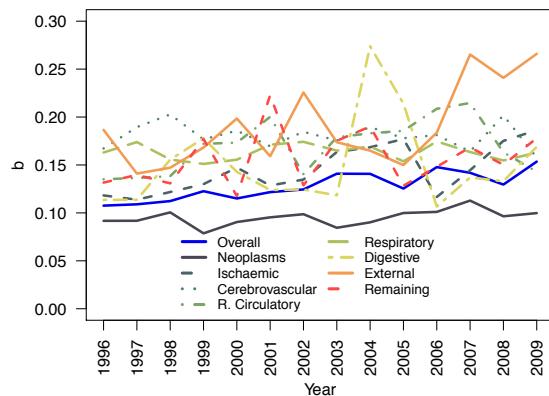
Figure 3.6: Estimated $\hat{b}(y)$ and $\hat{b}_i(y)$ by COD_i for all selected countries



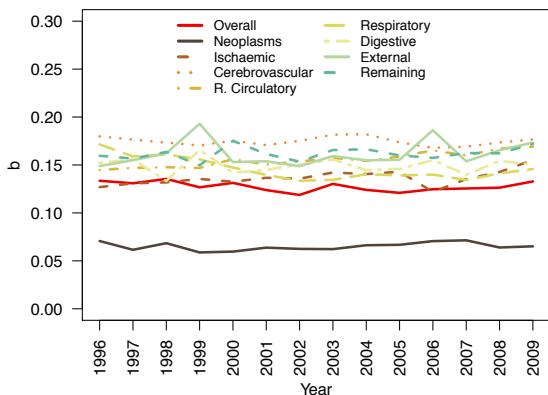
Panel 5: Norway, Females



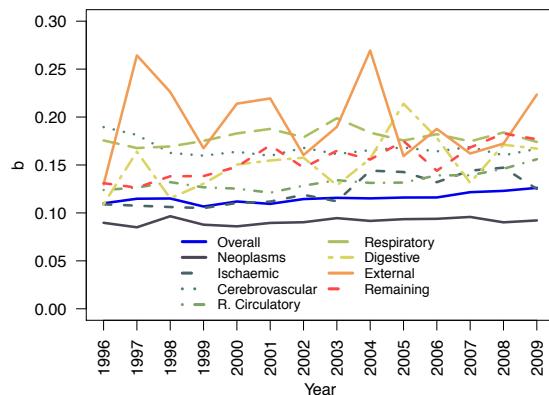
Panel 6: Norway, Males



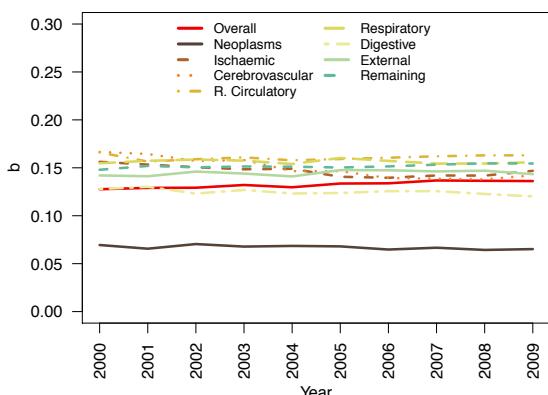
Panel 7: Netherlands, Females



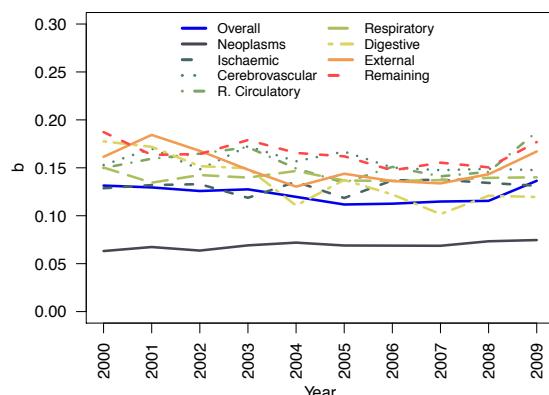
Panel 8: Netherlands, Males



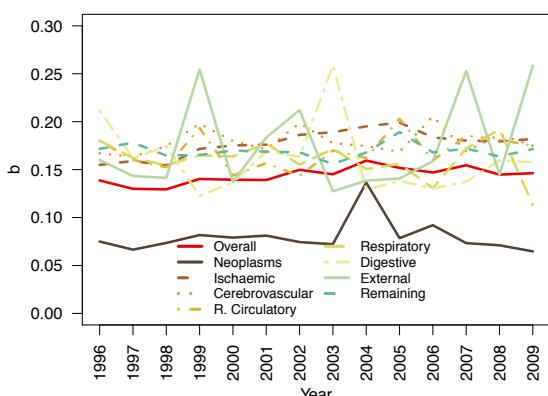
Panel 9: France, Females



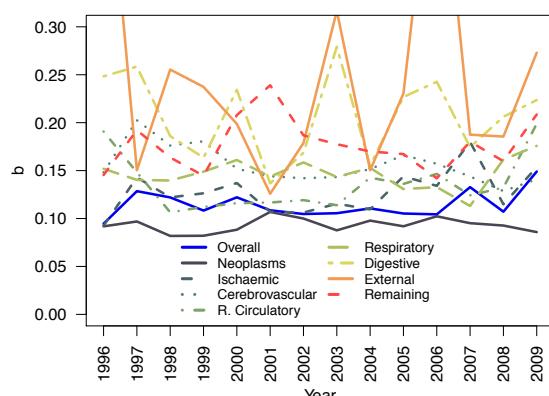
Panel 10: France, Males

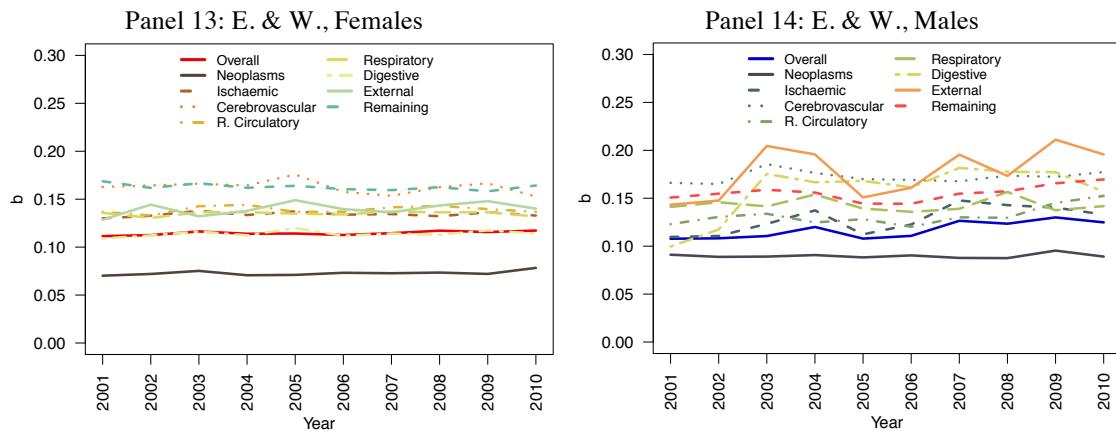


Panel 11: Finland, Females



Panel 12: Finland, Males





Source: WHOMD and HMD 2014, own elaboration

From the obtained results, we can observe that the COD that has most distinctive estimates for the rate of individual aging $\hat{b}_i(y)$ is neoplasms, to which is connected the lowest estimates. Nevertheless, some explanations might be advanced: 1) the incidence rate associated to neoplasms achieves its higher values before age 65, where mortality rates seem to decline around age 50/55; 2) following WHO (2014), around 30 % of all deaths related to neoplasms “*are due to five leading behavioral and dietary risks: high body mass index, low fruit and vegetable intake, lack of physical activity, tobacco use, alcohol use*”, and bring some uncertainty to cancer-related mortality patterns; 3) the stage of the diagnosis, that influences directly age-specific mortality rates, once that earlier diagnosis increases survival chances; and 4) a wide-ranging cause of death group, once that all types of neoplasms are concentrated in only one large group, and the overall observed pattern may not reflect directly all distinct neoplasms (for further discussion see Ukrainseva and Yashin, 2003).

Estimates obtained for causes with lower number of deaths, originating lower study subpopulations, present higher fluctuation patterns. Digestive system diseases and external causes of death are two good examples, but specifically the second, due to its strict connection with transport-accident. Country size and sex revealed also to be important, once that exists a higher number of females alive after age 65 and countries like Norway (5.1 M), Finland (5.4 M), Sweden (9.5 M) and Netherlands (16.8 M) have lower population size when compared with France (66 M), Spain (47.3 M) or England and Wales (57.9 M).

3.5. Discussion

Despite some criticism about focusing on the slope of age-specific mortality, in log-scale, to measure the rate of aging (Rozing and Westendorp, 2008) and some expositions about the *pros* and *contras* of this measure (de Gray, 2005), we believe that this approach is highly sustainable. Thus, and seeking to test Vaupel's hypothesis the individual rate of mortality increase was estimated under a ΓGM framework differentiating each COD as a subpopulation. Under this framework, we can also obtain the estimates for the population rate of aging, which in opposition to the individual's rate of aging, has been widely studied, either for human populations (e.g., Horiuchi and Wilmoth, 1997, 1998) or non-human (e.g., Carey and Liedo, 1998). These two measures not only are completely different, but also present very distinctive patterns, being the individual rate of aging constant across time, i.e., indicating how fast individuals in a population if the initial level of mortality stays unchanged and with no external influences (considered here with Makeham's c); and LAR varying age-wise, creating a bell-shaped pattern. Still, not only b differs from LAR, but also contributes to its estimation (Vaupel and Zhang, 2010).

Generally, the estimated rates of individual aging across different CODs, sexes and selected countries, perform a fairly stable evolution. Nevertheless, we cannot fully support Vaupel's hypothesis due the discrepancy between the obtained estimates by COD, but a major contribution for this topic is achieved. Our estimates also revealed that, with the exception of neoplasms, the estimates obtained for overall mortality are always lower when compared with different $\hat{b}_i(y)$. In this way, it seems that the different $\hat{b}_i(y)$ associated to different CODs appears to contribute to the “average” rate of individual aging, i.e., to the obtained estimates for overall mortality. Accordingly with Brody and Schneider (1986), our results present lower rates of aging for causes considered close to non-senescent processes, like neoplasms or external causes of death, and higher values for the ones in opposite direction, i.e., for example the case of the deaths caused by diseases of the circulatory system. The case of cancer strikes out as the cause that has the lowest rate of aging of all, factor that can be explained by the fact that “*in the human species, the population that reaches advanced age and has a decreased incidence of cancer, could be less prone to develop cancer and hence more fit to reach the maximum lifespan*” (Macieira-Coelho, 1986). Another explanation can be directly related with the origin of cancer, which is

connected with the unregulated growth of new cells, being a multistage process that starts with a pre-cancerous lesion and ends up with a malignant tumor. Nevertheless, at older ages the proliferation of cells is declining and possibly increases the time between cancer development stages (Ukrainsteva and Yashin, 2003).

As a result, many factors may have interfered with the obtained results: 1) model estimation employing aggregated data (5-year age groups); 2) data validity and viability; 3) the existence of competing risks to which each individual is exposed; and 4) the existence of a close relationship between behavior and some CODs as, e.g., neoplasms and external causes. Consequently, once that the obtained results cannot fulfill Vaupel's hypothesis (it only speaks in favor of a constant evolution across time), a possible complementary way is testing by the estimate of the individual rate of individual aging accordingly to groups of individuals that share the same behavioral pattern, e.g., smokers and non-smokers; athletes and non-athletes; or, alcohol consumption and non-consumption.

Summarizing, obtained estimates present more stable and less variation across time for countries with larger population sizes (Spain, France, and England and Wales), CODs with higher proportion of deaths beyond age 65, and in general for females, as consequence of a larger number of alive females on the analyzed age range.

3.6. Conclusions

Even with the obtained results showing that to each considered subpopulation it is obtained a different rate of individual aging, we cannot *completely* refute Vaupel's hypothesis. Nevertheless, we strongly believe that a major contribution in this direction was achieved. The constant rate of aging across all individuals, if proved, "*would fundamentally contribute to our understanding of how and why we age*" (Vaupel, 2010), and our findings are contributing, at least partially, on this direction. It seems that each cause of death contributes for the rate of aging for the overall population. In this way, we understand that, due to the different contributing risks to which all individuals are exposed (Chiang, 1991), it is more correct to test for example a group of individuals that died from one specific cause but that was exposed

to all the causes of death, e.g., smokers versus non-smokers, or people living with cancer versus people without this disease.

Still, the finding of an almost constant rate of aging across time for the causes less prone to human behavior suggests that besides different CODs are characterized by different rates of aging, it is possible that they share the same rate of aging within a subpopulation exposed to all competing risks. One example of a population exposed to all competing risks, that speaks in favor of a constant rate of individual aging is given by Zarulli (2012), where the author finds clear favorable evidences among the Australian prisoners in Java during WWII.

CHAPTER 4

REVISITING MORTALITY DECELERATION PATTERNS UNDER A GAMMA-GOMPERTZ-MAKEAHM FRAMEWORK

4.1. Introduction

Horiuchi and Coale (1990) proposed a mortality measure, designated later (Horiuchi and Wilmoth, 1997; Carey and Liedo, 1998) as the life-table aging rate (LAR), which captures the age-specific rate of mortality change for a given population $\bar{b}^*(x) = \ln(M(x)) - \ln(M(x-1))$. The rate of individual aging defined as the relative derivative of the baseline hazard of death from senescent causes, is a different characteristic, which is constant whenever the aging process is captured by a Gompertz curve (for further discussion on the rate of individual aging, see Missov and Vaupel 2015, Missov and Ribeiro 2015).

Gampe (2010) finds evidence for a leveling-off of human mortality at ages 110-114. If a mortality plateau exists, then it speaks in favor of a relative-risk model (Missov and Vaupel 2015) with a Gompertz-Makeham baseline $\mu(x) = ae^{bx} + c$ (Gompertz 1825; Makeham 1860), where a measures the mortality level at the starting adult age, b is the individual rate of aging itself, and c captures the risk of dying that is not associated with the aging process. Unobserved heterogeneity (frailty) can be captured by a gamma distribution with a unit mean and γ variance at the starting age (Vaupel et al. 1979, Missov and Finkelstein 2011, Missov and Vaupel 2015). This leads to the gamma-Gompertz-Makeham frailty model (Vaupel et al. 1979), which we will shortly address as the Γ GM. If we estimate its parameters a , b , c and γ , we can take advantage of the LAR representation by Vaupel and Zhang (2010) to estimate the population rate of aging (LAR).

Horiuchi, Cheung and Robine, 2012 focused on reconstructing model-based LARs by fitting a Kannisto model for homogeneous populations. In this chapter we focus on a Γ GM heterogeneous model to reflect the perception that populations consist of individuals that share the same baseline hazard, to which they are susceptible in a different (random) way. We also incorporate a Makeham term to account for possible (non-negligible) extrinsic mortality. If c is statistically significant

and is left out of the model, the estimates for the other Γ GM parameters will be biased (Missov and Nemeth 2014). As a result LAR will be also unspecified and it will be impossible to capture its bell-shaped pattern (Horiuchi and Coale 1990). This is perhaps one of the main reasons that Gavrilova and Gavrilov (2014) find no evidence of a deceleration pattern in the rate of population aging after age 80 in their simulation study. In the same paper, the empirical finding based on 24 single-year birth cohorts for four different countries (Canada, France, Sweden and the United States of America) using Human Mortality Database (2014) suggested, once more, that LAR does not change significantly with age. We believe that this results from the omission of the two-step “smoothing” procedure, essential to calculate LAR (Horiuchi and Coale, 1990).

In this chapter we first fit the Γ GM model to five countries: two from Southern Europe (Spain and Portugal), one from Western Europe (France), one from Northern Europe (Sweden), and one from Asia (Japan). Similarly to what was done in Chapter 2, our choice was based on the qualitatively different patterns of life-expectancy evolution over time. France and Sweden register high life expectancy at birth in the 1950s, but its rate of increase drops in the following decades. Spain, Portugal and Japan, on the other hand, experience a lower life expectancy at birth in the 1950s, but the rates of increase surpass the ones for the life-expectancy leaders at the time. As a result the three countries caught up: life expectancy in Spain even surpassed the one registered in Sweden, whereas Japan became the world leader best-practice country.

Second, we elaborate on the relationship between the estimated LARs and the rate of life expectancy increase in the chosen countries, as well as show how the estimated LARs reflect the age patterns of mortality deceleration – not only for the overall mortality, but also across causes of death (COD). At the same time we illustrate how well the LAR formula by Vaupel and Zhang (2010), which uses the estimated Γ GM parameters, fits the actual LAR.

Third, we test the “heterogeneity hypothesis” by Horiuchi and Wilmoth (1998), which states that a) deceleration occurs for the most major CODs, being less pronounced for CODs with lower death rates; and b) mortality deceleration should occur at later ages due to selection effects.

4.2. Data

Data on *overall death counts* $D(x, y)$ and *exposures* $E(x, y)$ derived from the *Human Mortality Database* (HMD, 2014: www.mortality.org) and *deaths by COD* $D_i(x, y)$ from the *World Health Organization Mortality Database* (WHOMD, 2014).

Like it was explained on the previous chapters, WHOMD cause of death (COD) data are only available by five-year age groups and once that we extract death counts according to ICD10, the timeline is restricted to the last available decade (depending on the country (see Table 4.1). We leave Portugal out of the analysis by COD as the last open-end age group is 85+ and not 95+ and that could interfere with obtained estimates. To avoid low subpopulation sizes and any lack of representativeness, we work with major COD groups: 1) Neoplasms, 2) Ischaemic Heart Diseases; 3) Cerebrovascular Diseases; 4) Remaining Diseases of the Circulatory System; 5) Diseases of the Respiratory System; 6) Diseases of the Digestive System; 7) External Causes of death; and 8) Remaining Causes of death. Following the WHO recommendation (1977) to avoid uncertainty that comes from changes and updates in the classification system, we base our research on the *underlying cause of death*, *i.e.*, the disease or injury, which initiated the series of events that lead to death.

Table 4.1: List of countries and years, for which we are able to reconstruct LAR for overall mortality (source: HMD, 2014) and by COD according to ICD10 (source: WHOMD, 2014), by fitting a GGM model

Countries	Study Period (Overall Mortality)	Study Period (COD)
Portugal	1950 - 2012	—
Spain	1950 - 2012	1999 - 2011
Sweden	1950 - 2011	1997 - 2010
France	1950 - 2012	2000 - 2010
Japan	1950 - 2012	1995 - 2011

Source: WHOMD and HMD 2014, own elaboration

4.3. Methods

4.3.1. The life-table aging rate

If data are available for single-year age groups, the rate of population aging (LAR), here addressed as $\bar{b}(x)$, can be obtained by the following formula (Horiuchi and Coale, 1990):

$$\bar{b}^*(x) = \ln(M(x)) - \ln(M(x-1)), \quad (4.1)$$

where $M(x)$ is the central death rate at age x .

Nevertheless, the small number of deaths at very old ages results in a large stochastic variation in death rates and, consequently, a two-step procedure need to be followed (Horiuchi and Coale, 1990):

- a. apply a five-year moving average to the central death rates $M(x)$ and then calculate LAR using (4.1);
- b. taking nine-year weighted moving averages on the results obtained by (4.1) applying:

$$\bar{b}_{emp}(x) = \sum_{n=-4}^{4} \frac{(5 - |n|)}{25} * \bar{b}^*(x + n). \quad (4.2)$$

This procedure lowers fluctuations due to possible high variation of the death rates and produces similar curves of $\bar{b}_{emp}(x)$.

4.3.2. Specifying LAR under a gamma-Gompertz-Makeham framework

Human populations are constituted by a combination of different heterogeneous subpopulations, where despite sharing the same hazard of death, individuals present different levels of susceptibility to death. Within this framework, we assume that the baseline hazard $\mu(x)$ follows a Gompertz-Makeham pattern (Gompertz, 1825; Makeham, 1860):

$$\mu(x) = a e^{bx} + c, \quad (4.3)$$

where the aging process is captured by b , obtained by the relative derivative of $a e^{bx}$, i.e., the Gompertz part of the equation, a refers to the starting level of mortality for a given adult age (where $x = 0$) and c corresponds to non-senescent mortality. Gampe in 2010, in her research, found evidence for a human mortality plateau at older age mortality when realized that mortality rates are leveling off beyond age 110, being favorable to the application of relative-risk models (Missov and Vaupel, 2015).

Again, if we assume frailty as being gamma-distributed with a unit mean and γ variance at the starting age of analysis, which is an additional standard (Beard, 1959; Vaupel et al., 1979) already proved theoretically (Steinsaltz and Watcher, 2006; Missov and Finkelstein, 2011), we end-up with a *gamma-Gompertz-Makeham* (Γ GM) *frailty model*. Thus, within a Γ GM theoretical framework, the hazard for the population is given by (Vaupel et al., 1979; Vaupel, 2002; Vaupel and Missov, 2014):

$$\bar{\mu}(x) = \frac{a e^{bx}}{1 + \frac{\gamma a}{b} (e^{bx} - 1)} + c. \quad (4.4)$$

where, a , b and c maintain the same meaning explained before and γ is the frailty variance at the initial age of analysis.

Like it was said before, considering the model in (4.4), the individual rate of aging is captured by the relative derivative of the Gompertz part $a e^{bx}$, but in opposition, the relative derivative of $\bar{\mu}(x)$ results in the obtainment of the population rate of aging. If the first is constant across age, corresponding to b in (3), the second, proposed by Horiuchi and Coale in 1990 and addressed later as the *life-table aging rate* (LAR), varies age-wise presenting a characteristic bell-shaped pattern (Horiuchi and Coale, 1990; Horiuchi and Wilmoth, 1997, 1998; Vaupel and Zhang, 2010). LAR has been widely studied, either for human populations (e.g., Horiuchi and Wilmoth, 1997, 1998) or non-human (e.g., Carey and Liedo, 1998), and in 2010, Vaupel and Zhang, explored an explicit relationship between the individual rate of mortality increase and the population rate of aging, proving that, not only b differs from LAR, but also contributes to the estimation of the second.

The life-table aging rate (Horiuchi and Coale 1990), measures the rate of aging at age x for a population, whose mortality follows a hazard function $\mu(x)$, is defined as:

$$\bar{b}(x) = \frac{1}{\mu(x)} \frac{d\mu(x)}{dx} = \frac{d \ln \mu(x)}{dx}, \quad (4.5)$$

In a GGM framework, an explicit relationship between the individual and the population rate of aging is presented by Vaupel and Zhang (2010):

$$\bar{b}(x) = b \left(1 - \frac{c}{\mu(x)}\right) - \gamma \left(1 - \frac{c}{\mu(x)}\right) (\mu(x) - c). \quad (4.6)$$

As it was explained in the chapter *introduction*, the inclusion of c in the model is important for capturing the bell-shaped pattern of $\bar{b}(x)$ as “*not all monotonically increasing logistic $\mu(x)$ result in bell-shaped $\bar{b}(x)$ patterns*” and “*if the factor c is excluded from the individual-level equation, then the resulting aggregate-level $\mu(x)$ function does not lead $\bar{b}(x)$ to a bell-shaped pattern*” (Horiuchi and Coale, 1990), and its omission can even result in biased estimates for the other parameters (Missov and Nemeth, 2014).

The age at which the relative derivative of $\mu(x)$ reaches its maximum is the age when mortality starts to decelerate. In a GGM framework we can easily derive a closed-form expression for the age of mortality deceleration:

$$x^* = \frac{1}{b} \ln \left(\frac{(b + c \gamma) c}{2 a b} + \frac{\sqrt{(b + c \gamma) c \gamma ((b + c \gamma) c - 4b(a \gamma - b))}}{2 a b \gamma} \right) \quad (4.7)$$

4.3.3. Fitting overall and cause-specific mortality

To capture accurately mortality dynamics across different periods we fitted model (4.4). As a result, in year y , we capture the overall force of mortality by:

$$\bar{\mu}(x, y) = \frac{a(y) e^{b(y)x}}{1 + \frac{\gamma(y)a(y)}{b(y)}(e^{b(y)x} - 1)} + c, \quad (4.8)$$

where $a(y)$ is the starting level of mortality, $b(y)$ is the rate of individual aging, $c(y)$ is the Makeham term, $\gamma(y)$ the frailty variance at the initial age of analysis x_0 ($x_0 < x$) among survivors from cohort $y - x$.

Studying different subpopulations from COD i , we assume that exposures by cause are the same as exposures for the overall mortality $E_i(x, y) = E(x, y)$ for all i, x and y . Following this assumption, cause-specific force of mortality can be expressed by:

$$\bar{\mu}_i(x, y) = \frac{a_i(y) e^{b_i(y)x}}{1 + \frac{\gamma_i(y)a_i(y)}{b_i(y)}(e^{b_i(y)x} - 1)} + c_i(y). \quad (4.9)$$

We are thus estimating a unique starting level of mortality $a_i(y)$, the rate of individual aging $b_i(y)$, the frailty's variance $\gamma_i(y)$ at the starting age of analysis, and Makeham's term $c_i(y)$ by COD i .

Introduced the model framework, fitting procedure holds on the assumption that $D_i(x, y) \sim \text{Poisson}(E(x, y) * \bar{\mu}_i(x, y))$ (Brillinger, 1986), where $D_i(x, y)$ denote the cause-specific death counts i , and $E(x, y)$ denote the age specific exposures, both in year y . Thus, for each COD $_i$ separately in year y we maximize a Poisson log-likelihood:

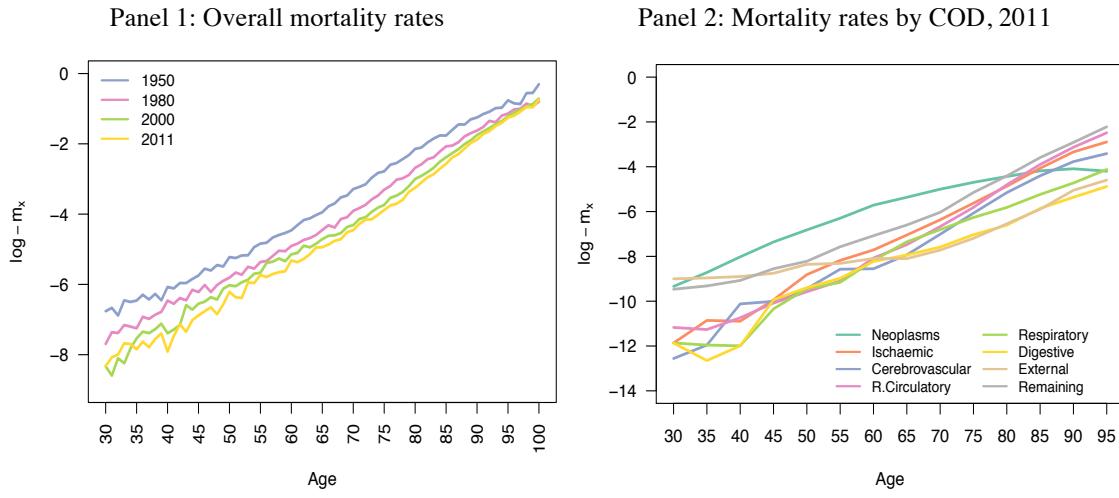
$$\ln L(a_i(y), b_i(y), \gamma_i(y), c_i(y)) = \sum_x [D_i(x, y) * \ln \bar{\mu}_i(x, y) - E(x, y) * \bar{\mu}_i(x, y)] \quad (4.10)$$

The same procedure is adopted to estimate the parameters for all causes combined (when $i = \emptyset$, we refer to (8)).

We start fitting both mentioned models (4.8) and (4.10) at age 65 for overall and cause-specific mortality. If in overall mortality, from this point onwards the mortality patterns follow an almost linear pattern on a logarithmic scale, followed by eventual deceleration, for some COD $_i$ subpopulations, the underlying mortality

patterns do not seem to follow a Γ GM-curve, particularly at earlier ages (see Figure 4.1) and parameter estimation would be meaningless.

Figure 4.1: Mortality rates for overall and cause-specific mortality, Sweden - Females



4.3.4. Data Smoothing

As explained before, we assume that the total number of deaths across age and year follow a Poisson distribution, but sometimes there is evidence for overdispersion (Breslow, 1984; Cameron and Trivedi, 1986; Camarda, 2008 and 2012). Overdispersion can be mainly found in countries with relatively poor data or historical data trends and is generally caused by age heaping or digit preference, i.e., the tendency to round counts or measurements to pleasant digits (Camarda, 2008). In order to stabilize the high variance that is associated with high age-specific death rates, we consider the latter on a log-scale and smooth them.

It is true that the considered model (Γ GM) is also a valid approach to smooth mortality², but once that overparametrization may influence the estimation procedure, our choice fell on a more flexible approach to describe age patterns and time trends. “*Because mortality developments generally display regular patterns, using smoothing approaches is a more natural choice for analyzing mortality changes than imposing a model structure*” (Camarda, 2012).

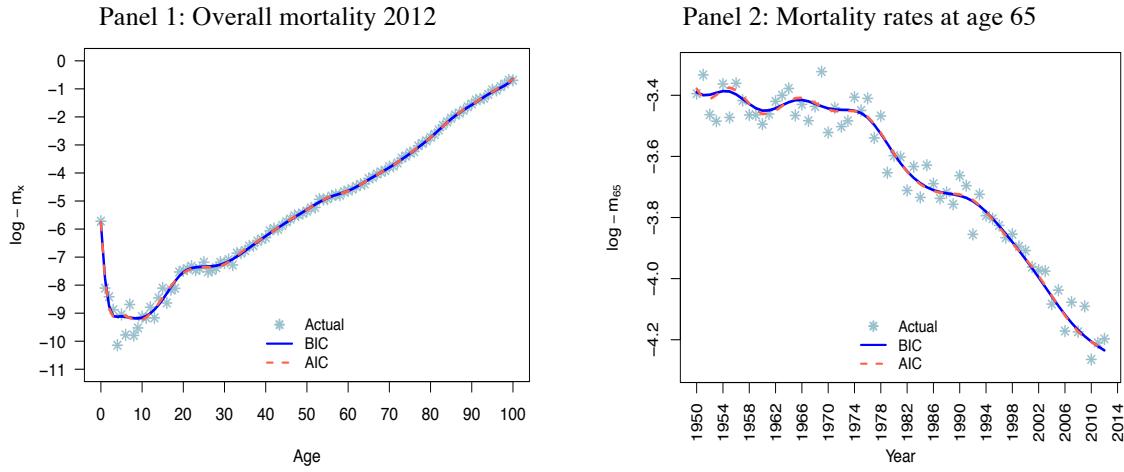
² Similarly, in the HMD the Kannisto model is used to smooth mortality rates at older ages (Wilmoth et al., 2007).

The use of smoothed data to construct life-tables results, as expected, in lower variance, providing therefore, better estimates of life expectancy (Booth, Hyndman and Tickle, 2014). Since we elaborate on model-based LAR estimates resulting from a FGM framework, data should be leveled among the selected countries to enable proper and accurate comparisons.

Although several methodologies have been proposed to deal with either one or two-dimensional data structures, we focus on the two-dimensional level, taking advantage of the MortalitySmooth (Camarda, 2012) R package (R Development Core Team, 2014). This package employs an approach using two-dimensional regression splines, or to be more specific, B-splines with penalties: P-splines (Camarda, 2012). Within this framework, and to be able to handle a smoothing procedure, the model needs to achieve an “optimal” value (or values) for the smoothing parameter. A non-optimal value influence negatively and increase estimates variance. Two of the most known procedures for this type of model construction are the Akaike information criterion (AIC) (Akaike, 1973), suggested by Eilers and Marx (1996, 2002), and the Bayesian information criterion (BIC) developed by Schwarz in 1978, penalizing more heavily the model complexity. Nevertheless, a solid fit, which is given by BIC, is desired when mortality rates are smoothed applying the P-splines approach (Currie *et al.*, 2004; Camarda, 2012).

Figure 4.2 exemplifies the differences between estimated smoothed curves for Portugal (the second-smallest in population size among the countries under study, known for its lower data quality prior to 1970) using both information criteria, BIC and AIC, for male overall mortality (panel 1) and at age 65 (panel 2), in 1950 and 2012. Both P-spline approaches are capturing well the evolution of mortality patterns over age and time. In panel 2, one can see a small difference in the first 8 years of fit, showing that if we employ the BIC penalization, the degree of smoothness does not lead to the overestimation of small fluctuations that are possibly caused by randomness.

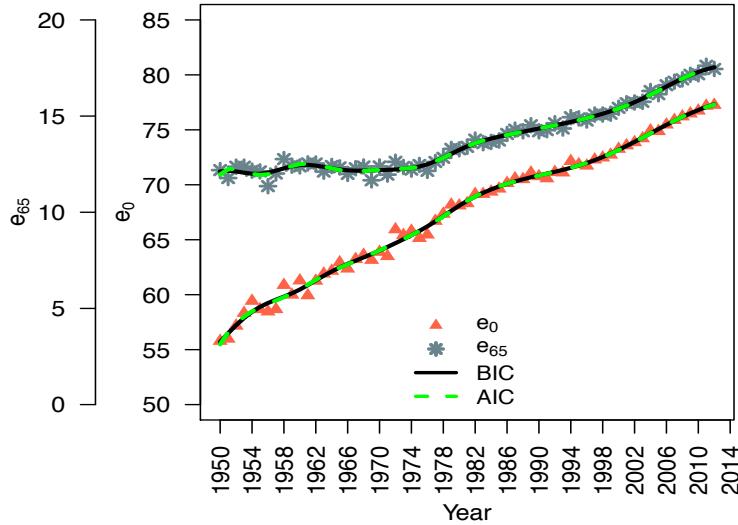
Figure 4.2: Actual and fitted death rates from a P-spline approach, Portugal - Males



Source: HMD 2014, own elaboration

As expected, smoothing mortality rates does not change life expectancy estimations (Figure 4.3), and similarly to what was shown in Figure 4.2, the differences between both penalties (BIC and AIC) are almost negligible and the overall trend is well captured.

Figure 4.3: Actual and fitted life expectancy from a P-spline approach, Portugal - Males



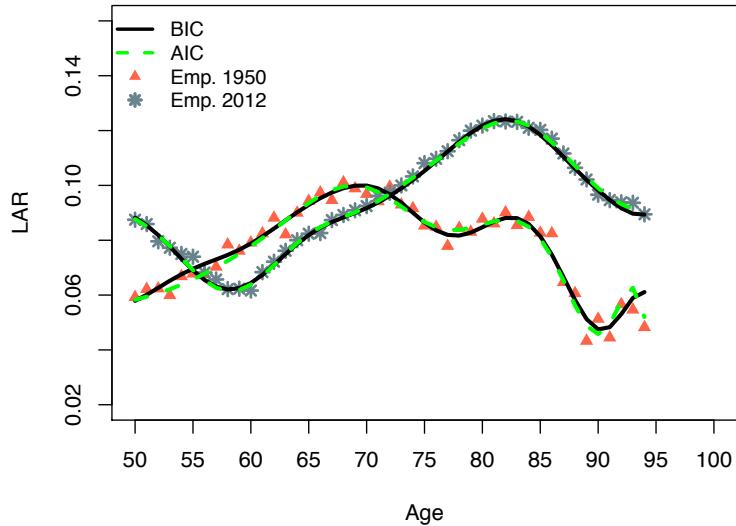
Source: WHOMD and HMD 2014, own elaboration

Nevertheless, it is not our intention to elaborate exclusively on life expectancy dynamics, but on the possible connection between life expectancy and the life-table aging rate. Figure 4.4, proves, once more, that the P-spline approach does not change the original information and fits well empirical LAR. Again, some minor differences

can be registered between both penalizations but the degree of smoothness resulting from BIC approach does not lead to the overestimation of small fluctuations.

The examples presented here reinforce the idea of BIC as the best approach, and especially that the use of smoothed data in our study will not influence final outcomes and conclusions.

Figure 4.4: Empirical and fitted LAR from a P-spline approach, Portugal - Males



Source: HMD 2014, own elaboration

4.3.5. Segmenting life expectancy dynamics

To elaborate on the relationship between estimated LARs and life expectancy dynamics, we differentiate between different period segments on the overall timeline, i.e., we estimate independently different regression lines that incorporate a piece-wise linear relationship between life expectancy and calendar year. This results in two (or more) line segments. Assuming a changing point i , the relationship between the different life expectancies (e_i) registered across years and the calendar year itself (y_i) is captured by:

$$e_i = \alpha + \beta_1 y_i + \beta_2 (y_i - \psi)_+ \quad (4.11)$$

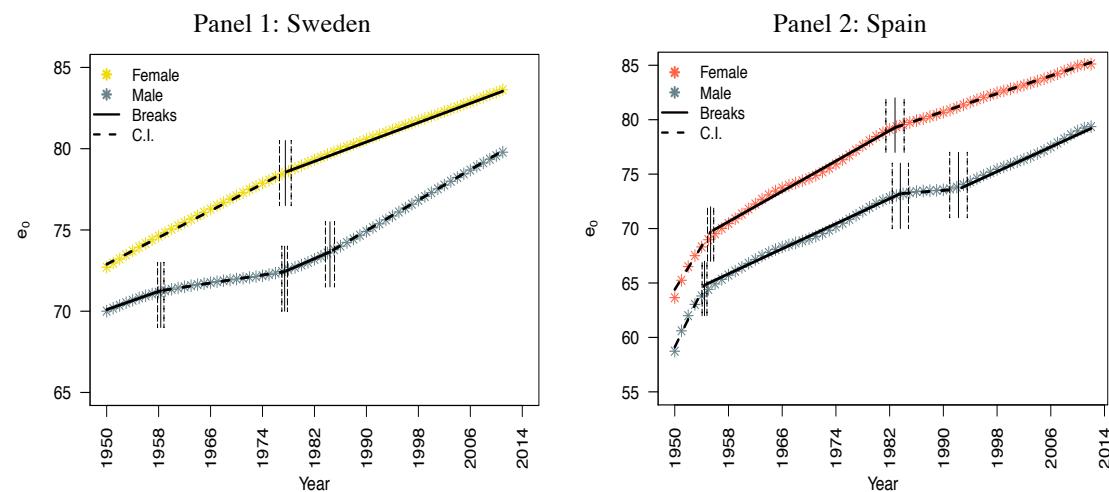
where α is the intercept, β_1 is the first segment slope, β_2 the difference-in-slopes for the second segment, ψ denotes the breakpoint, and $(y_i - \psi)_+ = (y_i - \psi) *$

$I(y_i > \psi)$ (Muggeo, 2003). The indicator function $I(\cdot)$ equals one when the condition in its argument is true. When the model does not detect a breakpoint, we end up with a simple linear regression model, i.e., ψ does not exist and β_2 is a statistical zero.

Figure 4.5 presents not only life expectancy at birth for Sweden and Spain, already calculated based on smoothed data, but also the segments that are statistically significant. It is easy to see that between 1950 and 2012 there exist segments with different slopes. In the Swedish case, the slope declined in 1977, which resulted in lower life-expectancy increase and let France, Spain and Japan move ahead (see Figure 2.1, Chapter 2). In comparison with males, females are exposed to fewer breaks in their life expectancy at birth over time, but it is clear that in the last segment for each country, males are catching up.

The inclusion of confidence intervals for the breaks in the plots provides additional insight. In the example of Spain (panel 2), the break registered in 1983 does not differ statistically between sexes, but it refers to two different slopes in life expectancy increase. For Sweden (panel 1), similar behavior was detected for the break estimated in 1977.

Figure 4.5: Segmented life expectancy at birth



Source: HMD 2014, own elaboration

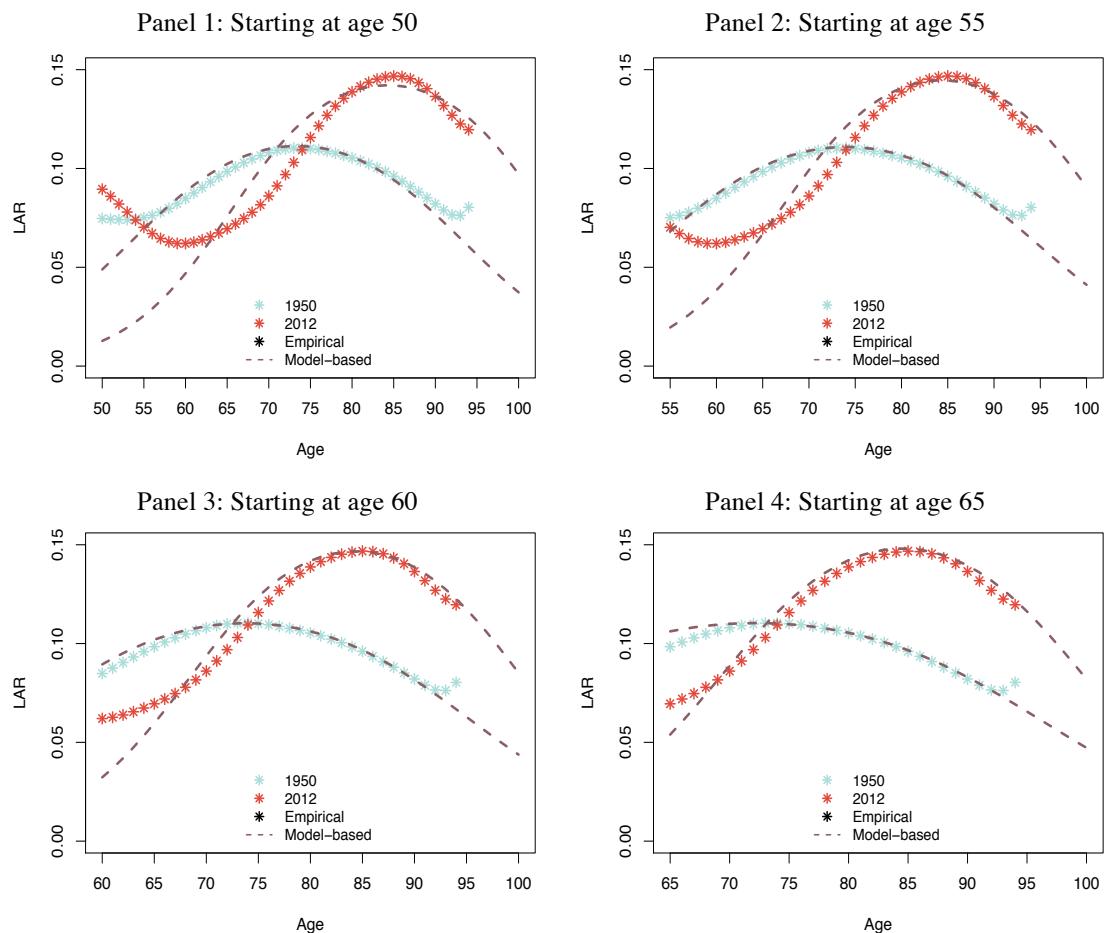
4.3.6. The starting age

As it was already widely studied and exemplified (Horiuchi and Coale, 1990; Horiuchi and Wilmoth, 1997 and 1998, among others), LAR patterns are not constant

but follow a distinguishing bell-shaped pattern. The exact starting age of this pattern is difficult to identify due to continuous mortality changes across time thus, it becomes important to identify the “best starting age” for fitting the GGM and evaluating its accuracy.

Figure 4.6 presents the results obtained by fitting a GGM model with four different starting ages x : 50, 55, 60 and 65. Our intention was to explore if a change in the starting age would affect model accuracy. As one can see, the higher the starting age the better the obtained accuracy for both sexes. Despite the fact that all model-based LARs seem to generate very accurate estimates for the age of mortality deceleration, Figure 4.6 shows that the best choice for males might be age 60 and for females age 65. However, choosing different starting ages by sex would open another discussion. Bearing in mind that we aim the best fit for both sexes and across all the selected countries, we decide to start our entire fitting procedures at age 65.

Figure 4.6: Model-based LAR with different starting ages, France - Female



Source: HMD 2014, own elaboration

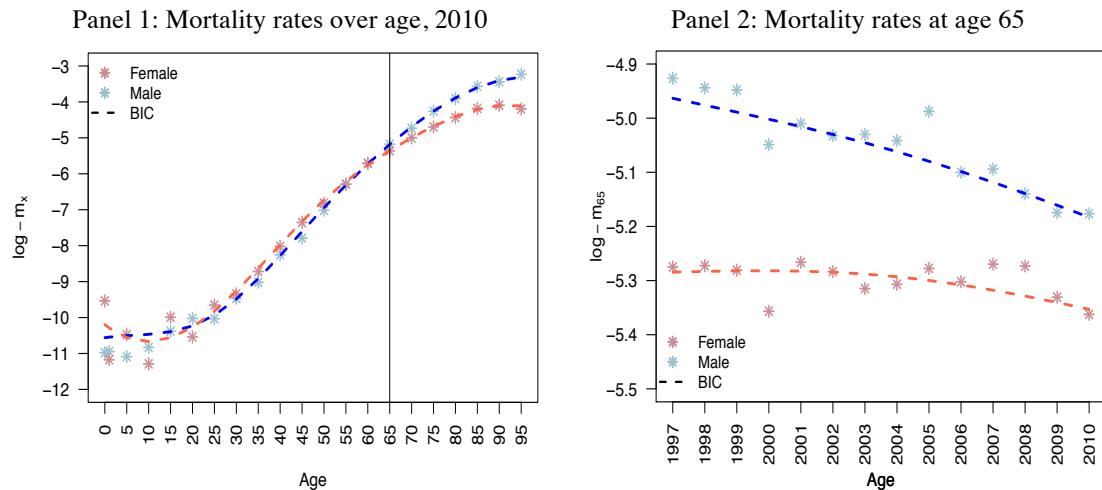
We focus on the last 60 years of mortality history (since 1950s), but always taking into account the limitations (availability and time range) associated with data quality and availability and for each selected country. Therefore, for each year in the available period (see Table 4.1), we estimate LAR for the overall mortality by fitting a GGM model beyond age 65.

4.3.7. Cause of death

Low subpopulation sizes are often problematic when fitting statistical model, so we can expect less accurate results for Portugal (10.5 M) and Sweden (9.6 M), the countries with the smallest population size in the list. Disaggregating COD data into subgroups lowers subpopulation sizes even more. As in the case of overall mortality, we smooth the data to lower variability and at the same time increase fitting accuracy. With COD we are dealing with even lower subpopulation sizes and even more distinct data fluctuation and as we saw before, smoothing the data applying a P-spline approach using BIC, penalizes more the model complexity and produces smoother results. Thus, we decided to employ directly the BIC approach instead of comparing both fits.

Figure 4.7, presents the results for Sweden, which is the country with the smallest population size in our list. Panel 1 shows that, beside the fact that original data are well approximated, the mortality patterns over age become smoother and after age 65 accuracy increases. Panel 2 provides a closer point of view, where the mortality rates registered at age 65 across the available years for Neoplasms are presented on a smaller y-axis scale. Thus, one can conclude that both, accuracy and smoothness are ensured, providing a better illustration of the mortality patterns.

Figure 4.7: Actual and fitted death rates from a P-spline approach, Sweden - Neoplasms

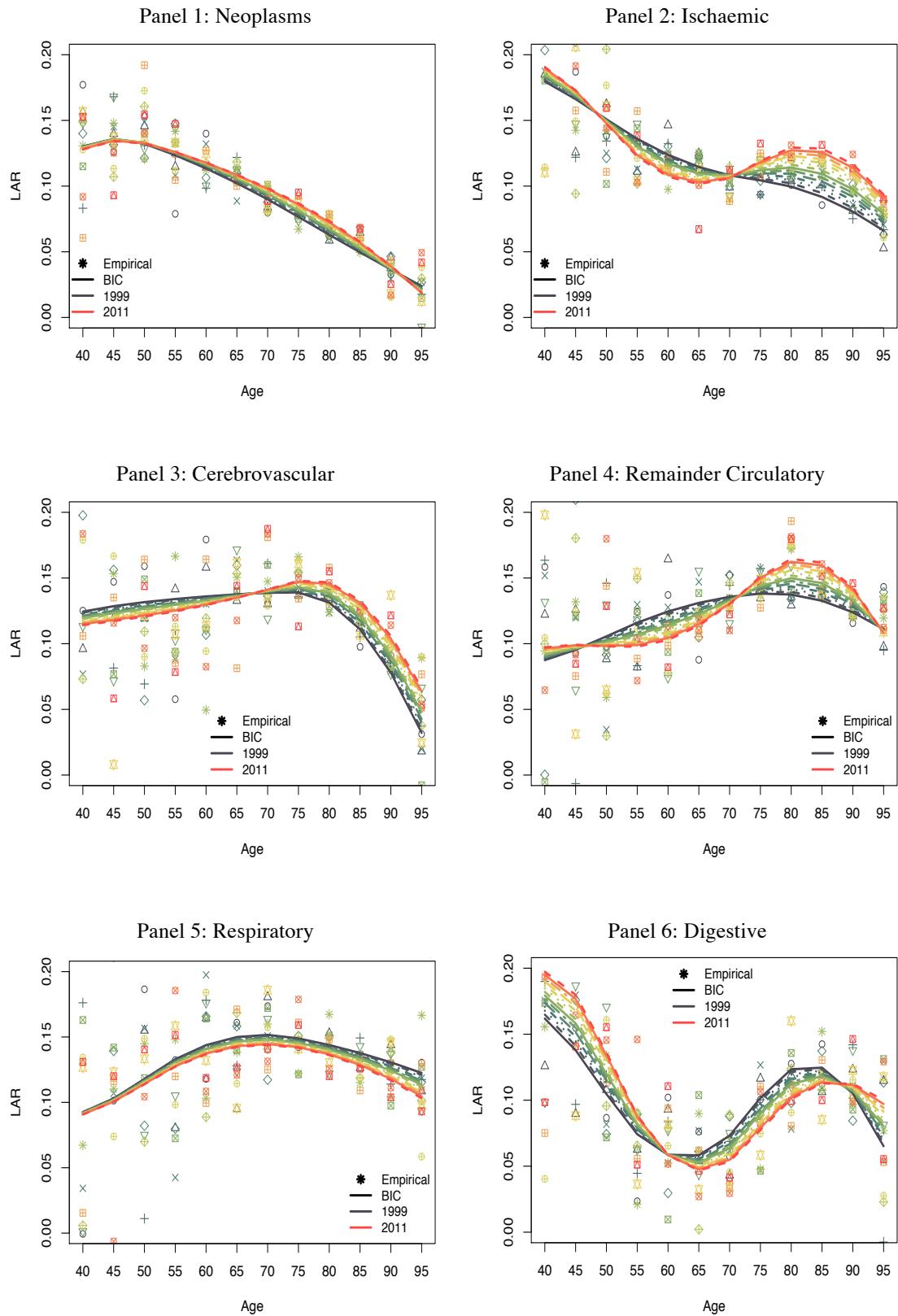


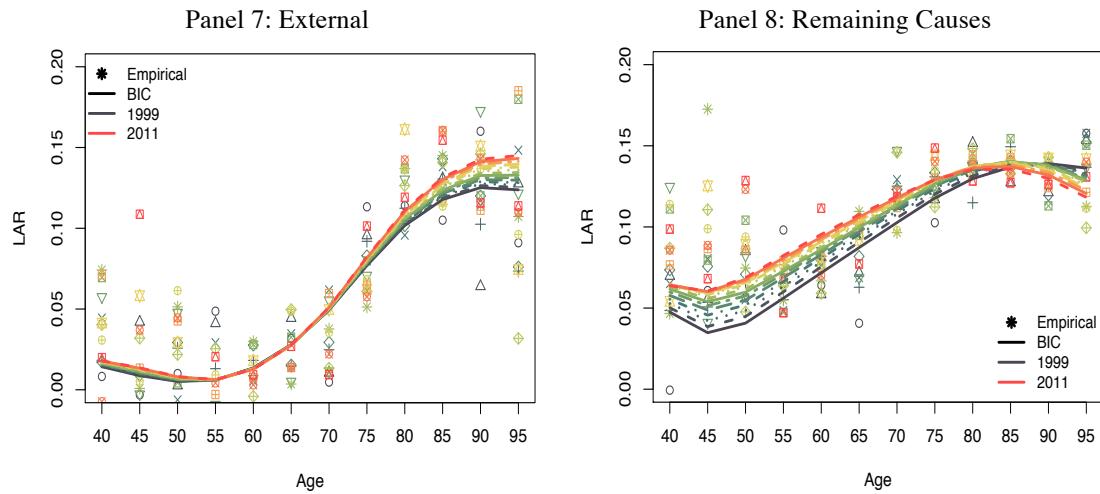
Source: WHOMD and HMD 2014, own elaboration

Nevertheless, like in the case of overall mortality, we expect that the smoothing procedure will not affect negatively the empirical LAR patterns. Figure 4.8 demonstrates that the P-spline approach applied to COD subpopulations does smooth empirical LAR, expressing clearly the associated patterns. We can clearly see in Sweden a mortality deceleration for neoplasms starts around age 45, in the male case. Between 1999 and 2011, there are some CODs that affect Swedish males mortality, presenting more pronounced bell-shaped patterns with time (Ischaemic, Circulatory and Respiratory diseases). Some other presented a shift in the age of mortality deceleration to older ages at the same time (Cerebrovascular, Digestive and External causes). Changes in LAR for neoplasms are almost imperceptible but perspectives some variations beyond age 60 and the group including remaining causes present not only a more pronounced pattern, but also a non-expected shift to younger ages. This situation might be associated with possible changes in how different CODs affect overall mortality, and some non-specified causes might become more influential than previously.

Figure 4.8 also shows that the starting adult age chosen to fit the GGM model will probably have higher influence on model-based LAR accuracy than for overall mortality, because some LAR deceleration pattern start at earlier ages and some other at older ages.

Figure 4.8: Empirical and fitted LAR from a P-spline approach by COD, Sweden - Male





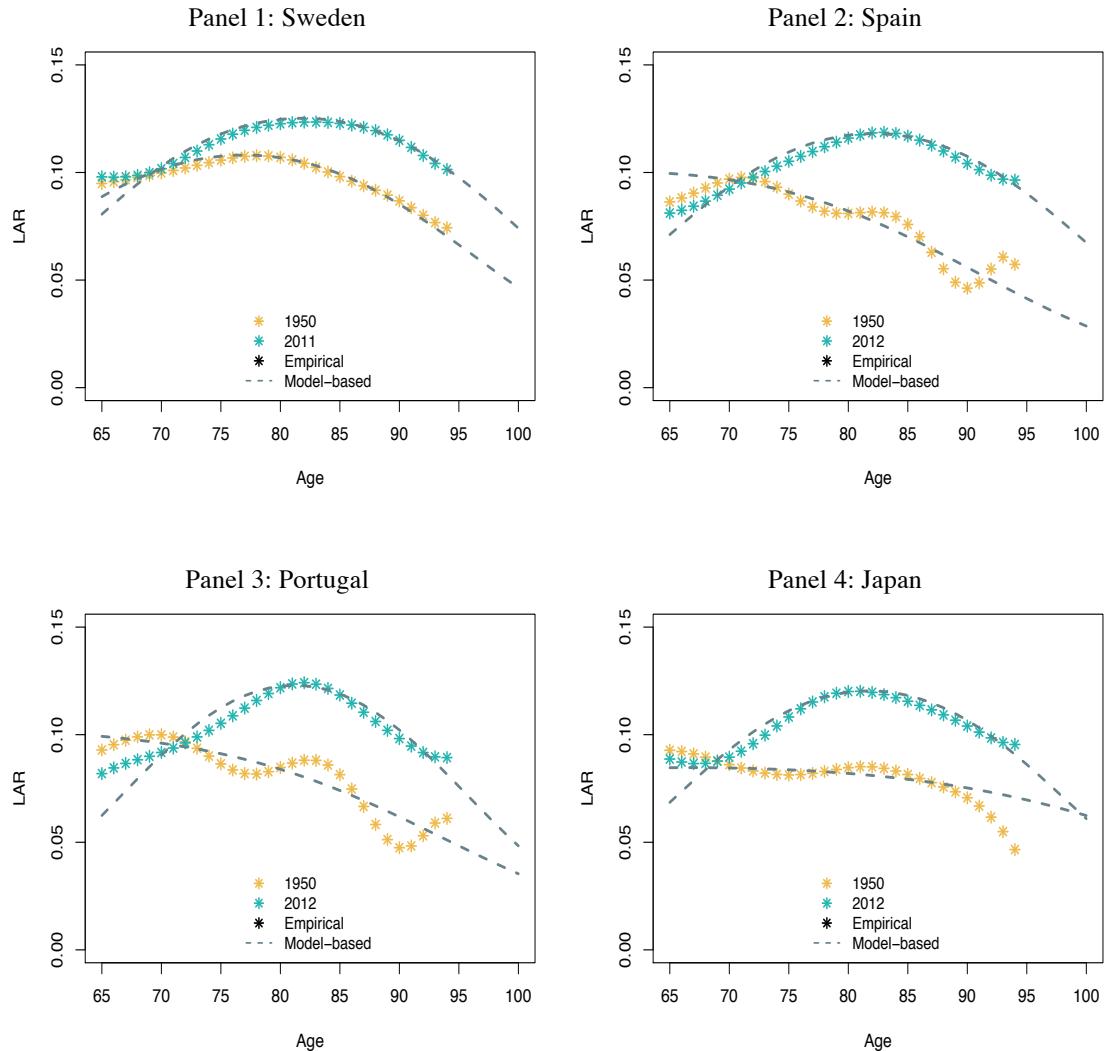
Source: WHOMD and HMD 2014, own elaboration

4.4. Results

4.4.1. Overall mortality

By fitting a GGM model from age 65 onwards, we estimate the population rate of aging $\bar{b}(x)$ by country and gender. Results for France and Spain, presented in Figure 4.9 indicate that the GGM model-based LAR fits well observed/empirical LAR. At the same time, it captures the observed shift in the age of mortality deceleration between 1950 and 2012, for both sexes, confirming therefore point b) of the “heterogeneity hypothesis”: with increasing lifespans, mortality deceleration shifts and occurs always at older ages. In panels 2 to 4, we can also detect a more pronounced pattern with time.

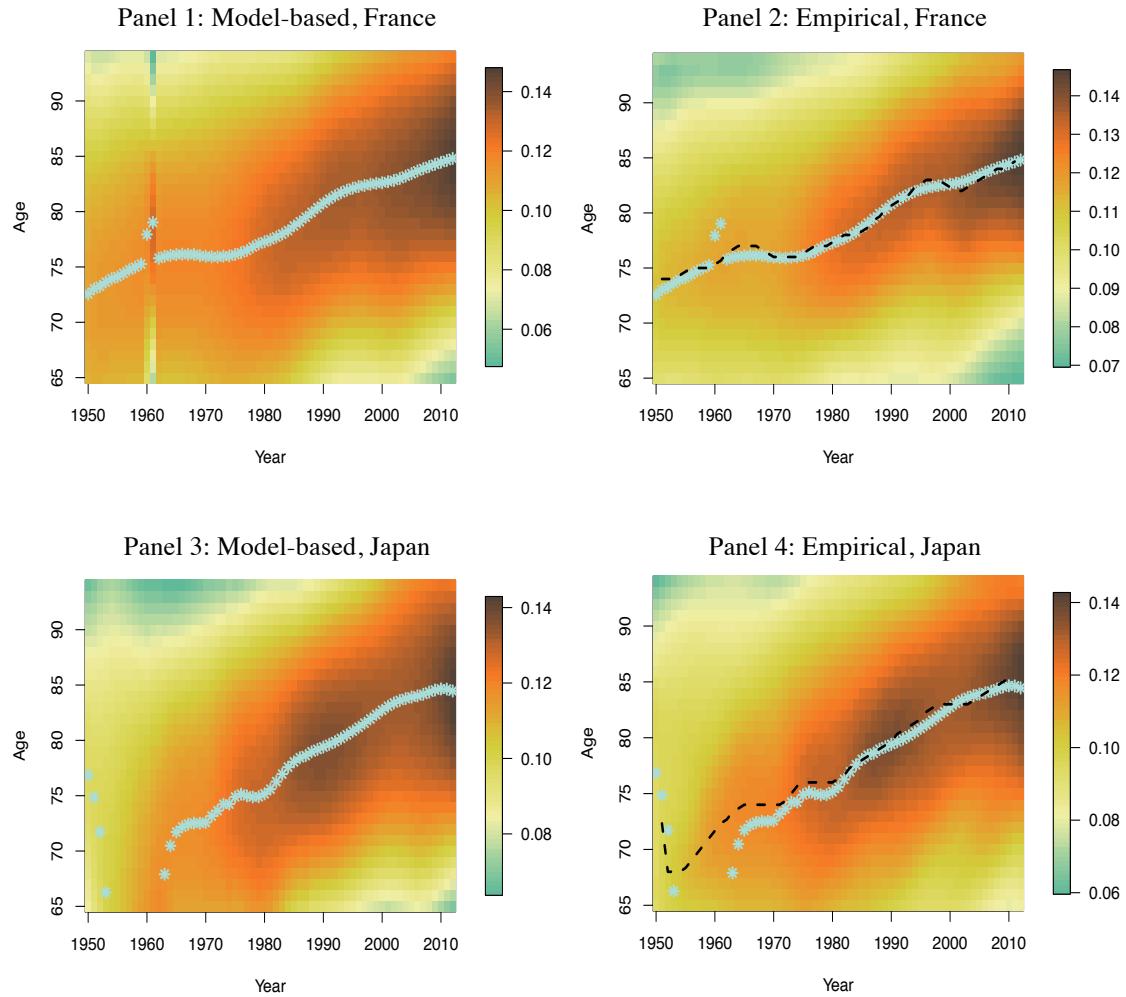
Figure 4.9: Model-based LAR goodness of fit for the Male example



Source: HMD 2014, own elaboration

Model-based LAR estimates a flatter pattern (larger darker shadow on model-based estimates compared to the empirical results) than it is really observed (Figures 4.9 and 4.10), but the age of mortality deceleration seems to be well captured for the French females (panel 1 and 2), being pretty similar to the empirical LAR. While in the French case we only find two main “deviations” from the observed values (panel 1, years 1960 and 1961), in the Japanese example the model captures the age of mortality deceleration well only after 1981. This difficulty in estimating the point where LAR finds its maximum results from the poor accuracy of model fitting in those years.

Figure 4.10: Model-based, Empirical LAR and the age of mortality deceleration (fitted: light-blue star dot; empirical: black dashed line), Females



Source: HMD 2014, own elaboration

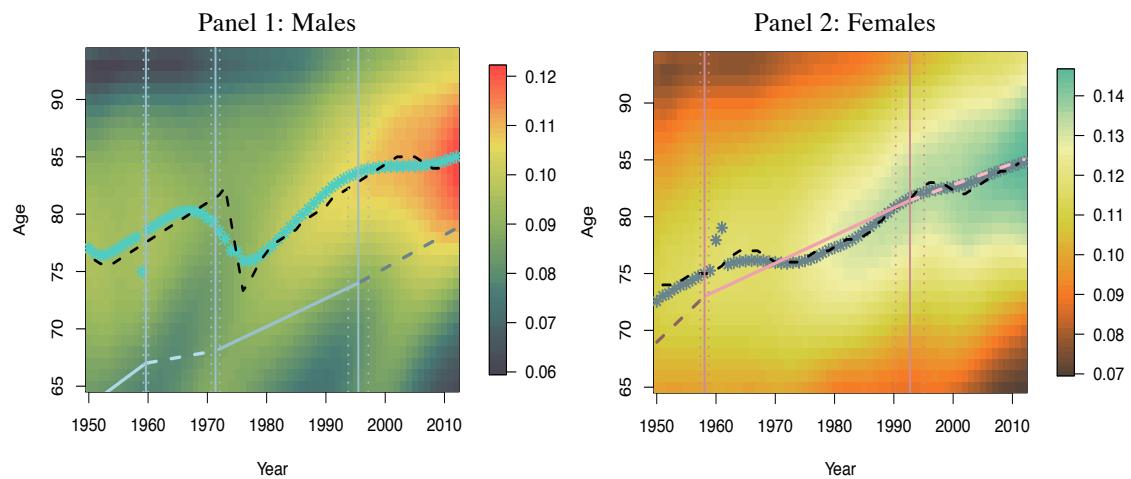
Figure 4.11 reinforces that the formula advanced in (4.6) estimates well LAR patterns for the overall mortality, despite the model estimation of a flatter pattern than really observed, especially in more recent years. If we add to the image (see French example in Figure 4.11) the segmented life expectancy patterns and corresponding breaks, we can see that even though the age of mortality deceleration considerably differs from life expectancy itself, the estimated breaks seem to detect some associated changes in the slope of life expectancy increase.

The results for French females (panel 2) suggest that at the first breakpoint we observe at the same time a steeper increase of life expectancy and, in the age of mortality deceleration (estimated and empirical). At the second breakpoint, there is a decline in the rate of life expectancy increase that comes along with a period of

additional turbulence in the age of mortality deceleration. At the third and last breakpoint, a new decline in the rate of life expectancy is noticeable and both patterns (x^* and e_0) look similar. In the male case (panel 1), the estimated age of mortality deceleration shows higher deviation from the empirical one (calculated based on empirical LAR), but the patterns are very alike.

In contrast to females, in the male case the estimated breaks and corresponding rates of increase in life expectancy substantially differ from the age of mortality deceleration. However, it seems, once again, that the estimated breaks suggest some connection between these two mortality indicators and changes in one pattern is complemented by changes in the other. From all the countries, despite that we only present here Japanese and French examples, France is the only one that fits better the observed LAR and gives better x^* estimates for the entire observed period. We believe this might be due to the high life expectancy values from the 1950s onwards. In Japan, for example, estimates become better when life expectancy increased substantially. It is also our guess that at the time when the male/female gap in life expectancy declines, x^* will be better captured by the model and life expectancy values will be closer to the age of mortality deceleration.

Figure 4.11: Empirical LAR, the age of mortality deceleration (fitted: light-blue star dot; empirical: black dashed line) and life expectancy segments, France



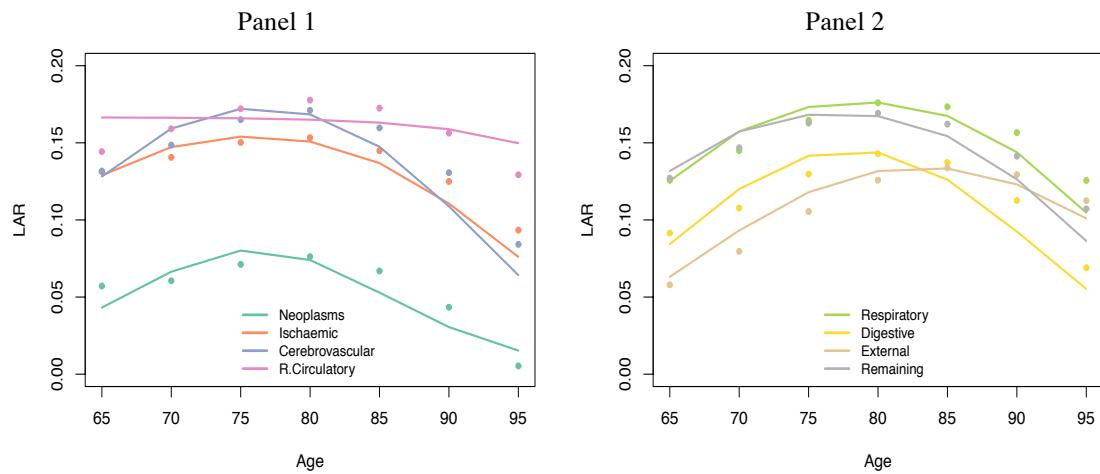
Source: HMD 2014, own elaboration

4.4.2. Causes of death as subpopulations

Considering different causes of death as subpopulations we maximize $\ln L$ presented in (4.10). As a result, we estimate 4 parameters by COD for each year, resulting in a total of 32 parameters estimated per year. We start fitting the model mentioned in (4.9) at age 65, i.e., from the 65-69 age group onwards. As discussed before, starting from this point onward the mortality patterns for all major CODs follow an almost linear pattern on a logarithmic scale, followed (in some cases) by an eventual deceleration (see Figure 1). Thus, fitting a GGM model (4.9) from age 65 onwards for the different COD subpopulations, we estimate the population rate of aging $\bar{b}(x)$ by country, gender and cause of death.

Results for Spain, presented in Figure 4.12 show that the GGM model-based LAR by COD does not fit well observed/empirical LAR. In general model-based LAR appears to capture well empirical patterns, however, in some cases seems to anticipate the age of mortality deceleration. In the example of Spain in Figure 4.12, the only COD that diverges completely from the observed pattern is the Remaining Diseases of the Circulatory System.

Figure 4.12: LAR by cause of death, Spain 2011 - Females

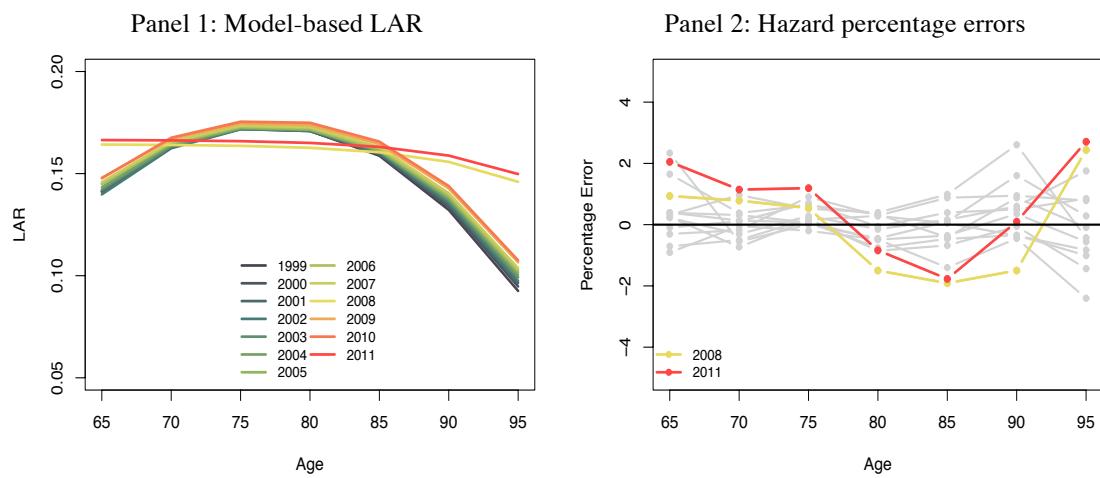


Source: WHOMD and HMD 2014, own elaboration

Trying to understand why, in the year of 2011, GGM estimated LARs are not capturing the empirical pattern, in Figure 4.13 we have all the model-based LAR estimated for the remaining diseases of the circulatory system across the 13 available years for Spain (panel 1) and the associated percentage error for the estimated hazards

of death. First, more than one year is identified presenting the same flat and incorrect LAR pattern, 2008 and 2011. Second, observing the percentage errors associated to the estimated hazards, we realize that, despite small, the associated errors for those two years are the highest for the age groups 80-84, 85-89, and 95+. In 2011, the same is registered for the age groups 70-74 and 75-79. Thus, we can conclude that even a small percentage error across age might influence the estimation of the bell-shaped pattern observed empirically.

Figure 4.13: Model-based LARs and hazards percentage errors for remaining diseases of the circulatory systems, Spain - Females



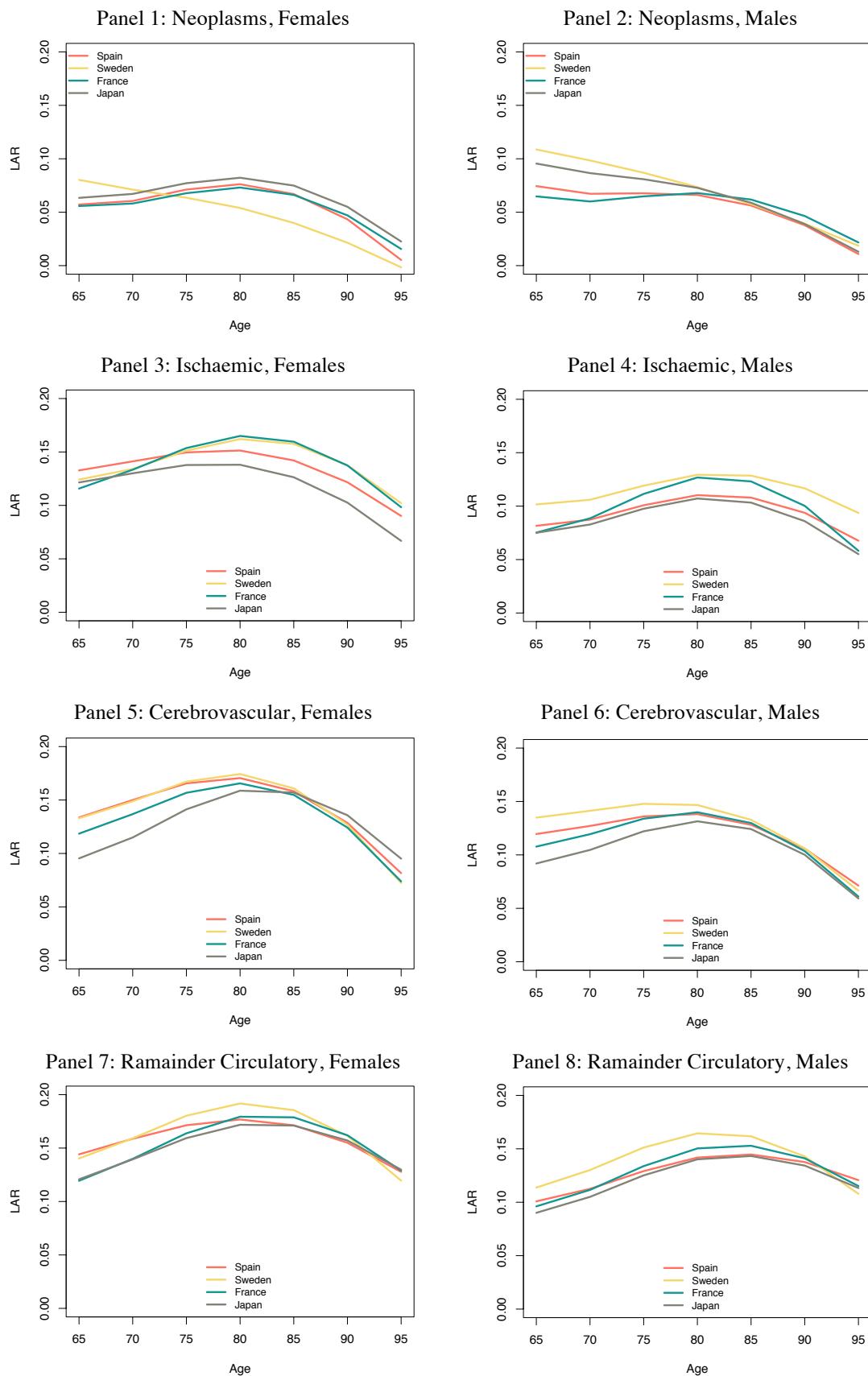
Source: WHOMD and HMD 2014, own elaboration

Figure 4.14 presents the obtained results for empirical LAR between ages 65 and 95. Each panel corresponds to the obtained LAR results by subpopulations (CODs) and sex, where the last available and transversal to all countries, year is presented. Our choice of presenting empirical LAR in opposition to the model-based comes from the difficulty that the formula advanced in (4.6) has to estimate the corresponding observed patterns by COD.

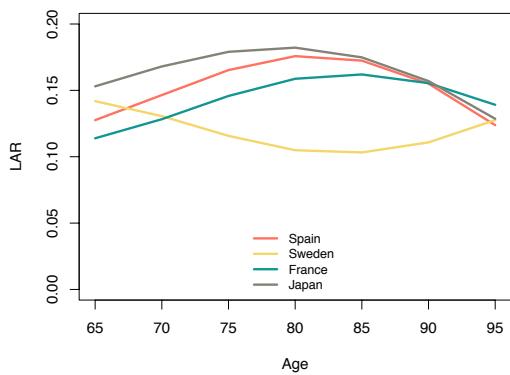
The obtained results confirm that for the considered major COD groups, deceleration is observed. However, we do not find clear evidence that CODs with lower death rates present less pronounced deceleration patterns.

In panels 9, 15 and 16, very distinct and non-bell-shaped patterns are found, however, in our opinion this is explained by the fact that mortality deceleration in those specific cases does not occur in the considered age interval.

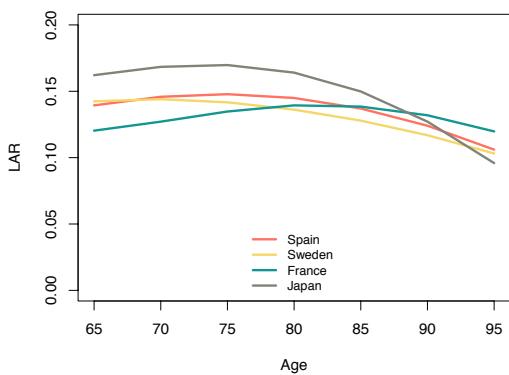
Figure 4.14: Empirical LAR by sex, COD, and country - 2010



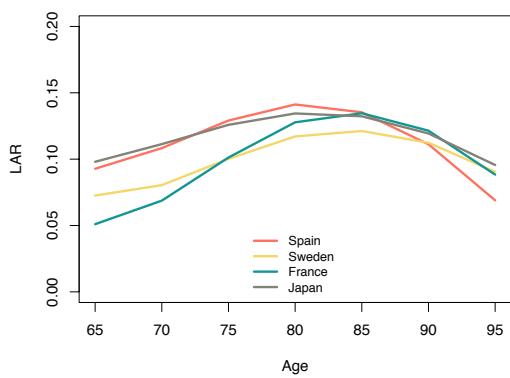
Panel 9: Respiratory, Females



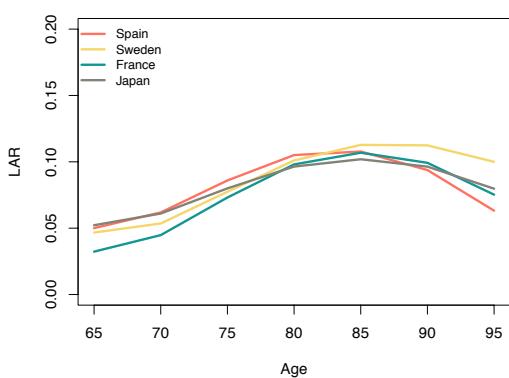
Panel 10: Respiratory, Males



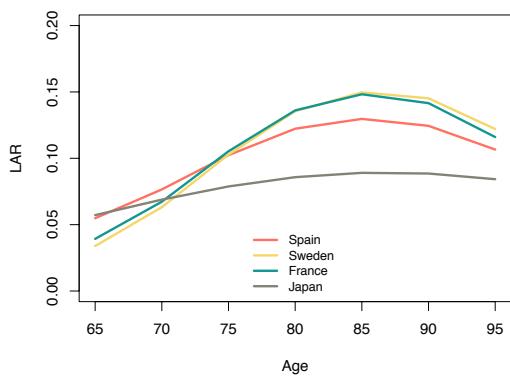
Panel 11: Digestive, Females



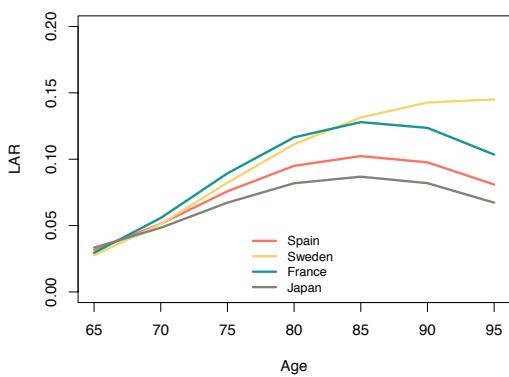
Panel 12: Digestive, Males



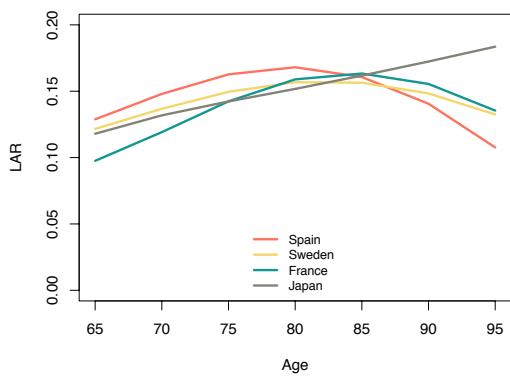
Panel 13: External, Females



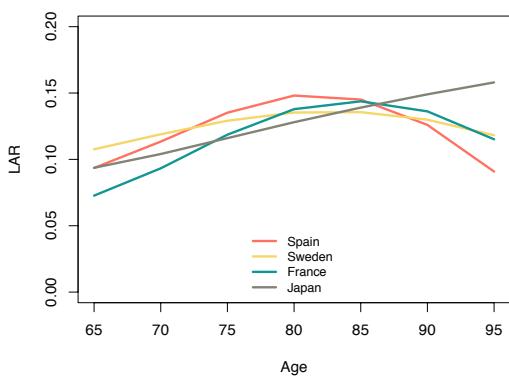
Panel 14 External, Males



Panel 15: Remaining Causes, Females



Panel 16: Remaining Causes, Males



Source: WHOMD and HMD 2014, own elaboration

4.5. Discussion

In opposition to the individual rate of aging, the population rate of aging, here addressed as LAR, requires the estimation of all four parameters in the model. Thus, within a Γ GM framework LAR requires the calculation of all a , b , c and γ (see Vaupel and Zhang, 2010).

A constant life-table aging rate would indicate that mortality increases almost linearly in a logarithmic scale. Nevertheless, either for overall mortality or discriminating by CODs, the obtained results capture an expected decrease with age. Thus, it indicates that the age-related increase in mortality rates at older ages is decelerating. Both, the estimated population and COD-specific LARs (when able to capture the observed pattern) present a non-constant pattern, expressing, excluding some rare exceptions, its characteristic bell-shaped pattern. For the exceptional cases, the factors that could have influenced estimates and observed patterns may be associated with 1) the use of aggregated data for CODs, which provides less detail than data for single ages, 2) the choice of fitting all models after age 65, when the underlying mortality patterns for some CODs might not completely follow a Γ GM-curve, 3) data credibility, specifically in the analysis by CODs, because especially at older ages, death certificates do not always reflect accurate information, 4) due to known differences in the mortality patterns associated to men and women, the model may present better fits when includes earlier information (i.e., if we start fitting at a younger or later age), 5) low subpopulation sizes (CODs or countries).

The estimation of the age of mortality deceleration seems either to be influenced by model accuracy and mortality improvements that appear to stabilize LAR patterns, presenting more defined and pronounced LAR patterns as it maximum shifts to older ages. France is a good example, as in the 1950s, the associated life expectancy at birth was already high (as a sign of better health) when compared with the other countries and sexes, still being today one of the best-practice countries in the analysis. Thus, we believe that improvements in health result in more distinct bell-shaped patters of LAR and better accuracy on the estimates.

Even when poorly approximated, the age of mortality deceleration denotes a possible connection with the life expectancy rate of increase, reacting differently within each break statistically significant.

4.6. Conclusions

Γ GM model-based LARs (for overall mortality and by COD) appear to be consistent with the “heterogeneity hypothesis” advanced by Horiuchi and Wilmoth (1998) as the age of mortality deceleration shifts to older ages with time in overall mortality and only neoplasms, as one of the CODs with lower mortality rates after age 65, presents less pronounced patterns. The Γ GM model-based LAR (Vaupel and Zhang 2010) fits observed LAR with high accuracy and captures the connection relationship between the observed shift and the rate of life expectancy increase in the five selected countries for overall mortality. Presented results for overall mortality are statistically more accurate because the analysis is based on a large number of observations in each age group. Although COD data are organized by five-year age groups, we sometimes run into problems associated with low subpopulation sizes, which might influence the accuracy of Γ GM parameter estimates. Disaggregation of causes of death into single-age groups can benefit further estimation of the associated LAR patterns.

Despite that our findings are mainly confirming previous findings of a bell-shaped pattern associated to the rate of aging for populations, with our results we contribute for a better understanding of the differences between this measure and the individual rate of aging.

CHAPTER 5

FORECASTING MORTALITY PATTERNS BY CAUSE OF DEATH: A COHERENT APPROACH

5.1. Introduction

Across time profound changes occurred in demographic paradigms. Some resulted in an extraordinary life expectancy increase and now the high rates of mortality of the past are being experienced later in life. Currently, the economic crisis witnessed in Europe, especially in the south, might disturb demographic evolutionary trends. Evolutionary mortality patterns accordingly to different causes of death are essential to give an insight not only for demographers, but also to policymakers and create robust foundations for future planning proposes.

In Japan, a female born in 2012 could expect to live slightly more than 86 years, resulting from an average 0.5 yearly increase since 1947 (HMD, 2014). It is well known that this increase is only possible due to significant improvements in health and consequent reductions in mortality rates (Vaupel, 2010). Those rates have been widely studied attempting to generate a universal law of mortality. Gompertz (1825) proposed a simple parametric model $\mu = ae^{bx}$ to explain adult mortality, accounting for the level of mortality at the starting age a , and for the rate of mortality increase with age b . Later, Makeham (1860) add a constant to the model $\mu = ae^{bx} + c$ improving the results. The inclusion of this new parameter allows differentiating either between background (c) and intrinsic (a) mortality. Despite the improvements brought by the inclusion of a third parameter in the model, mortality is still overestimated at oldest ages, leading to the suggestion of employing logistic models (Perks, 1932; Heligman and Pollard, 1980; Thatcher, 1999). Nevertheless, demographers view populations as a heterogeneous mixture of individuals that share the same baseline hazard of death, to which they are susceptible in a different and random way. Within this framework, by assumption (Gompertz, 1825; Vaupel et al., 1979), the baseline hazard follows a Gompertz curve.

Individual susceptibility can be reflected in different individual a 's (relative risk models), in different b 's (accelerated life models), or in both. However, recent evidence for a human mortality plateau (Gampe, 2010) speaks in favor of relative risks only. Letting a random variable Z , called frailty (Brass, 1971) capture individual susceptibility, leads, very generically, to the widely used gamma-Gompertz frailty model.

The models presented so far only capture changes in death rates over age. Several other approaches have been created to model different changes observed over time. One example is the Brass (Brass, 1971) relational model, where the author suggested a logit transformation of the probability of surviving, allowing to relate the standard and actual population by applying a simple regression function. One other example corresponds to the *age-period-cohort* (APC) models, and acknowledges possible changes in those three dimensions. Yet, the APC models struggle often with identification problems due to the linear relationship between age, period and cohort (Clayton and Schifflers, 1987). A third example corresponds to the method developed by Lee and Carter (1992). This method reduces the APC complexity by introducing a bi-linear model for the logarithmic mortality-rates: $\ln m_{x,t} = a_x + b_x * k_t + \varepsilon_{x,t}$. Since then, this methodology has been widely used in demography and it can be considered the standard in modeling and forecasting death rates (Camarda, 2008), even with the inclusion of some variants (Lee and Miller, 2001; Booth et al., 2002; Hyndman and Ullah, 2007), or even expressed in a Compositional Data Analysis equivalent definition (Oeppen, 2008). Nonetheless, in modeling mortality rates for two or more sub-populations it is required that the forecasts do not diverge from the ones obtained from the total population, being coherent. A very recent example of a coherent method is the product-ratio method developed by Hyndman, Booth and Yasmeen (2013). This method achieves coherence through the convergence to a set of appropriate constants of forecast age-specific ratios of death rates for any two sub-populations.

“As is well known, the estimation stage of Lee-Carter is a special case of principal component analysis, where the log-mortality data is summarized using only the first principal component” (Girosi and King, 2007) and that makes the model not appropriate for all causes of death independently. Nevertheless, in a theoretical perspective many authors support that breaking down mortality forecasts by cause

results in higher accuracy but practice also proves the opposite (Booth and Tickle, 2008). Studies like the one from Wilmoth (1995) found that decomposing mortality forecasts result not-rarely in higher mortality forecasts, however, expressing the Lee-Carter model in a conditional form results in very similar estimates in both approaches (Oeppen, 2008).

As a result, in this chapter we make use of the Oeppen's proposal of expressing the Lee-Carter method in compositional form, to elaborate coherent medium-term forecasts distinguishable by causes of death for countries with different cultural backgrounds and distinctive mortality patters, as could be seen in the previous chapters. Thus, we forecast mortality trends differentiated by cause of death for the next 30 years for Portugal, Japan, France, Sweden and Spain.

5.2. Lee-Carter model and (selected) variants

5.2.1. Lee and Carter (1992)

Lee and Carter (1992) proposed one of the most used methods to forecast mortality trends. Widely known as the Lee-Carter method, the method combines a demographic model of mortality with time series methods of forecasting (Booth et al., 2006). Originally, the authors made use of USA population and for the longest available time series (between 1900 and 1989) and defined log-mortality rates as:

$$\ln m_{x,t} = a_x + b_x * k_t + \varepsilon_{x,t} \quad (5.1)$$

where $m_{x,t}$ is the central mortality rate at age x and time t , a_x the average pattern of mortality by age, b_x the relative speed of mortality change at different ages, and k_t refers to the level of mortality at time t . Consequently, $\varepsilon_{x,t}$ is the set of disturbances that measure the divergence between the model and observed log-mortality rates. Parameter estimation is based on the average $\ln m_{x,t}$ over time for a_x , while b_x and k_t are estimated by singular value decomposition (SVD) (Booth et al., 2006).

Estimates are based on a set of factors for each considered age and time (Torri, 2009), and the described parameters have the same length as the number of ages and defined time periods, being need to add some constrains to parameter estimation

become well succeeded (Camarda, 2008): b_x , as the pattern of deviations from the previous ages as the parameter k_t changes, is constrained to sum to the unity; k_t , as the time varying mortality level index, sum to 0; and consequently a_x are set to equal the means over time of $\ln m_{x,t}$: $a_x = \frac{1}{2} \sum_{t=1}^n \ln m_{x,t}$.

Before forecasting, a second stage estimation of k_t needs to be accomplished, where the parameter is readjusted by refitting to total observed deaths, giving higher weight to ages at which deaths are higher counterbalancing the effect of using log-mortality estimates. Originally, k_t is extrapolated by ARIMA time series model, or more specifically, by a random walk with drift model:

$$k_t = k_{t-1} + d + e_t \quad (5.2)$$

where d is the average annual change in k_t and e_t refers to the uncorrelated errors. In this first model approach, the authors used the rates in the last year of fitting (jump-off year) as the jump-off rates, in opposition with, e.g., what Lee and Miller proposed later, in 2001.

5.2.2. Lee and Miller (2001)

Likewise it was said before, the Lee-Carter methodology was the starting point for many other variants proposed with the intention of getting improved outcomes. Lee and Miller (2001), e.g., proposed an approach that differs from the original one essentially in three ways (Booth et al., 2006): (1) the fitting period was narrowed, and instead of accounting information since 1900, the fitting period is starting now in 1950; (2) k_t is re-estimated by matching observed life expectancy at birth (e_0) rather than the observed number of deaths; and thirdly (3), instead of using the fitted rates as jump-off rates, actual observed rates are considered in the jump-off year. This last step was adopted by the authors to avoid the use of population data as necessarily required for fitting to death counts.

Overall outcome from this variant presents major improvements, mainly because one of the main error source was the mismatch between fitted rates and for the last year of the fitting period and actual death rates in that year (Booth et al., 2006).

5.2.3. Booth, Maindonald and Smith (2002)

The variant proposed by Booth, Maindonald and Smith (BMS) in 2002, introduced, once again some changes based on the original model. Booth et al. (2006), identified, once more, three main changes: (1) the fitting period is chosen based on a routine that identifies the most adequate and suitable fitting period, i.e., the fitting period choice is based on statistical goodness-of-fit criteria under the assumption of a linear period index k_t ; (2) the readjustment of k_t is now based on fitting to the age distribution of deaths, using a Poisson distribution to model death process; and (3) the jump-off rates are now, consequently different from the originals once that they are now taken to be the fitted rates based on the new methodological approach.

The original Lee carter approach lies on the assumption of a linear and constant decline in mortality rates over the fitting period (represented by k_t), however, sometimes that linearity does not hold on the overall period. In addition, the assumption of a constant b_x also increases estimates bias. Restricting the fitting period to maximize the linearity assumption, the BMS method tries to solve linearity issues and obtain more accurate results and the choice of the fitting period is done based on the ratio of the mean deviances of the fit of the original Lee-Carter method to the overall linear fit.

5.2.4. Hyndman and Ullah (2002)

In 2007, Hyndman and Ullah proposed a slightly different extension of the original Lee-Carter method, being expressed by:

$$\ln m_{x,t} = a(x) + \sum_{j=1}^J k_{t,j} b_j(x) + e_t(x) + \sigma_t(x) \varepsilon_{x,t} \quad (5.3)$$

where $a(x)$ is the average level of mortality by age across the different periods (it differs from a_x so that it corresponds to a smooth function of age where age obtained by applying a P-spline regression), $b_j(x)$ is the age coefficient and $k_{t,j}$ the time series coefficient. $k_{t,j}$ and $b_j(x)$ are estimated by using a principal component decomposition. The inclusion of two error terms in the equation allows distinguishing

concerning the differences between the observed rates and the smoothed curves $\sigma_t(x) \varepsilon_{x,t}$; and the difference between the smoothed curves and the fitted curves from the model $e_t(x)$.

Resuming, the Hyndman and Ullah (2007) approach uses the functional data paradigm developed by Ramsay and Silverman (2005) for modeling mortality rates, resulting in the extension of the Lee-Carter method in five main points Booth et al. (2006): (1) smoothed mortality rates are used instead, being estimated by applying nonparametric smoothing methods, as the P-spline regression; (2) more than one set of b_x and k_t is used; (3) different methods for modeling time series are used, as the state space models for exponential smooth; (4) robust estimation is an option in cases of external perturbations, as wars or epidemics, and finally (5) it does not readjust parameter k_t .

5.3. Compositional Data Analysis and the CoDa equivalent Lee-Carter method for mortality forecasting

5.3.1. Compositional Data Analysis (CoDa)

When we are dealing with many vectors of information expressed in percentages or densities, all having the same sum, we end up with compositional data. A composition thus, is defined as a vector of D positive components $x = [x_1, \dots, x_D]$ summing up to a given constant k (Oeppen, 2008; Boogaart and Tolosana-Delgado, 2013).

Aitchison (1986) argued that compositional data is significant for many subjects, revealing frequently noticeable variability from vector to vector. Despite that a typical example of compositional data concerns different subjects as Geology, Chemistry or Economics. Oeppen (2008) introduced it in demographic forecasts by modeling life-table d_x , which always sum to the life-table radix (l_0). The best way to deal with sum-constrained data is to work on the simplex. Nevertheless, working with relative information is not always straightforward, mainly because the unit-sum “*it is either ignored or improperly incorporated into the statistical modeling and from there, results an inadequate or irrelevant analysis with a doubtful or distorted inference*” (Aitchison, 1986), and the solution is employ a log-ratio transformation.

Therefore, the simplex is defined as the set of compositions (closed, once that are summing up to a constant). Therefore, a set of possible closed compositions can be defined as (Boogaart and Tolosana-Delgado, 2013):

$$\begin{aligned} \mathbb{S}^D &:= \left\{ \mathbf{x} = (x_i)_{i=1,\dots,D} : x_i \geq 0, \sum_{i=1}^D x_i = 1 \right\} \\ &= \left\{ \mathbf{x} = (x_1, \dots, x_D) : x_i \geq 0, \sum_{i=1}^D x_i = 1 \right\} \end{aligned} \quad (5.4)$$

i.e., each vector corresponds to a D-part of the *simplex*. Within the simplex, classical algebraic/geometric operations as addition, subtraction or multiplication, need to be replaced by compositional equivalents. Perturbation plays the role of addition and subtraction and is a closed component-wise product of the involved compositions (Oeppen, 2008; Boogaart and Tolosana-Delgado, 2013):

$$x = z \oplus \hat{\xi} = C[z_1 \hat{\xi}_1, \dots, z_D \hat{\xi}_D] \quad (5.5)$$

$$z = x \ominus \hat{\xi} = C \left[\frac{x_1}{\hat{\xi}_1}, \dots, \frac{x_D}{\hat{\xi}_D} \right] \quad (5.6)$$

On both cases, the closure operator ensures a unit sum:

$$C[x_1, \dots, x_D] = \frac{(w_1, \dots, w_D)}{(w_1 + \dots + w_D)} \quad (5.7)$$

Powering or power transformation replaces the product of a vector by a scalar and is defined as the closed powering of the components by a given scalar (Boogaart and Tolosana-Delgado, 2013), i.e., the compositional multiplication:

$$\lambda \otimes x = C[x_1^\lambda, \dots, x_D^\lambda] \quad (5.8)$$

Lastly, the Aitchison scalar product for compositions provides a replacement for the conventional scalar product and is defined as:

$$\langle x, y \rangle_A = \frac{1}{D} \sum_{i>j}^D \ln \frac{x_i}{x_j} \ln \frac{y_i}{y_j} \quad (5.9)$$

Conversely, the set of compositions together with these three operations build a $(D - 1)$ -dimensional Euclidean space structure on the simplex. “*This means that we can translate virtually anything defined for real vectors to compositions, as an Euclidean space is always equivalent to the real space*” (Boogaart and Tolosana-Delgado, 2013). Having a data set of D columns and N rows, where all sum to unit, the centered log-ratio transformation is defined as:

$$CLR(z) = \ln \left[\frac{z_1}{g(z)}, \dots, \frac{z_D}{g(z)} \right] \quad (5.10)$$

and its inverse by:

$$CLR^{-1}(z) = C[\exp(z_1), \dots, \exp(z_D)] \quad (5.11)$$

Centering the same data set is done by getting the composition geometric mean ξ , and subtracting it from each row.

$$\begin{aligned} \xi &= cen(x) = C[\exp\{E(\ln x)\}] \\ &= C[g_1, \dots, g_D] \end{aligned} \quad (5.12)$$

5.3.2. Low rank approximation

A usual task when we have a given matrix \mathbf{A} with m columns and n rows ($m \times n$) is to determine an approximate factorization, i.e., elaborate a low-rank approximation of a matrix. Imagine that the given matrix \mathbf{A} can be approximated by:

$$\underset{m \times n}{\mathbf{A}} \approx \underset{m \times r}{\mathbf{B}} \underset{r \times n}{\mathbf{C}} \quad (5.13)$$

where the inner dimension r is called the numerical approximation of a matrix, and then a ran- r approximation of the matrix can be computed.

One way of decomposition of a $m \times n$ matrix, real or complex, is to proceed a *singular value decomposition* (SVD):

$$\mathbf{A} = \mathbf{U}\Sigma\mathbf{V}^* \quad (5.14)$$

where \mathbf{U} corresponds to a $m \times r$ unitary matrix (\mathbf{U} columns are called the *left singular vectors*), \mathbf{V} is other $r \times n$ unitary matrix (\mathbf{V} columns are called the *right singular vectors*), and Σ is a strictly positive $r \times r$ diagonal matrix with s_{\square} singular values:

$$\Sigma = \begin{bmatrix} s_1 & & \\ & \ddots & \\ & & s_r \end{bmatrix} \quad (5.15)$$

Thus, assuming that \mathbf{A} corresponds to a compositional data matrix, applying the compositional operators above defined, we get:

$$A_n^{(r)} = \hat{\xi} \oplus (U_{n1}s_1 \otimes V_1) \oplus \dots \oplus (U_{nr}s_r \otimes V_r), \quad r < D \quad (5.16)$$

where U correspond to the left singular vectors, s to the singular values, and finally V to the centered log ratio of the transposed right singular vectors.

5.3.3. The Lee-Carter CoDa equivalent mortality forecasting model

The CoDa equivalent to the Lee-Carter method for forecasting mortality trends suggested by Oeppen (2008) allows not only forecasting mortality trends for overall changes mortality rates (*single-decrement forecast*), but also disaggregate those by cause of death (*multiple-decrement forecast*). In opposition to most known approaches, Oeppen suggested using the life-table distribution of deaths d_x and once that the sum equals the life-table radix (l_0), we end up with compositional data. d_x for overall mortality and d_x^i for cause-specific mortality can be obtained by single and

multiple-decrement life-tables, respectively. Following Preston et al. (2001), we have that:

$$\sum_i {}_n d_x^i = {}_n d_n \quad (5.17)$$

and

$$\sum_x \sum_i {}_n d_x^i = 1 \quad (5.18)$$

Despite the existence of some studies ensuring that independent forecast of cause of death mortality do not produce plausible results (e.g., Wilmoth, 1995), the constraint imposed by the sum to the unit of each of the d_x^i compositional vector, ensures that “*changes in the density by age and cause have to be compensated by changes in other ages and causes*” (Oeppen, 2008).

The suggested method can be summarized in the following steps (Oeppen, 2008):

- I. Construction of a matrix A , with $N * D$ size of the $d_{x,t}$, where t corresponds to the study years ($t = 1 \dots N$) organized by row, and x to ages ($x = 1 \dots D$) organized by column. As explained before, each row of matrix A corresponds to the constrained sum of 1 (the life-table radix).
- II. Center the matrix A by calculating the age-specific and subtracting it from each row by using a CoDa operator. Centering the matrix provides a better visualization of the structure. After this procedure, we end up with a different matrix B .
- III. The next step is to transform matrix B into the real space, obtaining consequently a matrix C , by calculating the centered log-ratios of each row.
- IV. Then, we proceed to the decomposition of matrix C by applying SVD decomposition.
- V. Construct the low rank- r approximation of matrix C , where the forecasted rows are already included.

- VI. At this step we transform back the matrix into compositional data and for that we use the inverse of the centered log-ratio, ending up with matrix B^* .
- VII. Finally, using again a CoDa operator, we add back the geometric means, obtaining not only the a low rank compositional matrix A^* with fitted $\hat{d}_{x,t}$, but also with the forecast.

Likewise Oeppen (2008) suggested, in our study, since that modeling is focused on $d_{x,t}$ it seems to have no reason to adjust the left singular vector to match average life expectancy or deaths in total or by age, or scaling the right singular vectors as they sum to zero. In opposition with the original Lee-Carter method, while defining its CoDa equivalent, it was not found evidence for using an ARIMA model (0,1,0), but rather an ARIMA (0,2,2), which offers a better fit to cause of death mortality trends.

5.4. Data

Data on *overall death counts* $D(x,y)$ and *exposures* $E(x,y)$ derived from the *Human Mortality Database* (HMD, 2014: www.mortality.org) and *deaths by COD* $D_i(x,y)$ from the *World Health Organization Mortality Database* (WHOMD, 2014).

Like it was explained on the previous chapters, WHOMD cause of death (COD) data is only available by five-year age groups. In this chapter longer time series were needed to produce a more accurate forecast, thus, our choice fell on the combination of ICD9 and ICD10 COD classification. Disruptions between both classifications should be almost imperceptible once that major changes correspond to large COD desegregation and we are dealing with major CODs groups.

Again, to avoid low subpopulation sizes and any lack of representativeness, and now big disruptions between ICD classifications, we work with major COD groups: 1) Neoplasms, 2) Ischaemic Heart Diseases; 3) Cerebrovascular Diseases; 4) Remaining Diseases of the Circulatory System; 5) Diseases of the Respiratory System; 6) Diseases of the Digestive System; 7) External Causes of death; and 8) Remaining Causes of death.

We used five selected countries to follow a concise and complementary analysis throughout entire monograph, correspond once again to France, Portugal,

Japan, Sweden and Spain. Portugal, is a special case here, once that on the WHOMD Portuguese data for both ICDs presents some changes in the classification over time, we make use of EUROSTAT, where COD data is available between 1994 and 2010.

Consequently, due to data issues, Portugal is here the country with the smallest time series (Table 5.1), while Japan (1979 to 2011), France (1979 to 2011) and Spain (1980 to 2012) present the largest ones. Lastly, Sweden is in intermediary position, with data available between 1987 and 2012.

Table 5.1: Discrimination of International Classification of Diseases (ICD) by Country

Countries	ICD 9	ICD 10
Portugal	—	1994 - 2010
Spain	1980 - 1998	1999 - 2011
Sweden	1987 - 1996	1997 - 2010
France	1979 - 1994	1995 - 2011
Japan	1979 - 2000	2001 - 2011

Source: WHOMD and EUROSTAT 2014, own elaboration

5.5. Results

5.5.1. Distribution of deaths by cause across age

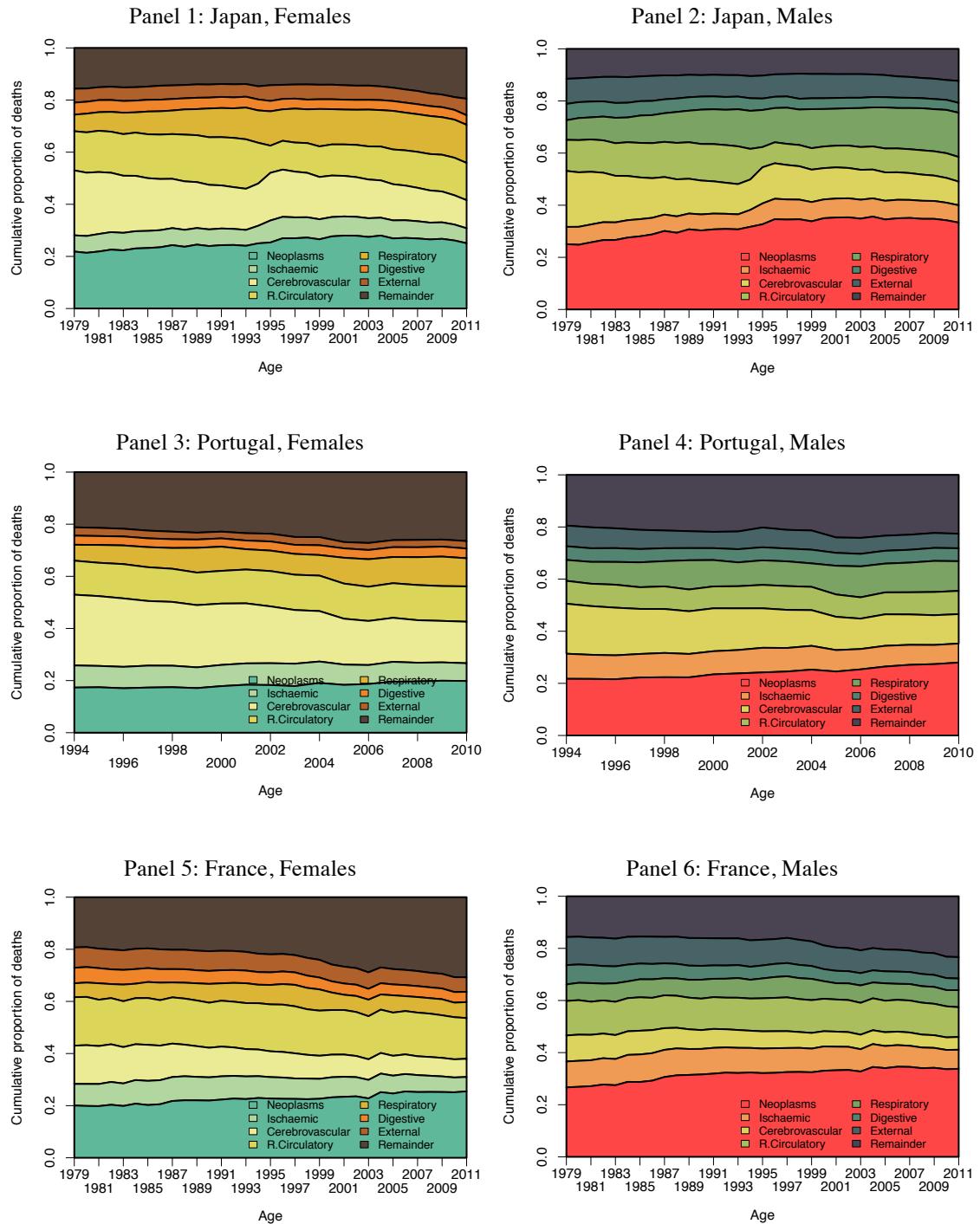
The elaboration of a Stack plot (Figure 5.1) allows not only to identify the impact of the different CODs by sex and country, but also to identify possible disruptions caused by changes in the ICD classificatory system. We can immediately identify that Japan is the country with the biggest disruption in 1995, when the ICD10 data starts. This disruption is especially identified related to diseases of the circulatory system (here represented by: ischaemic heart diseases, cerebrovascular diseases, and remaining causes of the circulatory system).

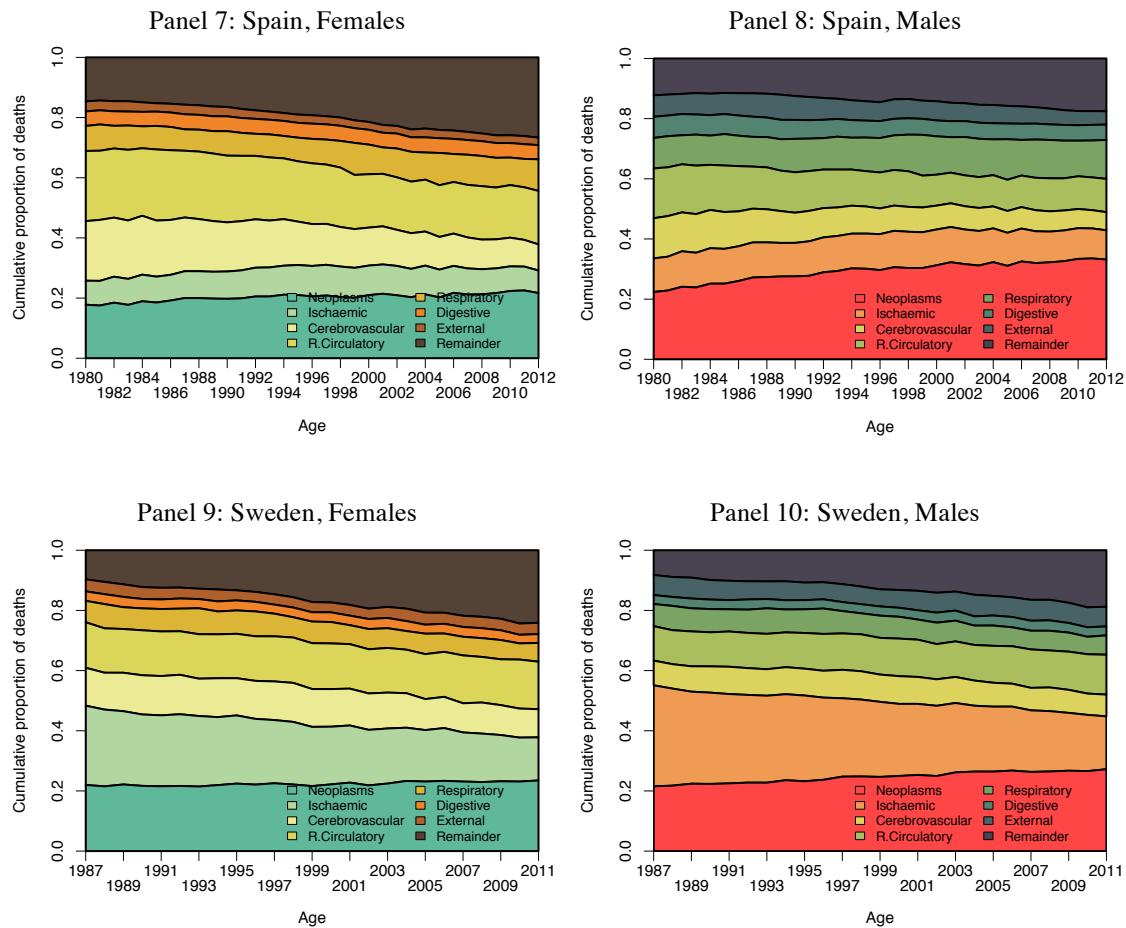
France, even in a much smaller scale, is one other country that can be identified possible perturbing disruptions in what concerns the forecasting process. It seems that till year 2000, almost all COD groups besides neoplasms and remaining causes, were declining and after that year onwards their proportion on overall mortality increased.

All the other countries present much less disruption when the ICD change occurs (Table 5.1), but is still curious to observe that Portugal, that only refers to ICD

10, also presents some changes in the observed patterns consistent with changes in the classification of diseases.

Figure 5.1: Cumulative proportion of deaths across time by COD





Source: WHOMD, HMD and EUROSTAT 2014, own elaboration

Analyzing now Figure 5.1 distribution of deaths by cause and not only identifying possible disruptions caused by changes in ICD, it is possible to identify three main general facts common to all countries:

- 1) in the past were the diseases of the circulatory system that played the major role, i.e., contributed to overall mortality with the higher number of deaths;
- 2) deaths caused by neoplasms were already numerous in the beginning of each time series, and its influence seems to grow as time goes by;
- 3) in the beginning of each time series, deaths were mainly caused by circulatory system diseases and neoplasms, but now it is possible to identify a raising influence of a mixture of remaining causes that had a smaller negative impact.

5.5.2. The singular vector

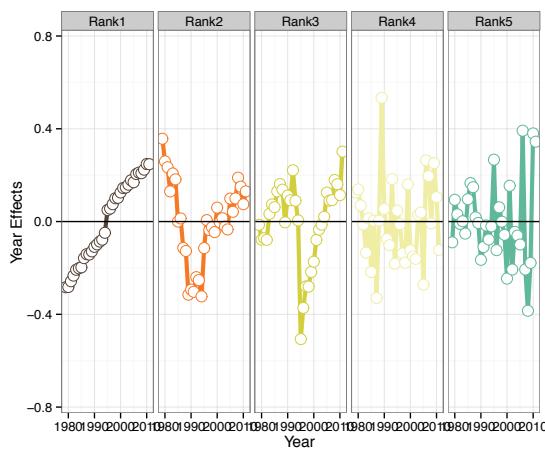
As it was explained above, the CoDa equivalent Lee-Carter model to forecast mortality is, similarly to the original, based on a low rank matrix approximation. In this case however, we can define the rank-r approximation to use. Figure 5.2 represents the first five left singular vectors by sex and country to “help” us to decide which low rank to apply. Vectors presenting identifiable patterns rather than random noise suggest that these vectors still have an important contribution to the model. Lets analyze the Japanese example. Independently of sex, it can be seen that from the fourth vector onwards, we are only able to identify basically a random noise. Consequently, Japanese forecasts are based on a rank-3 matrix approximation.

The Spanish case is one other example of a rank-3 choice for matrix approximation, once that the same pattern identified for Japan is repeating here, and especially in the male case.

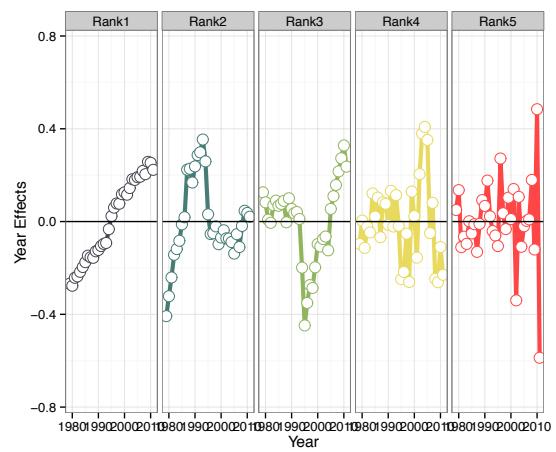
In a general way, it seems that for remaining three countries (Portugal, France and Sweden), a rank-2 approximation is the best choice. In the Swedish female specific case, it even seems that a rank-1 approximation would be enough, but once that we are dealing with a time series that includes two different ICDs (and a shorter one for Portugal), our choice was to perform a rank-2 approximation for those countries.

Figure 5.2: First five left singular vectors by sex and country

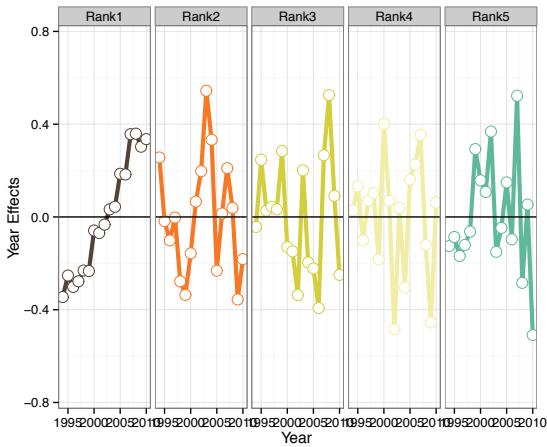
Panel 1: Japan, Females



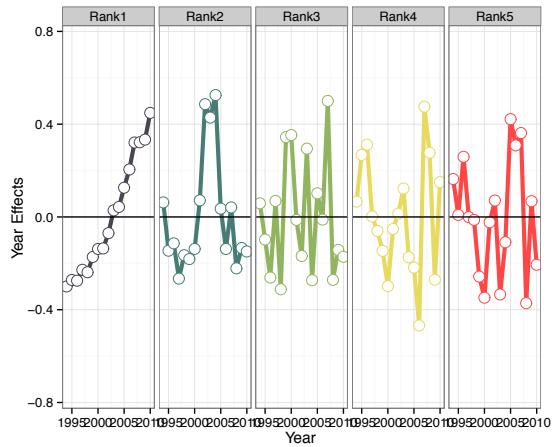
Panel 2: Japan, Males



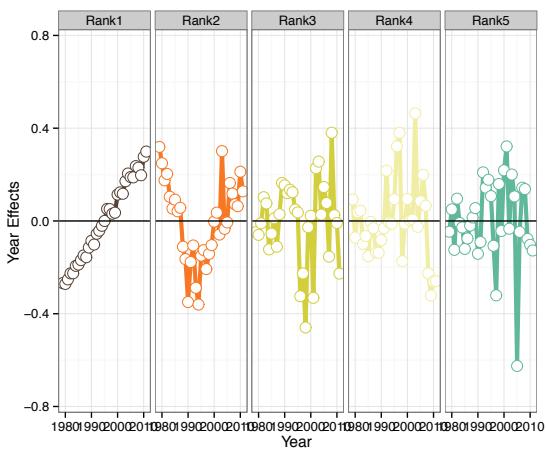
Panel 3: Portugal, Females



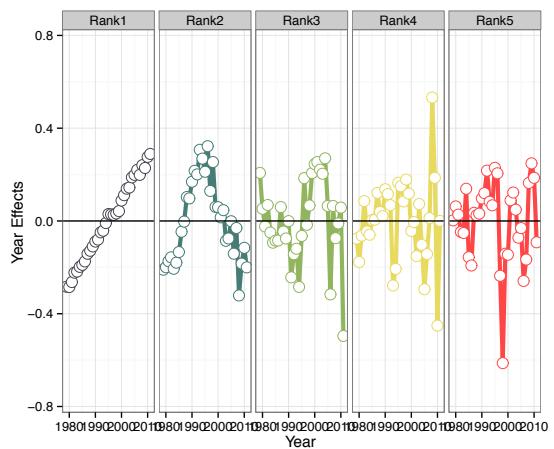
Panel 4: Portugal, Males



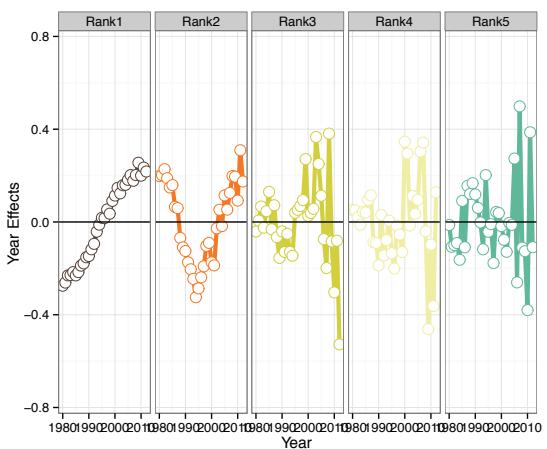
Panel 5: France, Females



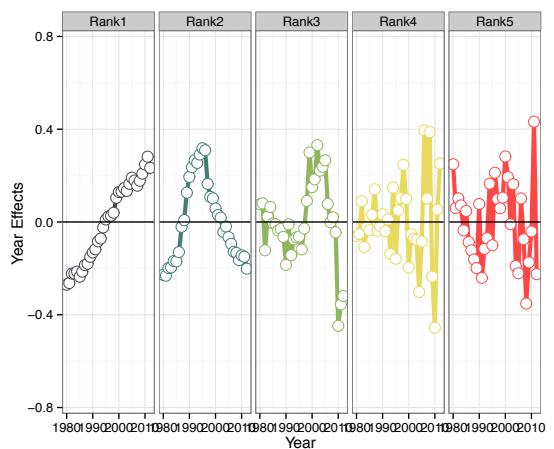
Panel 6: France, Males



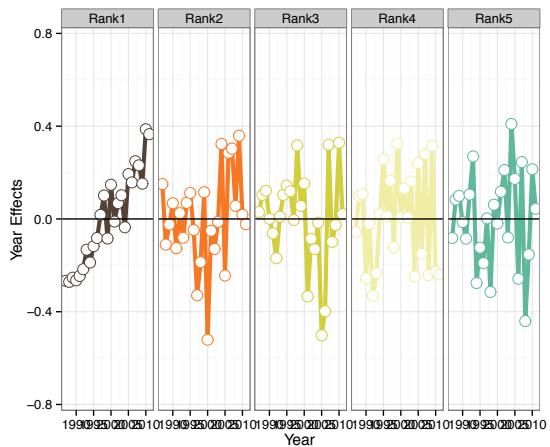
Panel 7: Spain, Females



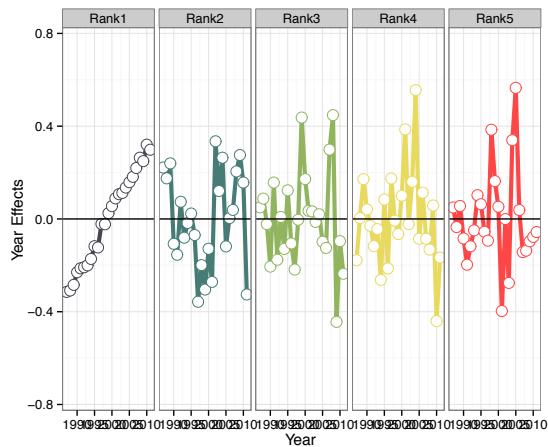
Panel 8: Spain, Males



Panel 9: Sweden, Females



Panel 10: Sweden, Males



Source: WHOMD, HMD and EUROSTAT 2014, own elaboration

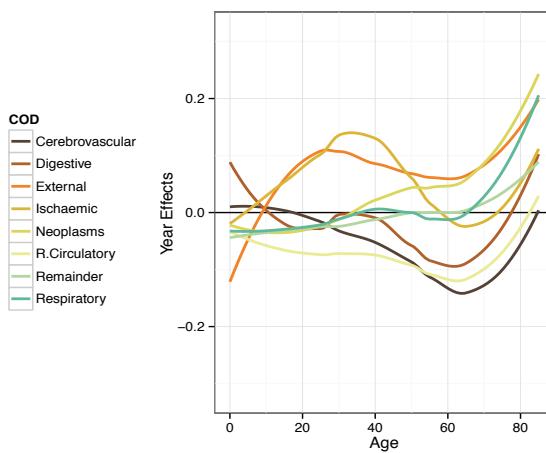
Data presented in Figure 5.3 correspond to the first age right singular vectors, i.e., to the age factors for the all analyzed CODs. This information is presented here deriving from LOESS³ smoothing curves to allow a better understanding of obtained results. When the presented values are positive, it indicates an increase of deaths related to the associated COD, while negative values are result of exactly the opposite.

Thus, the obtained results confirm what was identified before, recognizing Neoplasms and the group corresponding to the remaining CODs as the ones that mostly increase, especially after age 20. At older ages was registered an increase of deaths referent to almost all causes. This situation is related with the fact that nowadays, deaths are occurring mainly at older ages and even a small “positive” influence is detected.

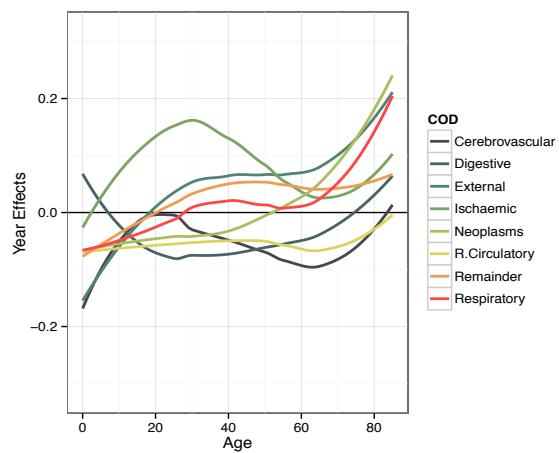
³ LOESS is a powerful non-parametric but simple method for fitting smooth curves to data.

Figure 5.3: First right singular vectors by sex and country (*loess smooth*)

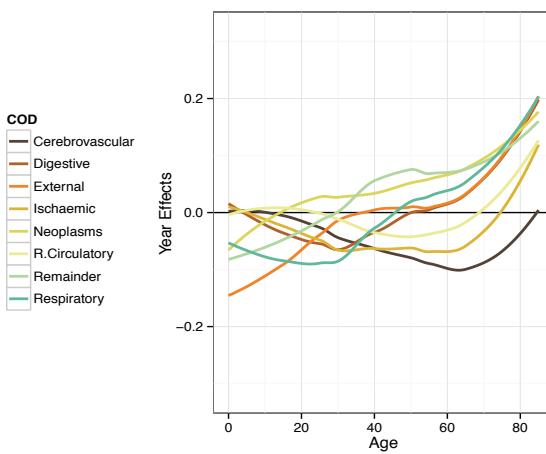
Panel 1: Japan, Females



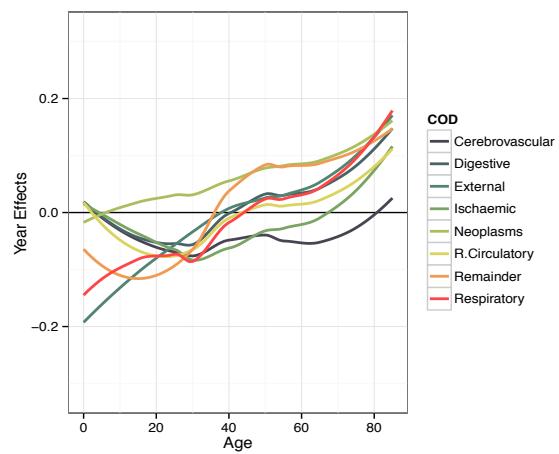
Panel 2: Japan, Males



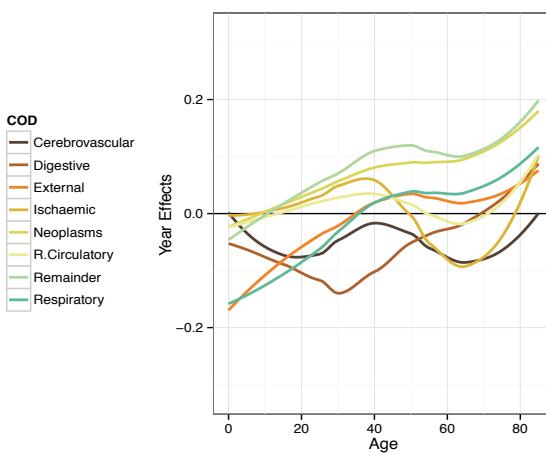
Panel 3: Portugal, Females



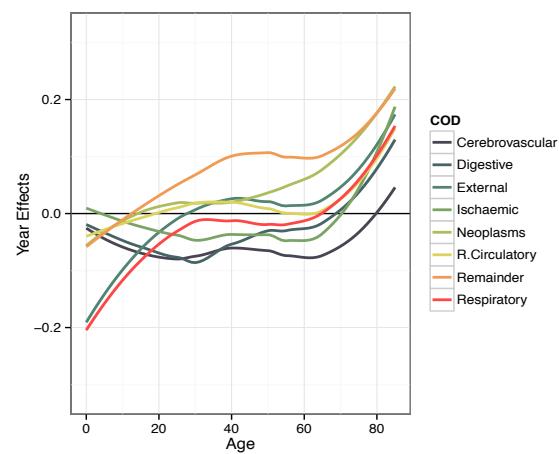
Panel 4: Portugal, Males



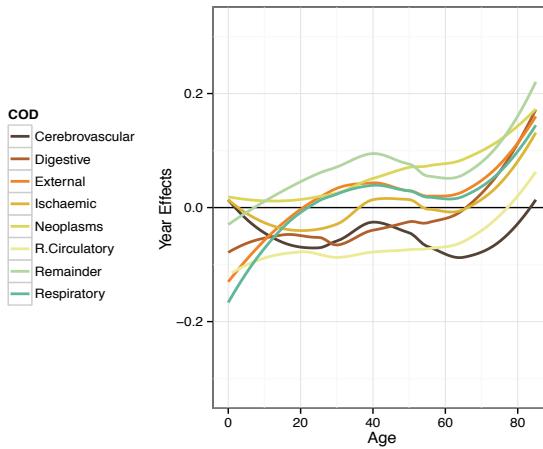
Panel 5: France, Females



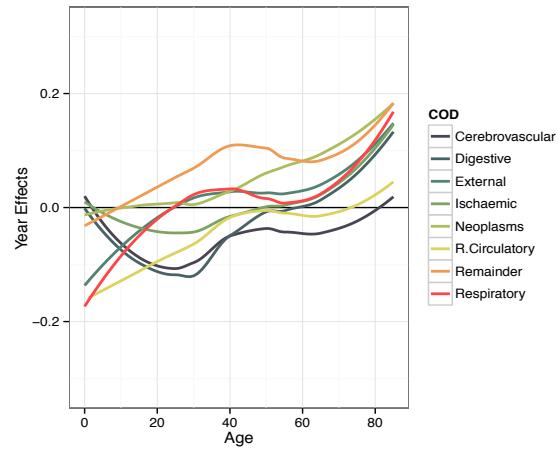
Panel 6: France, Males



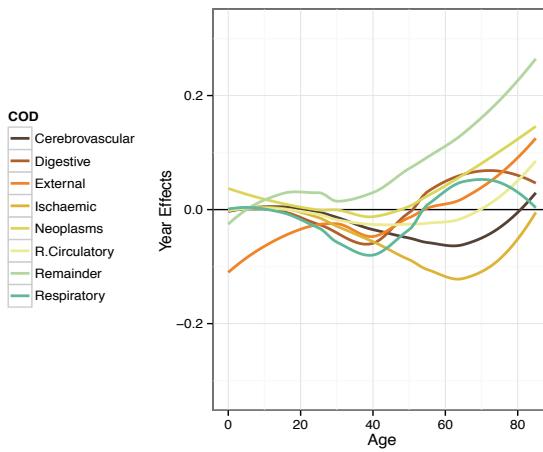
Panel 7: Spain, Females



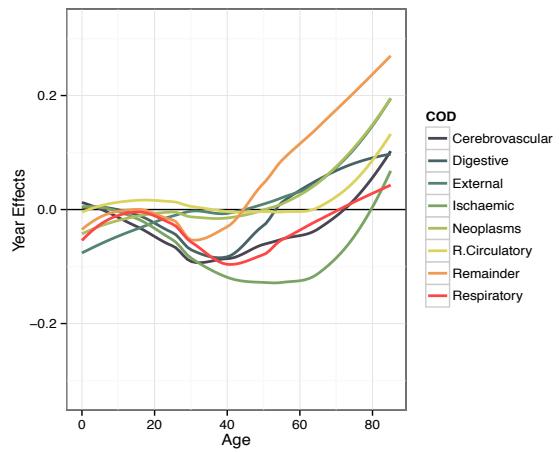
Panel 8: Spain, Males



Panel 9: Sweden, Females



Panel 10: Sweden, Males



Source: WHOMD, HMD and EUROSTAT 2014, own elaboration

5.5.3. Decomposing mortality forecasts by COD

Figure 5.4 shows the period life-table probability that a newborn has to die from a specific cause, where circles correspond to the used data to fit the model (observed), continuous lines represent the rank-r approximation (identified above), and dashed lines are the forecasted trends.

Generally it's discernible that the low rank approximation used accordingly to each country, presents very alike results, even in presence of very high fluctuations as the one observed in the Japanese situation. Nevertheless, it seems that those fluctuations interfere with the forecasts and in some CODs present very unconformable results. The clearest example is related to the remaining causes of death group, to which real data is presenting an increasing pattern over time, and the

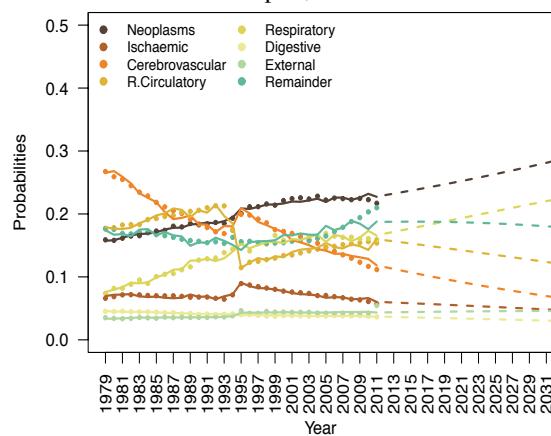
forecast predicts a decline. However, problems with forecasts associated to neoplasms and respiratory diseases are also recorded.

For all other countries, it's possible to realize that forecasted trends follow previous evolution and seem to progress accordingly. Obtained results suggest that the probability of dying from neoplasms is going to be kept constant over time, but there are very different CODs (i.e., the remaining COD group) that need some intervention once that they are already leading death probabilities for females in France, Spain and Sweden.

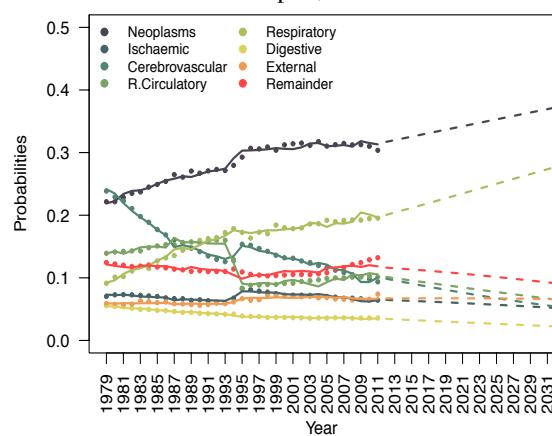
From the group from the selected analyzed countries, Portugal presents distinctive evolutionary patterns from the rest. The increasing mortality pattern associated to neoplasms is similar to the other countries, however, more than the remaining group of CODs, it seems that if not controlled, the diseases of the respiratory system will become one of the biggest death contributors.

Figure 5.4: Period life-table probability at birth of dying from a specific COD

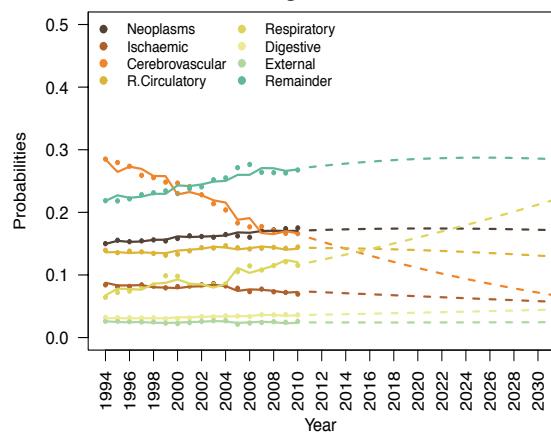
Panel 1: Japan, Females



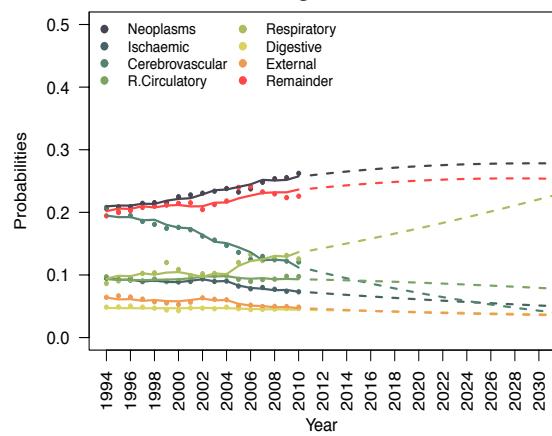
Panel 2: Japan, Males



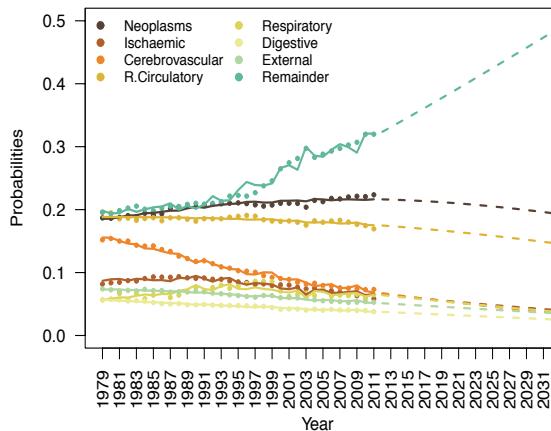
Panel 3: Portugal, Females



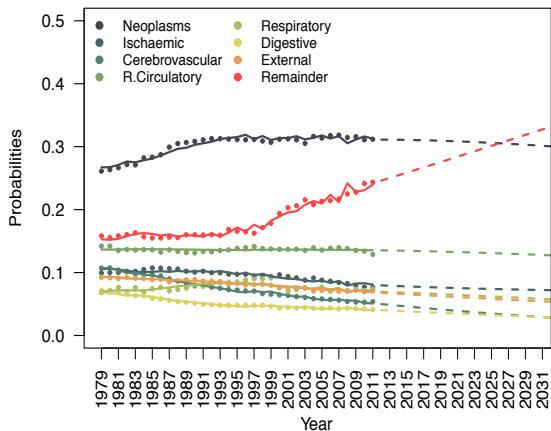
Panel 4: Portugal, Males



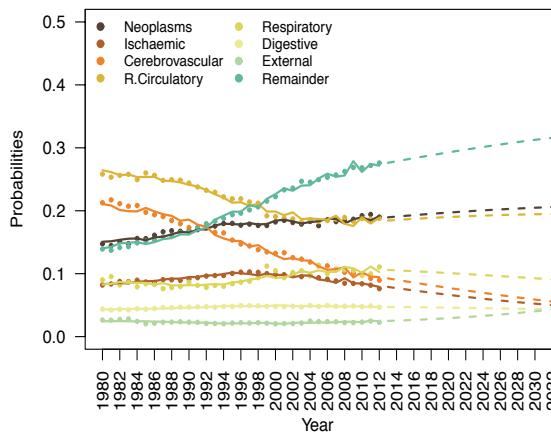
Panel 5: France, Females



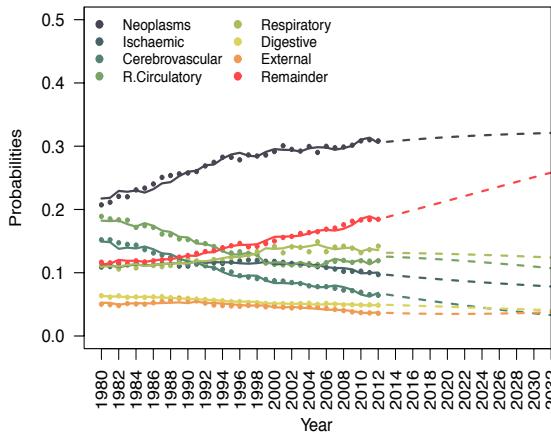
Panel 6: France, Males



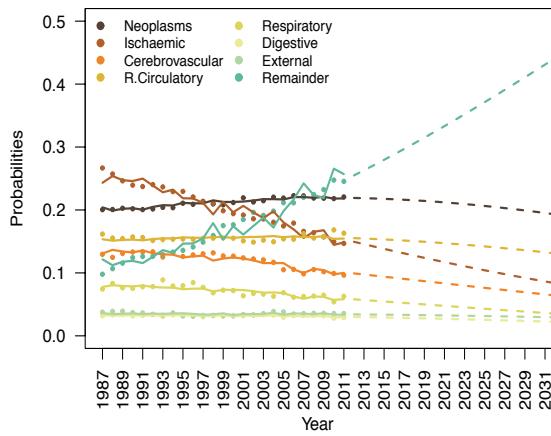
Panel 7: Spain, Females



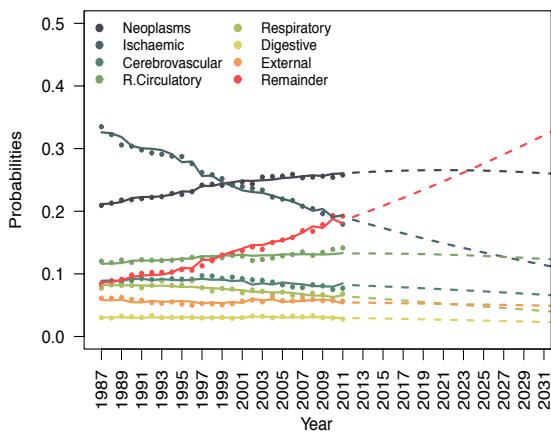
Panel 8: Spain, Males



Panel 9: Sweden, Females



Panel 10: Sweden, Males



Source: WHOMD, HMD and EUROSTAT 2014, own elaboration

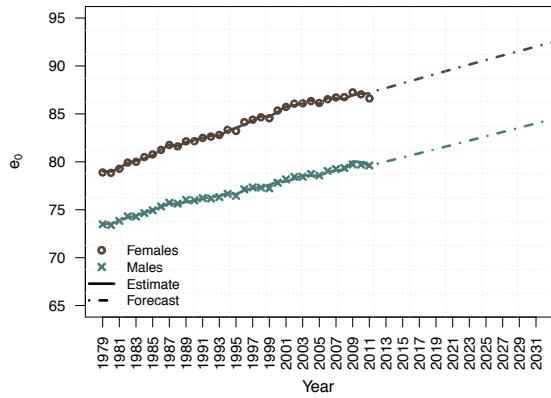
5.5.4. Prospective e_0

Figure 5.5 presents the life expectancy at birth estimated (continuous lines) and forecasted (dashed lines) from the multiple-decrement approach for all the countries under observation and by sex. Obtained results suggest that the multiple-decrement model is able to estimate with high accuracy the already observed life expectancy at birth.

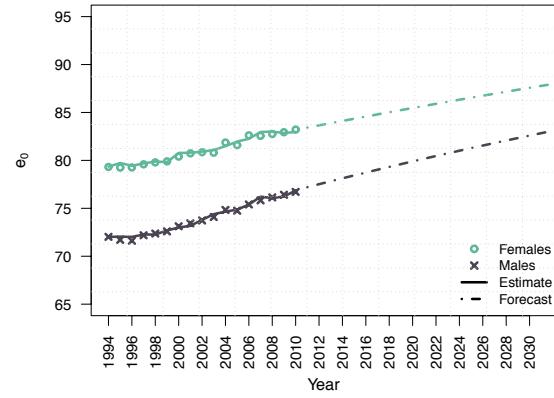
From an overall point of view, the obtained results suggest that the life expectancy at birth gap that differentiate both sexes tends to decline with time. Japan is the only country that seems to contradict this observation.

Figure 5.5: Observed and forecast e_0 by country and sex

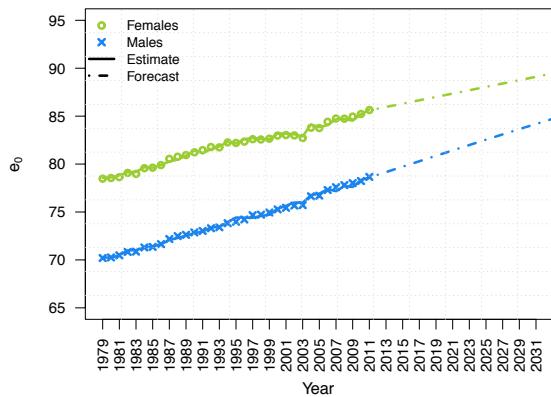
Panel 1: Japan



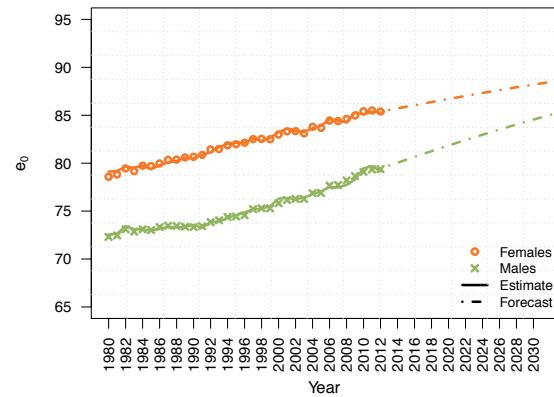
Panel 2: Portugal



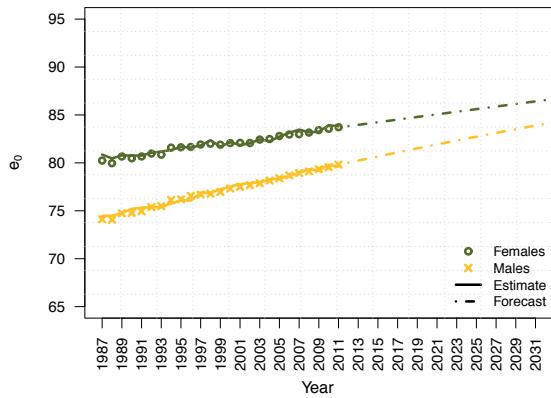
Panel 3: France



Panel 4: Spain



Panel 5: Sweden



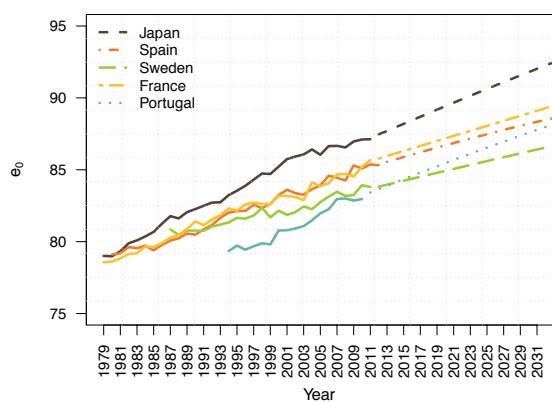
Source: WHOMD, HMD and EUROSTAT 2014, own elaboration

Comparing forecasted e_0 within sexes (Figure 5.6) demonstrates that if forecasts became reality, Japanese females will be still leading in 2030 and all the other countries will find difficulties to reduce the gap. We also can expect that Portuguese females leave the bottom and around 2030 present a very narrow gap when compared with France and Spain.

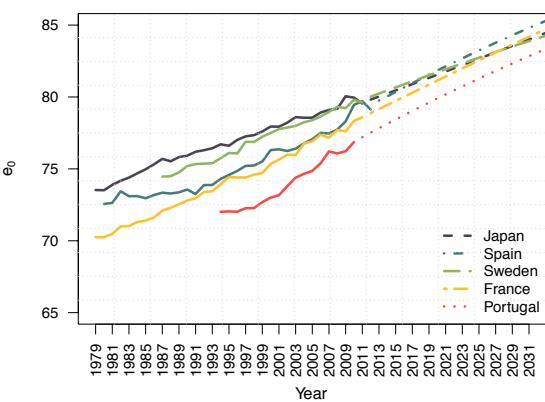
Male forecasts indicate that the slope of increase suggests a steeper evolution of life expectancy, but here, Portugal is not expected to leave the bottom. Spain, on the contrary, is expected to take the lead around 2023.

Figure 5.6: e_0 prospective patterns aggregated by sex

Panel 1: Females



Panel 2: Males



Source: WHOMD, HMD and EUROSTAT 2014, own elaboration

5.6. Discussion

The CoDa equivalent to the Lee-Carter model to forecast mortality trends proposed by Oeppen (2008) is definitely an excellent option to consider when the goal is to forecast multiple decrement processes as mortality by COD.

Obtained estimates for observed values concerning life expectancy at birth and the period life-table probability at birth of dying from a specific COD, revealed high accuracy and are not more pessimistic as identified for other methods by Wilmoth (1995). Nevertheless, in opposition with the original study when the method was proposed (Oeppen, 2008), it seems that forecasted trends for Japan are not conclusive and not accurate enough. Nevertheless, despite the accuracy presented by the model here employed, the true is that most of COD forecasts seem not reliable at this level.

At our knowledge, accuracy might be mainly connected with the fact that the jump-off is not being corrected, likewise some above explained variants of the original methodology. This thought arises from the fact that the CoDa approach has difficulty to estimate accurately most recent patterns. In the female Japanese case, e.g., the model is completely overestimating observed life expectancy at birth for 2011. Once again, if we take another careful look to the forecast decomposed by COD, the same situation is registered, and the extrapolation of past trends seems to be more accurate if the last available year of information is fitted well.

Smoothing the data before model fitting also may bring higher accuracy to estimates once that variance is reduced. Nonetheless, we guess that the major cause for inaccuracy problems related with this method results from changes in the ICD.

Disaggregation of causes of death into single-age groups could also benefit further estimates and forecast of multiple-decrement mortality patterns.

5.7. Conclusions

The CoDa model produces undeniable good approximates for observed mortality patterns and overall results indicate that, in most of the countries, there is a distinctive group of remaining cause of death with growing implications on overall mortality. Neoplasms do not seem to be expected to reduce their participation on overall mortality, and a constant pattern of evolution is extrapolated. Together with

neoplasms in both sexes, is expected that the remaining diseases of the circulatory system not specified here, continue to negatively influence female mortality.

Portugal was the only country under observation where is expected to observe an increase on the probability of dying from respiratory system diseases, however, it appears not influencing negatively the evolution of life expectancy at birth, mainly in the female case.

CHAPTER 6

CONCLUSIONS

6.1. Overall conclusions

As exposed in the Introduction Chapter (Chapter 1), the overall aim of this study is to contribute to mortality research with important knowledge that comes from investigating mortality trends and its related processes.

As a result, we can confirm that likewise some other previous studies: neoplasms, diseases of the circulatory and respiratory system are the principal CODs and the ones that influence the most, negatively, life expectancy for populations. However, we perspective that if the rate of increase in life expectancy at birth keeps recent evolutionary patterns, it is possible that the male/female gap keep diminishing and all analyzed countries can aim for Japanese values. Despite fewer attention given to less expressive causes in overall mortality, we identify that the group of remaining CODs is one of the most important in influencing negatively life expectancy. Likewise Oliveira and Mendes acknowledged (2010) the emergent importance associated with this wide-ranging group of CODs might be intimately related with shift of high rates of mortality to older ages.

Seeking to test Vaupel's hypothesis on the individual rate of mortality increase, we were also able to distinguish between the individual's rate of aging, and the widely studied LAR (e.g., Horiuchi and Wilmoth, 1997, 1998). The two measures present very distinctive patterns. The individual rate of aging is constant across time, and LAR is varying age-wise, creating a bell-shaped pattern. b not only differs from LAR, but also contributes to its estimation (Vaupel and Zhang, 2010). Despite the fairly stable evolution estimated b across different CODs, sexes and selected countries, we cannot fully support Vaupel's hypothesis. With the exception of neoplasms, the estimates obtained for overall mortality are always lower when compared with different $\hat{b}_i(y)$ and it seems that the different $\hat{b}_i(y)$ associated to different CODs contribute to the "average" rate of individual aging. Accordingly with Brody and Schneider (1986), our results present lower rates of aging for causes considered close to non-senescent processes, like neoplasms or external causes of

death, and higher values for the ones in opposite direction, i.e., for example the case of the deaths by diseases of the circulatory system. The case of cancer strikes out as the cause that has the lowest rate of aging of all, factor that can be explained by the fact that “*in the human species, the population that reaches advanced age and has a decreased incidence of cancer, could be less prone to develop cancer and hence more fit to reach the maximum lifespan*” (Macieira-Coelho, 1986). Another explanation can be directly related with the origin of cancer, which is connected with the unregulated growth of new cells, being a multistage process that starts with a pre-cancerous lesion and ends up with a malignant tumor. Nevertheless, at older ages the proliferation of cells is declining and possibly increases the time between cancer development stages (Ukrainsteva and Yashin, 2003).

Γ GM model-based LARs (for overall mortality and by COD) appear to be consistent with the “heterogeneity hypothesis” advanced by Horiuchi and Wilmoth (1998) as the age of mortality deceleration shifts to older ages with time in overall mortality and only neoplasms, as one of the CODs with lower mortality rates after age 65, presents less pronounced patterns. The Γ GM model-based LAR (Vaupel and Zhang 2010) fits observed LAR with high accuracy and captures the connection relationship between the observed shift and the rate of life expectancy increase in the five selected countries for overall mortality.

The CoDa equivalent to the Lee-Carter model to forecast mortality trends proposed by Oeppen (2008) is definitely an excellent option to consider when the purpose is to forecast multiple decrement processes as mortality by COD. Obtained estimates for observed values concerning life expectancy at birth and the period life-table probability at birth of dying from a specific COD, revealed high accuracy and are not more pessimistic as identified for other methods by Wilmoth (1995). Forecasts identified a distinctive group of remaining cause of death with growing implications on overall mortality. Neoplasms do not seem to be expected to reduce their participation on overall mortality, and a constant pattern of evolution is extrapolated.

Portugal was the only country under observation where is expected to observe an increase on the probability of dying from respiratory system diseases, however, it appears not influencing negatively the evolution of life expectancy at birth, mainly in the female case.

Summarizing, despite that the main motivation of this study did not revealed full confirmation, we strongly believe that the analysis complementary to all the subject resulted in precious information that may contribute to changes in specific areas seeking to improve public health.

The constant rate of aging across all individuals, under the same COD was proved to be a reality. But if proved to be real for the individual exposed to all competing risks and with different behaviors (e.g., smokers vs. non-smokers), it “*would fundamentally contribute to our understanding of how and why we age*” (Vaupel, 2010). Our findings are contributing, at least partially, on this direction. We also provide a complete and in-depth examination of the population by applying the formulation on (4.6), showing that the rate of individual aging not only differs from the rate of aging for the population, but also contributes for its calculation.

6.2. Insights and contributions for future research

Any investigation, even when is not able to confirm previously stated hypothesis, gives important contributions to future research. In our case, even that the obtained results cannot fulfill Vaupel’s hypothesis, we understand that, due to the different contributing risks to which all individuals are exposed (Chiang, 1991), a possible complementary way is testing by the estimate of the individual rate of individual aging accordingly to groups of individuals that share the same behavioral pattern, e.g., smokers and non-smokers; athletes and non-athletes; or, alcohol consumption and non-consumption individuals. Disaggregation of causes of death into single-age groups can benefit further estimations.

In what concerns to the performed mortality forecasts, at our knowledge, accuracy might be essentially connected with the fact that the jump-off is not being rectified likewise some above explained variants of the original methodology, and that should be evaluated. Smoothing the data before model fitting also may bring higher accuracy to estimates once that variance is reduced. Nonetheless, we guess that the major cause for inaccuracy problems related with this method come from changes in the ICD.

6.3. Limitations of the study

During the study elaboration, many difficulties were found and almost all are related with data availability. First of all, data available for mortality discriminated by COD, only exists in the aggregate form of five-year age groups. Second, the open age interval for those kind of data is almost always 85+, and with increasing lifespan, it is not enough and might interfere with the obtained results. Thirdly, when we disaggregate data by COD and sex, we end up with smaller data subpopulations, increasing the estimates variance. This situation becomes even worst when we are focusing on countries with small population, as e.g. Portugal or Finland. Fourthly, changes between ICD in the different countries resulted in disruptions in the correspondent year of transition that seem to influence the outcome.

Nonetheless, we also can refer here some theoretical limitations, or better, limitations that will always need to be in mind when we are dealing with CODs. Some CODs are intimately connected with behavioral patterns, for example, around 30 % of neoplasms “*are due to five leading behavioral and dietary risks: high body mass index, low fruit and vegetable intake, lack of physical activity, tobacco use, alcohol use*” (WHO, 2014). Another issue is related to the existence of competing risks to which each individual is exposed that need always to be taken into account.

REFERENCES

- Aitchison, J. (1986). The statistical analysis of compositional data. London: Chapman and Hall.
- Akaike, H. (1973). Information theory and an extension of the maximum likelihood principle. B. Petrov and F. Czaki (Eds.), Second International Symposium on Information Theory, Budapest, Hungary.
- Arriaga, E. (1984). Measuring and explaining the change in life expectancies, *Demography* 21: 83-96.
- Baudisch, A. (2008). Inevitable Aging? Contributions to evolutionary-demographic theory. Demographic Research Monographs Series, Springer.
- Beard, R.E. (1959). *Note on some mathematical mortality models*. Wolstenholme G.E.W., O'Connor M. (eds.) Little, Boston: Brown and Company, 302-311.
- Bongaarts, J. and Feeney, G. (1993). Estimating mean lifetime. *PNAS* 100, 13127-13133.
- Boogaart K.G. van den and Tolosana-Delgado R. (2013). Analyzing compositional data with R. Springer.
- Booth, H., R. J. Hyndman, L. Tickle, and P. de Jong (2006). Lee-Carter mortality forecasting: a multi-country comparison of variants and extensions. *Demographic Research* 15 , 289{310.
- Booth, H., Maindonald, J. and Smith, L. (2002). Applying Lee-Carter under conditions of variable mortality decline. *Population Studies* 56(3), 325-336.
- Booth, H. and Tickle, L. (2008). Mortality modelling and forecasting: A review of methods. *Annals of Actuarial Science* 3(1-2), 3{43.
- Brass, W. (1971). On the scale of mortality. In: W. Brass (Ed.), Biological Aspect of Demography. London: Taylor & Francis.
- Breslow, N. (1984). Extra-Poisson Variation in Log-Linear Models. *Applied Statistics* 33.
- Brillinger, D.R. (1986). The natural variability of vital rates and associated statistics. *Biometrics*, 693-734.
- Brody, J.A. and Schneider, E.L. (1986). Diseases and disorders of aging: an hypothesis. *Journal of Chronic Diseases* 39, 871-8 Oeppen Oeppen 76.
- Camarda, C.G. (2008). Smoothing methods for the analysis of mortality development. PhD Thesis: Universidad Carlos III de Madrid, Department of Statistics.

- Camarda, C.G. (2012). MortalitySmooth: An R package for smoothing Poisson counts with p-splines. *Journal of statistical software*, 50-1.
- Cameron, A.C. and P.K. Trivedi (1986). Econometric Models Based on Count Data: Comparisons and Applications of Some Estimators and Tests. *Journal of Applied Econometrics* 1.
- Canudas-Romo, V., Glei, D., Gómez-Redondo, R., Coelho, E. and Boe, C. (2008). Mortality changes in the Iberian Peninsula in the last decades of the twentieth century. *Population-E*, 63 (2), 319-344.
- Canudas-Romo, V. (2010). Three measures of longevity: time trends and record values. *Demography*, 47(2), 299-312.
- Carey, J.R. and Liedo, P. (1998). Sex-specific life table aging rates in large medfly cohorts. *Experimental Gerontology* 30, 315-325.
- Chiang, C.L. (1991). Competing risks in mortality analysis. *Annual Review of Public Health* Vol. 12, 2881-307.
- Clayton, D. and Schifflers, E. (1987). Models for temporal variation in cancer rates. II: Age-period-cohort models. *Statistics in Medicine* 6, 469-481.
- Comfort, A. (1964). Aging: The Biology of Senescence. Routledge & Kegan Paul, London.
- Currie, I.D., Durban, M. and Eilers, P.H.C. (2004). Smoothing and Forecasting Mortality Rates. *Statistical Modelling* 4.
- De Gray, D.N.J., (2005). The rate of aging: a counterproductively undefinable term. *Rejuvenation Research* 8(2), 77.
- De Magalhães, J., Cabral, J. and Magalhães, D. (2005). The influence of genes on the aging process of mice: a statistical assessment of the genetics of aging. *Genetics* 169(1), 265-274.
- Eilers P.H.C. and Marx, B.D. (1996). Flexible Smoothing with B-splines and Penalties (with discussion). *Statistical Science* 11 (2).
- Eilers, P.H.C. and Marx, B.D. (2002b). Multivariate calibration with temperature interaction using two-dimensional penalized signal regression. *Chemometrics and Intelligent Laboratory Systems* 66.
- Engel, L.W., Strauchen, J.A., Chiazzé, L. Jr. and Heid, M. (1980). Accuracy of death certification in an autopsied population with specific attention to malignant

- neoplasms and vascular diseases. *American Journal of Epidemiology*, 111(1), 99-112.
- EUROSTAT (2014). Cause of death statistics. Available at http://epp.eurostat.ec.europa.eu/statistics_explained/index.php/Causes_of_death_statistics#Further_Eurostat_information (acceded on September 2014).
- Fernandes, A.A. (2007). Determinantes da mortalidade e da longevidade:Portugal numa perspectiva europeia. Análise Social, vol XLII (183).
- Finch, C.E. (1990). Longevity, Senescence, and the Genome. The University of Chicago Press, Chicago and London.
- Finkelstein, M.S. and Esaulova, V. (2006). Asymptotic behavior of a general class of mixture failure rates. *Advances in Applied Probability* 38, 242-262.
- Flurkey, K., Papaconstantinou, J., Miller, R. and Harrison, D. (2001). Lifespan extension and delayed immune and collagen aging in mutant mice with defects growth hormone production. *Proceedings of the National Academy of Sciences of the United States of America* 98(12), 6736-6741.
- Gampe, J. (2010). Human mortality beyond age 110. In: H. Maier, J.G.B. Jeune, J.M. Robine, and J.W. Vaupel (eds), *Supercentenarians*, Heidelberg, Springer, 219-230.
- Gavrilova, N.S. and Gavrilov L.A. (2014). Mortality revisited: using the LAR approach. PAA, Boston.
- German, R.R., Fink, A.K., Heron, M., Stewart, S.L., Johnson, C.J., Finch, J.L., Yin, D. and the Accuracy of Cancer Mortality Study Group (2011). The accuracy of cancer mortality statistics based on death certificates in the United States. *Cancer Epidemiology* 35(2), 126-131.
- Girosi, F. and King, G. (2007). Understanding the Lee-Carter Mortality Forecasting Method. Technical Report. Rand Corporation.
- 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. *Philosophal Transactions of the Royal Society of London* 115, 513-585.
- Hamilton, W.D. (1966). The moulding of senescence by natural selection. *Journal of Theoretical Biology*, 12:12-45.
- Heligman, L. and Pollard, J.H. (1980). The age pattern of mortality. *Journal of the Institute of Actuaries* 107(1), 49-80.
- HMD (2014). The Human Mortality Database. <http://www.mortality.org/>.

- Horiuchi, S., Cheung, S.L.K. and Robine, J.M. (2012). Cause of death decomposition of old-age mortality compression. In: 2012 annual meeting of the Population Association of America (PAA).
- Horiuchi, S. and Coale, A.J. (1990). Age patterns of mortality for older women: An analysis using the age-specific rate of mortality change with age. *Mathematical Population Studies* 2(4), 245-267.
- Horiuchi, S. and Wilmoth, J.R. (1997). Age patterns of the life table aging rate for major causes of death in Japan, 1951-1990. *Journal of Gerontology, Biological Sciences* 52A(1), B67-B77.
- Horiuchi, S. and Wilmoth, J.R. (1998). Deceleration in the age pattern of mortality at older ages. *Demography* 35(4), 391-412.
- Hyndman, R.J., Booth, H. and Yasmeen, F. (2013). Coherent mortality forecasting: the product-ratio method with functional time series models. *Demography* 50(1), 261-283.
- Hyndman, R.J. and Ullah, M.S. (2007). Robust forecasting of mortality and fertility rates: a functional data approach. *Computational Statistics and Data Analysis* 51(10), 4942-4956.
- Japan Statistical Yearbook 2014 (2014). Statistics Bureau, Ministry of International Affairs and Communications. Available at <http://www.stat.go.jp/english/data/nenkan/index.htm> (accessed on September 2014).
- Johnson, T. (1990). Increased life-span of age-1 mutants in *Caenorhabditis elegans* and lower Gompertz rate of aging. *Science* 249(4871), 908-912.
- Kannisto, V. (2001). Mode and Dispersion of the Length of Life. Population: An English Selection 13.
- Kircher, T., Nelson, J. and Burdo, H. (1985). The autopsy as a measure of accuracy of the death certificate. *New England Journal of Medicine* 313(20), 1263-1269.
- Lee, R.D. and Carter, L.R. (1992). Modeling and forecasting U.S. Mortality. *Journal of the American Statistical Association* 87(419), 659-671.
- Lee, E.D. and Miller, T. (2001). Evaluating the performance of the Lee-Carter method for forecasting mortality. *Demography* 38(4), 537-549.
- Lilienfeld, D.E. and Stolley, P.D. (1994). *Foundations of Epidemiology*. Oxford University Press, New York.

- Lin, Y.J., Seroude, L. and Benzer, S. (1998). Extended life-span and stress resistance in the *Drosophila* mutant methuselah. *Science* 282, 943-946.
- Macieira-Coelho, A. (1986). Cancer and aging. *Experimental Gerontology* 21(6), 483-495.
- Mair, W., Goymer, P., Pletcher, S.D. and Partridge, L. (2003). Demography of dietary restriction and death on *Drosophila*. *Science* 301(5640), 1731-1733.
- Makeham, W.M. (1860). On the law of mortality. *Journal of the institute of actuaries* 13, 283-287.
- Meslé, F. (2006). Causes of death among the oldest-old: validity and comparability. In: J.M. Robine, E.M. Crimmins, S. Horiuchi, and Z. Yi (eds), *Human longevity, individual life duration, and the growth of oldest-old population*, International Studies in Population, Volume 4, 191-214.
- Meslé, F. and Vallin, J. (2006). The health transition: trends and prospects. In: Caselli G., Vallin J. and Wunsch G. Eds. *Demography, analysis and synthesis. A treatise in demography*. New York: Elsevier, 247–602.
- Missov, T.I. and Nemeth, L. (2014). How wrong could parameter estimation be? Statistical consequences of fitting the wrong model to human mortality data. Presented at the 2014 European Population Conference, Budapest, Hungary.
- Missov, T.I. and Ribeiro, F. (2015). Do Individuals age at the same rate? Findings from cause of death data”. (under review)
- Missov, T.I. and Vaupel, J.W. (2015). Mortality implications of mortality plateaus. *SIAM Review* 57.
- Missov T.I. and Finkelstein, M. (2011). Admissible mixing distributions for a general class of mixture survival models with known asymptotics. *Theoretical population biology* 80.
- Modelmog, D., Rahlenbeck, S. and Trichopoulos, D. (1992). Accuracy of death certificates: a population-based, complete-coverage, one-year autopsy study in East Germany. *Cancer Causes Control*, Volume 3, 541-546.
- Morais, M.G. (2002). Causas de Morte no Século XX: Transição e Estruturas da Mortalidade em Portugal Continental. Edições Colibri and CIDEHUS.UE, Lisboa.
- Muggeo, V. (2003). Estimating regression models with unknown break-points. *Statistics in Medicine* 22, 3055-3071.

- Oeppen, J. (2008). Coherent forecasting of multiple-decrement life tables: a test using Japanese cause-of-death data. In *European Population Conference 2008*. European Association for Population Studies, July.
- Oeppen, J. and Vaupel, J.W. (2002). Broken limits to life expectancy. *Science* 296, 1029-1031.
- Oliveira, I.T. and Mendes, M.F. (2010). A diferença de esperança de vida entre homens e mulheres; Portugal de 1940 a 2007. *Análise Social*, vol. XLV (194), 115-138.
- Oliveira, M. M., Afonso, A. and Filipe, P. (1994). Perfil da mortalidade por causas de morte para os distritos de Portugal. In *Actas do XII congresso da Sociedade Portuguesa de Estatística*, Évora.
- Omran, A.R. (1971). The epidemiologic transition: a theory of the epidemiology of population change. *The Milbank Memorial Fund Quarterly*, vol. 49(4), 509-538.
- Perks, W. (1932). On some experiments in the graduation of mortality statistics. *Journal of the Institute of Actuaries* 63, 12-40.
- Preston, S.H. Heuveline, P. and Guillot, M., (2001). Demography: Measuring and Modeling Population Processes, Oxford: Blackwell Publishers Ltd. Ryder.
- Ramsay J.O. and Silverman B.W. (2005). Functional Data Analysis. 2nd edition. Springer-Verlag, New York.
- Rau, R., Soroko, E., Jasilionis, D. and Vaupel, J.W. (2008). Continued reductions in mortality at advanced ages. *Population and Development Review* 34, 747-768.
- Rodriguez, G. (2006). Demographic translation and tempo effects: an accelerated failure time perspective. *Demographic Research* 14, 85-110.
- Rozing, M. and Westerndorp, R. (2008). Parallel lines: nothing has changed? *Aging Cell* 7(6), 924-927.
- Salinari, G. and Santis, G.D. (2014). Comparing the rate of senescence across time and space. *Population* 69, 191-216.
- Santana, P. (2005). Saúde e Morte em Portugal. Estudo da Mortalidade “Evitável”. II Encontro Português de Demografia. Demografia e População: os novos desafios, FCG Lisboa.
- Schwarz G. (1978). Estimating the dimension of a model. *The Annals of Statistics*, 6-2.

- Schottenfeld, D., Eaton, M., Sommers, S.C., Alonso, D.R. and Wilkinson, C. (1982). The autopsy as a measure of accuracy of the death certificate. *Bulletin of the New York Academy of Medicine* 58(9), 778-794.
- Shkolnikov, V., Valkonen, T., Begun A. and Andreev, E. (2001). Measuring inter-group inequalities in length of life. *GENUS*, LVII (n°34), 33-62.
- Steinsaltz, D.R. and Wachter, K.W. (2006). Understanding Mortality Rate Deceleration and Heterogeneity. *Mathematical Population Studies* 13.
- Thatcher, A.R. (1999). The long-term pattern of adult mortality and the highest attained age (with discussion). *Journal of the Institute of Royal Statistical Society* 127(1), 5-43.
- Torri, T. (2009): Assessment and transfer of the longevity risk: new methods and applications. Ph.D. Dissertation. University of Rome “La Sapienza”.
- Tuljapurkar, S., Li, N. and Boe, C. (2000). A universal pattern of mortality decline in G7 countries. *Nature* 405, 789-792.
- Ukraintseva, S.V. and Yashin, A.I. (2003). Individual aging and cancer risk: how are they related? *Demographic Research* 9, Article 8, 163-196.
- Vaupel, J.W. (2010). Biodemography of human aging. *Nature* 464(7288), 536-542.
- Vaupel, J.W. (2002). Life expectancy at current rates vs current conditions: a reflexion stimulated by Bongaarts and Feeney's "How long do we live?". *Demographic Research* 7, 365-378.
- Vaupel, J.W., Manton, K. and Stallard, E. (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography* 16, 855-860.
- Vaupel, J.W. and Missov, T.I. (2014). Unobserved population heterogeneity: a review of formal relationships. *Demographic Research* 31, 659-686.
- Vaupel, J.W. and Yashin, A.I. (1985). Heterogeneity's ruses: some surprising effects of selection on population dynamics. *The American Statistician* 39, 176-185.
- Vaupel, J.W. and Zhang, Z. (2010). Attrition in heterogeneous cohorts. *Demographic Research* 23, Article 26, 737-748.
- Wilmoth, J.R. (1995). Are mortality projections always more pessimistic when disaggregated by cause of death? *Mathematical Population Studies* 5 (4),
- Wilmoth, J.R., Andreev, K., Jdanov, D.A., Glei, D.A., Boe, C., Bubeheim, M., Philipov, D., Shkolnikov, V. and Vachon, V. (2007). Methods protocol for the Human Mortality Database.

<http://www.mortality.org/Public/Docs/MethodsProtocol.pdf>.

World Health Organization (2014, November 18). Media Centre Fact Sheet N°297.

Retrieved from: <http://www.who.int/mediacentre/factsheets/fs297/en/>.

WHOMD (2014). The World Health Organization Mortality Database.

http://www.who.int/healthinfo/mortality_data/en/.

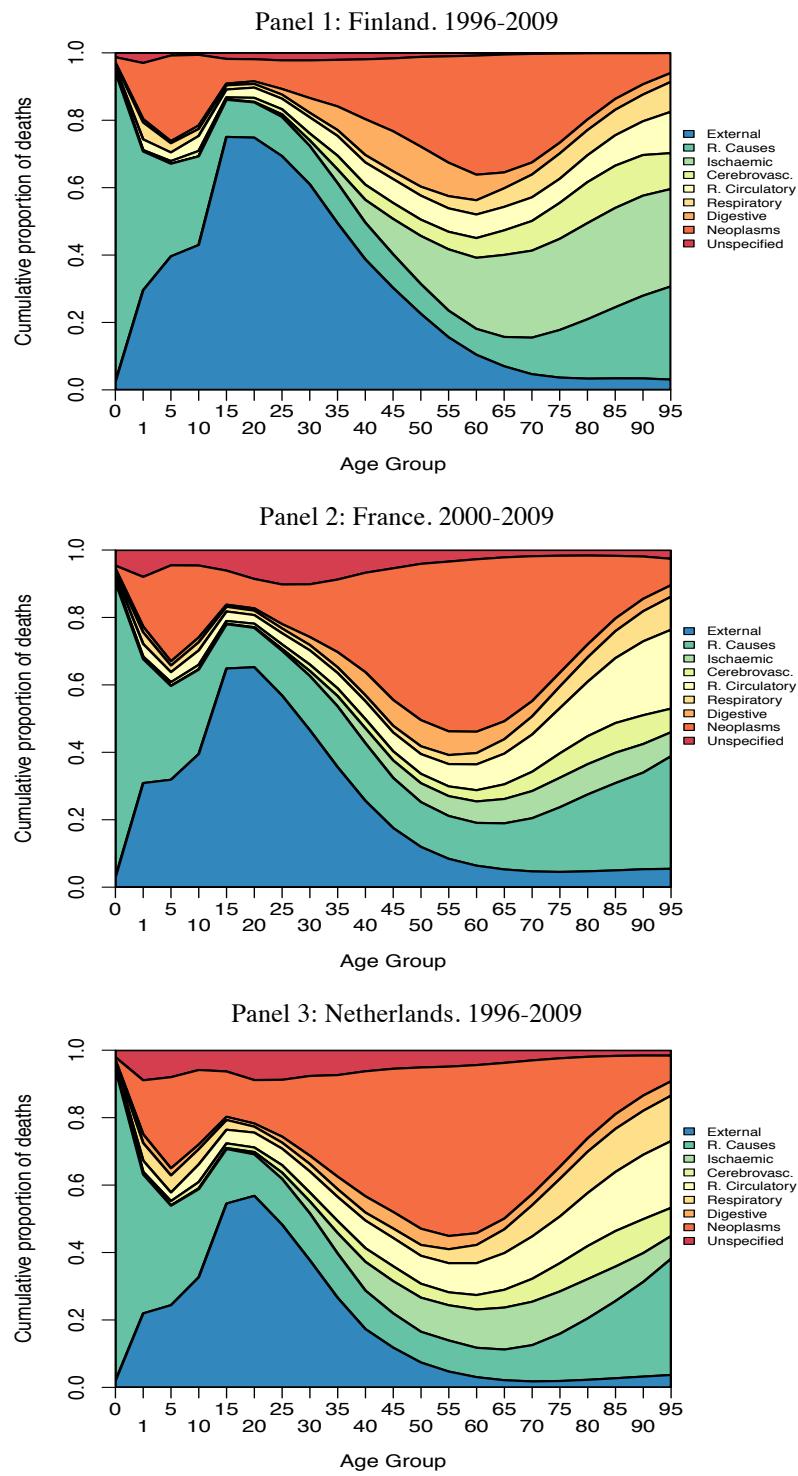
World Health Organization (1977). Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death, based on the recommendations of the Ninth Revision Conference, 1975. Geneva: World Health Organization (WHO).

Zarulli, V. (2012). Mortality shocks and the human rate of aging. *Max Planck Institute for Demographic Research Working Paper* 2012-019.

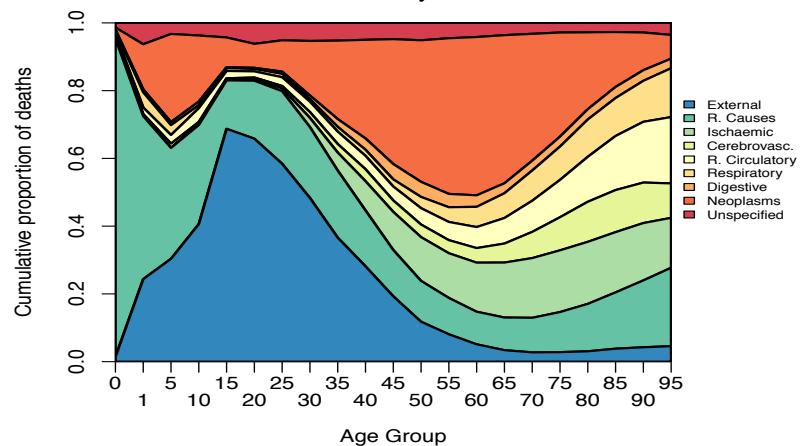
Appendix

To Chapter 3 – The Individual rate of aging by cause of death: testing Vaupel's hypothesis

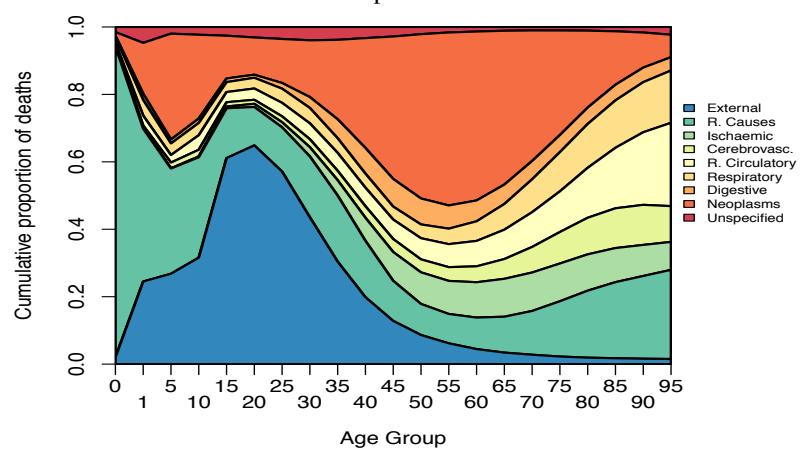
Figure A. 1: Proportion of age-specific deaths by cause



Panel 4: Norway. 1996-2009



Panel 5: Spain. 2003-2009



Source: WHOMD 2014, own elaboration

Table A. 1: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for neoplasms mortality in Spain

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00339	0,00046	0,076	0,007	0,281	0,0716	0,00000010	0,00109416
2004	0,00323	0,00044	0,081	0,008	0,379	0,0768	0,00000003	0,00180696
2005	0,00326	0,00044	0,077	0,007	0,336	0,0729	0,00000018	0,00077657
2006	0,00318	0,00035	0,081	0,006	0,402	0,0642	0,00002167	0,00037077
2007	0,00310	0,00032	0,082	0,006	0,424	0,0625	0,00000006	0,00096547
2008	0,00302	0,00031	0,083	0,006	0,431	0,0644	0,00000009	0,00079030
2009	0,00308	0,00033	0,079	0,006	0,323	0,0625	0,00000001	0,00221177

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00753	0,00142	0,085	0,012	0,326	0,0825	0,00130855	0,00147083
2004	0,00729	0,00113	0,088	0,010	0,380	0,0717	0,00126775	0,00117839
2005	0,00557	0,00081	0,105	0,010	0,510	0,0764	0,00280427	0,00085590
2006	0,00785	0,00122	0,080	0,009	0,307	0,0647	0,00032538	0,00126022
2007	0,00795	0,00111	0,082	0,008	0,351	0,0617	0,00000026	0,00160098
2008	0,00761	0,00092	0,085	0,007	0,386	0,0568	0,00000286	0,00095604
2009	0,00437	0,00059	0,118	0,010	0,642	0,0827	0,00363643	0,00063979

Source: WHOMD and HMD 2014, own elaboration

Table A. 2: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for ischaemic heart diseases mortality in Spain

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00075	0,00006	0,148	0,006	0,424	0,0681	0,00000001	0,00044633
2004	0,00069	0,00005	0,150	0,006	0,448	0,0666	0,00000000	0,00129817
2005	0,00062	0,00004	0,159	0,006	0,554	0,0674	0,00000000	0,00075806
2006	0,00057	0,00004	0,155	0,006	0,472	0,0692	0,00000001	0,00047005
2007	0,00058	0,00004	0,147	0,005	0,314	0,0659	0,00000000	0,00068815
2008	0,00053	0,00004	0,150	0,005	0,367	0,0659	0,00000000	0,00085026
2009	0,00046	0,00003	0,155	0,005	0,355	0,0612	0,00000000	0,00054517

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00255	0,00042	0,101	0,012	0,157	0,0834	0,00000924	0,00043587
2004	0,00140	0,00019	0,143	0,011	0,443	0,0859	0,00095129	0,00021672
2005	0,00202	0,00036	0,111	0,013	0,216	0,0935	0,00031747	0,00038137
2006	0,00140	0,00021	0,132	0,012	0,360	0,0916	0,00074560	0,00023573
2007	0,00200	0,00041	0,102	0,014	0,105	0,1042	0,00000026	0,00060271
2008	0,00181	0,00031	0,104	0,012	0,102	0,0906	0,00000005	0,00099896
2009	0,00164	0,00046	0,114	0,021	0,213	0,1604	0,00005535	0,00048675

Source: WHOMD and HMD 2014, own elaboration

Table A. 3: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for cerebrovascular diseases mortality in Spain

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00059	0,00003	0,185	0,004	0,673	0,0533	0,00000000	0,00046301
2004	0,00056	0,00003	0,180	0,005	0,642	0,0575	0,00000000	0,00065174
2005	0,00054	0,00003	0,179	0,005	0,607	0,0574	0,00000000	0,00081648
2006	0,00051	0,00003	0,173	0,004	0,513	0,0560	0,00000001	0,00033212
2007	0,00050	0,00003	0,171	0,004	0,493	0,0561	0,00000000	0,00087809
2008	0,00047	0,00003	0,171	0,004	0,500	0,0577	0,00000000	0,00047876
2009	0,00045	0,00002	0,163	0,004	0,301	0,0524	0,00000000	0,00050953

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00111	0,00012	0,154	0,009	0,375	0,0727	0,00000007	0,00035200
2004	0,00093	0,00012	0,159	0,011	0,421	0,0857	0,00011719	0,00013403
2005	0,00100	0,00012	0,153	0,010	0,373	0,0814	0,00000019	0,00022252
2006	0,00092	0,00016	0,148	0,015	0,302	0,1140	0,00000001	0,00137042
2007	0,00091	0,00011	0,150	0,010	0,361	0,0843	0,00000269	0,00012633
2008	0,00078	0,00011	0,158	0,012	0,455	0,0996	0,00008255	0,00012373
2009	0,00072	0,00009	0,160	0,011	0,445	0,0965	0,00006033	0,00010828

Source: WHOMD and HMD 2014, own elaboration

Table A. 4: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for remaining circulatory system diseases mortality in Spain

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00080	0,00004	0,172	0,004	0,215	0,0485	0,00000001	0,00043717
2004	0,00077	0,00004	0,168	0,004	0,165	0,0473	0,00000000	0,00086714
2005	0,00077	0,00004	0,170	0,004	0,183	0,0471	0,00000000	0,00101342
2006	0,00069	0,00003	0,173	0,004	0,232	0,0468	0,00000001	0,00031879
2007	0,00072	0,00003	0,169	0,003	0,156	0,0426	0,00000000	0,00156180
2008	0,00071	0,00003	0,169	0,003	0,134	0,0419	0,00000000	0,00086651
2009	0,00065	0,00002	0,171	0,003	0,198	0,0392	0,00000000	0,00206201

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00128	0,00015	0,139	0,010	0,000	0,0723	0,00030004	0,00017275
2004	0,00124	0,00015	0,140	0,010	0,037	0,0747	0,00026726	0,00017122
2005	0,00116	0,00014	0,145	0,010	0,051	0,0735	0,00030340	0,00015661
2006	0,00113	0,00015	0,141	0,011	0,031	0,0825	0,00026114	0,00017019
2007	0,00129	0,00016	0,135	0,010	0,000	0,0789	0,00007501	0,00017971
2008	0,00126	0,00015	0,135	0,010	0,011	0,0783	0,00008794	0,00017047
2009	0,00098	0,00011	0,150	0,010	0,127	0,0793	0,00034841	0,00013031

Source: WHOMD and HMD 2014, own elaboration

Table A. 5: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for respiratory system diseases mortality in Spain

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00045	0,00003	0,179	0,005	0,350	0,0622	0,00000000	0,00069918
2004	0,00040	0,00003	0,173	0,005	0,291	0,0661	0,00000001	0,00033285
2005	0,00046	0,00003	0,178	0,005	0,338	0,0589	0,00000001	0,00037466
2006	0,00038	0,00002	0,169	0,005	0,166	0,0611	0,00000000	0,00091057
2007	0,00043	0,00002	0,167	0,004	0,174	0,0572	0,00000001	0,00031291
2008	0,00041	0,00002	0,167	0,004	0,121	0,0554	0,00000000	0,00115766
2009	0,00038	0,00002	0,167	0,004	0,104	0,0517	0,00000000	0,00147060

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00114	0,00010	0,199	0,008	0,679	0,0615	0,00064334	0,0012435
2004	0,00148	0,00013	0,161	0,008	0,343	0,0619	0,00000005	0,00049797
2005	0,00170	0,00014	0,158	0,007	0,315	0,0572	0,00000002	0,00078011
2006	0,00138	0,00013	0,155	0,008	0,265	0,0644	0,00000228	0,00015006
2007	0,00144	0,00013	0,162	0,008	0,366	0,0624	0,00000004	0,00051051
2008	0,00133	0,00012	0,161	0,008	0,319	0,0639	0,00000006	0,00039651
2009	0,00132	0,00011	0,157	0,007	0,301	0,0620	0,00000007	0,00034135

Source: WHOMD and HMD 2014, own elaboration

Table A. 6: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for digestive system diseases of mortality in Spain

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00041	0,00005	0,138	0,009	0,361	0,1021	0,00000000	0,00115913
2004	0,00008	0,00000	0,215	0,004	0,670	0,0816	0,00051691	0,00002073
2005	0,00008	0,00000	0,215	0,004	0,670	0,0828	0,00051691	0,00002158
2006	0,00008	0,00000	0,215	0,005	0,670	0,0833	0,00051691	0,00002271
2007	0,00037	0,00004	0,134	0,008	0,233	0,0970	0,00000002	0,00026553
2008	0,00035	0,00003	0,138	0,007	0,284	0,0870	0,00000000	0,00084763
2009	0,00035	0,00003	0,135	0,007	0,269	0,0865	0,00000332	0,00004178

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00090	0,00030	0,097	0,023	0,000	0,1525	0,00024386	0,00031640
2004	0,00060	0,00017	0,123	0,021	0,148	0,1550	0,00049103	0,00018665
2005	0,00057	0,00015	0,132	0,020	0,302	0,1478	0,00050957	0,00016770
2006	0,00068	0,00022	0,104	0,023	0,000	0,1579	0,00037701	0,00023405
2007	0,00086	0,00030	0,094	0,024	0,000	0,1655	0,00014564	0,00031591
2008	0,00066	0,00021	0,111	0,023	0,133	0,1699	0,00028383	0,00022128
2009	0,00047	0,00012	0,134	0,020	0,327	0,1625	0,00048262	0,00013748

Source: WHOMD and HMD 2014, own elaboration

Table A. 7: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for external causes of mortality in Spain

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00016	0,00006	0,106	0,025	0,000	0,2435	0,00011208	0,00006931
2004	0,00020	0,00007	0,106	0,022	0,000	0,2161	0,00003554	0,00007355
2005	0,00020	0,00006	0,109	0,019	0,000	0,1908	0,00003555	0,00006364
2006	0,00015	0,00004	0,121	0,018	0,000	0,1922	0,00007298	0,00004570
2007	0,00016	0,00004	0,117	0,017	0,000	0,1857	0,00004450	0,00004665
2008	0,00017	0,00004	0,113	0,016	0,000	0,1829	0,00002112	0,00004500
2009	0,00013	0,00003	0,124	0,014	0,000	0,1619	0,00006107	0,00003328

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00013	0,00006	0,167	0,036	0,533	0,2704	0,00060459	0,00007656
2004	0,00041	0,00017	0,096	0,028	0,000	0,1899	0,00024775	0,00018337
2005	0,00038	0,00022	0,097	0,040	0,000	0,2613	0,00028884	0,00023764
2006	0,00033	0,00016	0,104	0,034	0,012	0,2346	0,00028824	0,00017456
2007	0,00029	0,00013	0,112	0,034	0,034	0,2451	0,00029180	0,00014412
2008	0,00012	0,00005	0,177	0,036	0,638	0,2995	0,00047152	0,00006838
2009	0,00023	0,00009	0,133	0,030	0,311	0,2373	0,00030928	0,00010136

Source: WHOMD and HMD 2014, own elaboration

Table A. 8: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for the remaining causes of death of mortality in Spain

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00128	0,00006	0,165	0,004	0,290	0,0418	0,00000001	0,00052326
2004	0,00116	0,00005	0,168	0,004	0,356	0,0435	0,00000003	0,00029912
2005	0,00114	0,00005	0,171	0,003	0,374	0,0408	0,00000000	0,00083406
2006	0,00109	0,00005	0,168	0,003	0,328	0,0407	0,00000000	0,00639336
2007	0,00111	0,00004	0,167	0,003	0,293	0,0376	0,00000000	0,00415979
2008	0,00110	0,00004	0,167	0,003	0,283	0,0372	0,00000426	0,00005695
2009	0,00114	0,00004	0,162	0,003	0,220	0,0340	0,00000001	0,00040089

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2003	0,00159	0,00014	0,154	0,008	0,160	0,0590	0,00030170	0,00016602
2004	0,00176	0,00017	0,143	0,008	0,112	0,0614	0,00000015	0,00034951
2005	0,00184	0,00018	0,144	0,008	0,144	0,0617	0,00000016	0,00035655
2006	0,00148	0,00014	0,156	0,008	0,234	0,0650	0,00020280	0,00016449
2007	0,00135	0,00012	0,169	0,008	0,361	0,0656	0,00036668	0,00014626
2008	0,00157	0,00014	0,154	0,008	0,228	0,0627	0,00005087	0,00015896
2009	0,00127	0,00011	0,172	0,007	0,389	0,0628	0,00048178	0,00012922

Source: WHOMD and HMD 2014, own elaboration

Table A. 9: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for overall mortality in Finland

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,01018	0,00103	0,139	0,008	0,192	0,0574	0,00118356	0,00118127
1997	0,01108	0,00117	0,130	0,008	0,121	0,0577	0,00013773	0,00131917
1998	0,01070	0,00110	0,129	0,007	0,109	0,0565	0,00000001	0,00867534
1999	0,00964	0,00104	0,140	0,008	0,226	0,0637	0,00012555	0,00119953
2000	0,00926	0,00092	0,139	0,007	0,180	0,0586	0,00053254	0,00106616
2001	0,00883	0,00090	0,139	0,007	0,176	0,0610	0,00069288	0,00104458
2002	0,00759	0,00075	0,150	0,007	0,228	0,0610	0,00169225	0,00090789
2003	0,00787	0,00078	0,145	0,007	0,205	0,0611	0,00116529	0,00091978
2004	0,00574	0,00059	0,159	0,008	0,306	0,0674	0,00319523	0,00073870
2005	0,00603	0,00064	0,152	0,008	0,230	0,0691	0,00248648	0,00078531
2006	0,00623	0,00065	0,147	0,008	0,186	0,0692	0,00200576	0,00077729
2007	0,00540	0,00053	0,155	0,007	0,218	0,0691	0,00294702	0,00065973
2008	0,00618	0,00060	0,145	0,007	0,148	0,0673	0,00190328	0,00072299
2009	0,00587	0,00053	0,146	0,007	0,162	0,0647	0,00219680	0,00065218
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,02804	0,00470	0,095	0,012	0,024	0,0656	0,00003450	0,00489150
1997	0,01860	0,00294	0,129	0,013	0,229	0,0755	0,00796982	0,00319241
1998	0,01991	0,00335	0,122	0,014	0,183	0,0786	0,00662657	0,00359371
1999	0,02253	0,00389	0,108	0,013	0,083	0,0737	0,00278095	0,00412313
2000	0,01839	0,00279	0,122	0,012	0,144	0,0694	0,00520186	0,00303321
2001	0,02196	0,00354	0,109	0,012	0,085	0,0690	0,00000117	0,00375497
2002	0,02217	0,00348	0,105	0,012	0,051	0,0657	0,00000135	0,00368799
2003	0,02070	0,00338	0,106	0,012	0,035	0,0676	0,00004048	0,00358712
2004	0,01744	0,00314	0,111	0,013	0,051	0,0760	0,00346777	0,00335876
2005	0,01784	0,00312	0,105	0,013	0,001	0,0730	0,00207163	0,00331083
2006	0,01847	0,00338	0,104	0,013	0,008	0,0784	0,00087293	0,00356306
2007	0,01224	0,00180	0,133	0,012	0,170	0,0741	0,00748102	0,00200498
2008	0,01584	0,00311	0,107	0,014	0,000	0,0868	0,00263763	0,00330948
2009	0,00919	0,00137	0,149	0,012	0,251	0,0815	0,01044860	0,00160300

Source: WHOMD and HMD 2014, own elaboration

Table A. 10: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for neoplasms mortality in Finland

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00373	0,00244	0,075	0,035	0,183	0,2251	0,00066248	0,00254026
1997	0,00467	0,00191	0,067	0,020	0,206	0,1374	0,00000001	0,01442005
1998	0,00429	0,00159	0,074	0,019	0,251	0,1384	0,00000056	0,00166289
1999	0,00406	0,00145	0,082	0,020	0,406	0,1587	0,00001814	0,00152318
2000	0,00415	0,00162	0,079	0,021	0,354	0,1601	0,00000034	0,00206517
2001	0,00389	0,00213	0,081	0,030	0,396	0,2384	0,00022399	0,00222672
2002	0,00399	0,00215	0,074	0,028	0,285	0,2071	0,00000025	0,00318920
2003	0,00411	0,00213	0,072	0,026	0,226	0,1912	0,00000278	0,00221289
2004	0,00140	0,00059	0,137	0,029	0,766	0,2614	0,00278243	0,00070222
2005	0,00376	0,00107	0,079	0,015	0,337	0,1286	0,00000007	0,00296060
2006	0,00262	0,00128	0,092	0,028	0,419	0,2425	0,00141252	0,00136745
2007	0,00380	0,00086	0,073	0,012	0,289	0,1139	0,00003110	0,00090672
2008	0,00400	0,00141	0,071	0,018	0,281	0,1515	0,00000008	0,00367305
2009	0,00398	0,00192	0,065	0,023	0,196	0,1844	0,00000083	0,00198724
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00820	0,00320	0,092	0,026	0,306	0,1530	0,00000014	0,00621545
1997	0,00770	0,00272	0,097	0,024	0,395	0,1501	0,00000024	0,00409855
1998	0,00805	0,00323	0,082	0,025	0,209	0,1401	0,00000023	0,00493389
1999	0,00790	0,00268	0,082	0,021	0,235	0,1213	0,00000019	0,00454051
2000	0,00736	0,00274	0,088	0,024	0,269	0,1396	0,00000029	0,00373888
2001	0,00668	0,00245	0,107	0,027	0,423	0,1669	0,00000521	0,00256882
2002	0,00698	0,00146	0,100	0,015	0,438	0,1041	0,00000006	0,00434283
2003	0,00689	0,00209	0,088	0,019	0,257	0,1159	0,00000011	0,00472403
2004	0,00657	0,00220	0,098	0,023	0,370	0,1427	0,00000009	0,00549566
2005	0,00621	0,00170	0,092	0,018	0,277	0,1128	0,00000003	0,00712662
2006	0,00631	0,00198	0,102	0,022	0,431	0,1477	0,00000011	0,00445835
2007	0,00619	0,00200	0,095	0,021	0,357	0,1421	0,00000591	0,00209127
2008	0,00607	0,00257	0,093	0,027	0,367	0,1829	0,00000052	0,00266184
2009	0,00625	0,00261	0,086	0,025	0,279	0,1628	0,00004575	0,00271180

Source: WHOMD and HMD 2014, own elaboration

Table A. 11: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for ischaemic heart diseases mortality in Finland

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00272	0,00036	0,155	0,010	0,472	0,0853	0,00000007	0,00118714
1997	0,00242	0,00035	0,159	0,011	0,455	0,0939	0,00000003	0,00176893
1998	0,00246	0,00035	0,154	0,011	0,394	0,0900	0,00000007	0,00115988
1999	0,00202	0,00029	0,172	0,011	0,509	0,0960	0,00000000	0,01626090
2000	0,00181	0,00027	0,175	0,012	0,484	0,1000	0,00014978	0,00033361
2001	0,00174	0,00023	0,176	0,011	0,502	0,0938	0,00000001	0,00193346
2002	0,00152	0,00021	0,186	0,011	0,553	0,0961	0,00000006	0,00080278
2003	0,00139	0,00019	0,189	0,011	0,588	0,0964	0,00000011	0,00053287
2004	0,00107	0,00017	0,195	0,013	0,588	0,1157	0,00021883	0,00022606
2005	0,00099	0,00016	0,199	0,013	0,599	0,1182	0,00030345	0,00021821
2006	0,00113	0,00017	0,184	0,012	0,465	0,1137	0,00000003	0,00092306
2007	0,00112	0,00015	0,180	0,011	0,399	0,1069	0,00000000	0,00325391
2008	0,00105	0,00015	0,180	0,011	0,346	0,1154	0,00000004	0,00065480
2009	0,00093	0,00013	0,182	0,011	0,362	0,1164	0,00005260	0,00017205
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00953	0,00266	0,092	0,020	0,114	0,1076	0,00004212	0,00276594
1997	0,00516	0,00140	0,143	0,023	0,393	0,1413	0,00345327	0,00154462
1998	0,00640	0,00201	0,122	0,025	0,257	0,1486	0,00206662	0,00215333
1999	0,00595	0,00167	0,126	0,023	0,233	0,1345	0,00200717	0,00180662
2000	0,00488	0,00124	0,137	0,021	0,276	0,1262	0,00249869	0,00137429
2001	0,00669	0,00319	0,107	0,035	0,081	0,1924	0,00000016	0,00595897
2002	0,00640	0,00209	0,106	0,024	0,031	0,1345	0,00001887	0,00222556
2003	0,00572	0,00137	0,115	0,018	0,131	0,1086	0,00000149	0,00147056
2004	0,00554	0,00174	0,110	0,023	0,071	0,1330	0,00010031	0,00185403
2005	0,00314	0,00080	0,144	0,021	0,171	0,1296	0,00223340	0,00091805
2006	0,00368	0,00100	0,134	0,022	0,137	0,1366	0,00143872	0,00110811
2007	0,00206	0,00048	0,180	0,020	0,422	0,1359	0,00304555	0,00060407
2008	0,00413	0,00122	0,115	0,022	0,000	0,1390	0,00055894	0,00131763
2009	0,00236	0,00062	0,154	0,021	0,212	0,1458	0,00267023	0,00072970

Source: WHOMD and HMD 2014, own elaboration

Table A. 12: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for cerebrovascular diseases mortality in Finland

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00123	0,00019	0,167	0,013	0,520	0,1095	0,00000001	0,00148485
1997	0,00117	0,00020	0,163	0,014	0,436	0,1151	0,00000002	0,00132397
1998	0,00096	0,00020	0,174	0,017	0,579	0,1446	0,00000659	0,00024860
1999	0,00072	0,00015	0,198	0,017	0,798	0,1482	0,00022061	0,00019889
2000	0,00089	0,00018	0,180	0,016	0,610	0,1412	0,00000009	0,00052332
2001	0,00086	0,00016	0,171	0,015	0,467	0,1331	0,00000000	0,00257366
2002	0,00059	0,00014	0,198	0,018	0,689	0,1611	0,00033210	0,00019178
2003	0,00080	0,00017	0,178	0,017	0,610	0,1518	0,00000001	0,00165131
2004	0,00072	0,00017	0,175	0,019	0,530	0,1728	0,00002377	0,00021501
2005	0,00067	0,00017	0,169	0,019	0,411	0,1755	0,00000078	0,00020428
2006	0,00044	0,00010	0,206	0,018	0,789	0,1738	0,00014845	0,00013341
2007	0,00057	0,00015	0,169	0,019	0,387	0,1916	0,00006471	0,00017821
2008	0,00051	0,00011	0,187	0,017	0,703	0,1742	0,00000000	0,00164867
2009	0,00056	0,00009	0,175	0,012	0,547	0,1362	0,00000945	0,00012469
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00177	0,00073	0,148	0,037	0,372	0,2242	0,00036467	0,00079786
1997	0,00109	0,00038	0,203	0,034	0,766	0,2202	0,00105516	0,00045883
1998	0,00137	0,00046	0,176	0,032	0,552	0,2048	0,00068502	0,00052925
1999	0,00127	0,00043	0,181	0,032	0,594	0,2074	0,00056659	0,00050493
2000	0,00141	0,00054	0,152	0,033	0,307	0,2033	0,00033151	0,00060953
2001	0,00155	0,00046	0,144	0,025	0,271	0,1592	0,00000000	0,00600225
2002	0,00145	0,00044	0,142	0,026	0,276	0,1649	0,00000002	0,00259190
2003	0,00148	0,00049	0,143	0,028	0,278	0,1756	0,00000005	0,00166704
2004	0,00118	0,00044	0,152	0,032	0,309	0,2017	0,00018311	0,00049992
2005	0,00107	0,00036	0,166	0,030	0,490	0,1985	0,00030977	0,00041652
2006	0,00117	0,00041	0,157	0,030	0,443	0,2058	0,00000009	0,00105103
2007	0,00107	0,00044	0,144	0,034	0,202	0,2248	0,00016877	0,00049815
2008	0,00119	0,00044	0,144	0,034	0,202	0,2248	0,00016877	0,00049815
2009	0,00099	0,00041	0,141	0,033	0,191	0,2262	0,00021356	0,00045969

Source: WHOMD and HMD 2014, own elaboration

Table A. 13: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for remaining circulatory system diseases mortality in Finland

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00046	0,00013	0,180	0,022	0,204	0,1758	0,00031240	0,00017194
1997	0,00059	0,00018	0,160	0,023	0,124	0,1812	0,00010449	0,00022043
1998	0,00062	0,00019	0,153	0,023	0,094	0,1831	0,00006415	0,00023887
1999	0,00034	0,00011	0,193	0,025	0,377	0,2007	0,00030098	0,00015390
2000	0,00063	0,00019	0,144	0,023	0,034	0,1846	0,00000001	0,00166816
2001	0,00048	0,00017	0,157	0,026	0,051	0,2122	0,00019938	0,00020802
2002	0,00060	0,00018	0,144	0,022	0,004	0,1790	0,00000000	0,00359209
2003	0,00043	0,00013	0,170	0,024	0,247	0,2034	0,00008786	0,00016616
2004	0,00046	0,00015	0,162	0,026	0,240	0,2234	0,00005950	0,00018774
2005	0,00025	0,00008	0,203	0,025	0,519	0,2295	0,00036384	0,00012484
2006	0,00043	0,00013	0,160	0,023	0,103	0,2145	0,00008812	0,00016685
2007	0,00035	0,00009	0,186	0,021	0,385	0,2087	0,00015870	0,00012773
2008	0,00036	0,00009	0,181	0,020	0,315	0,1984	0,00013072	0,00012786
2009	0,00041	0,00010	0,175	0,018	0,335	0,1885	0,00012634	0,00012745
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00056	0,00030	0,191	0,050	0,427	0,2984	0,00141121	0,00038650
1997	0,00081	0,00042	0,148	0,045	0,073	0,2561	0,00101103	0,00048610
1998	0,00146	0,00106	0,106	0,056	0,008	0,3041	0,00040917	0,00112790
1999	0,00128	0,00089	0,112	0,054	0,000	0,2967	0,00039790	0,00095698
2000	0,00114	0,00069	0,116	0,046	0,000	0,2515	0,00039153	0,00075366
2001	0,00112	0,00070	0,117	0,049	0,000	0,4423	0,00027566	0,00075225
2002	0,00100	0,00064	0,119	0,050	0,000	0,2803	0,00057726	0,00070888
2003	0,00121	0,00059	0,114	0,038	0,017	0,2150	0,00026409	0,00064776
2004	0,00060	0,00048	0,143	0,065	0,037	0,3748	0,00095816	0,00056060
2005	0,00059	0,00041	0,136	0,054	0,000	0,3086	0,00094451	0,00049451
2006	0,00059	0,00033	0,147	0,046	0,016	0,2860	0,00082908	0,00038607
2007	0,00090	0,00053	0,124	0,045	0,001	0,2741	0,00066419	0,00059320
2008	0,00077	0,00054	0,132	0,053	0,000	0,3243	0,00088791	0,00062246
2009	0,00038	0,00018	0,199	0,041	0,461	0,2820	0,00123691	0,00025868

Source: WHOMD and HMD 2014, own elaboration

Table A. 14: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for respiratory system diseases mortality in Finland

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00049	0,00011	0,180	0,019	0,252	0,1522	0,00000002	0,00073923
1997	0,00048	0,00015	0,162	0,024	0,056	0,1848	0,00015753	0,00018894
1998	0,00055	0,00016	0,155	0,021	0,000	0,1684	0,00000000	0,01152571
1999	0,00056	0,00015	0,165	0,021	0,220	0,1708	0,00000001	0,00148511
2000	0,00051	0,00013	0,164	0,020	0,122	0,1653	0,00000001	0,00159184
2001	0,00040	0,00012	0,177	0,024	0,320	0,2052	0,00014396	0,00016232
2002	0,00047	0,00013	0,155	0,020	0,025	0,1720	0,00000000	0,00205414
2003	0,00040	0,00011	0,172	0,021	0,280	0,1882	0,00000000	0,00248831
2004	0,00034	0,00015	0,151	0,033	0,000	0,2803	0,00006752	0,00019158
2005	0,00020	0,00011	0,156	0,041	0,000	0,3471	0,00024151	0,00014625
2006	0,00026	0,00013	0,131	0,034	0,000	0,2993	0,00004332	0,00015650
2007	0,00014	0,00007	0,172	0,037	0,274	0,3477	0,00017874	0,00010381
2008	0,00011	0,00006	0,192	0,042	0,760	0,4226	0,00032006	0,00009574
2009	0,00037	0,00020	0,113	0,034	0,099	0,3078	0,00002354	0,00023082
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00192	0,00050	0,152	0,024	0,202	0,1452	0,00000000	0,00629348
1997	0,00193	0,00055	0,140	0,025	0,120	0,1492	0,00000001	0,00464478
1998	0,00199	0,00057	0,140	0,025	0,132	0,1500	0,00000001	0,00387180
1999	0,00168	0,00061	0,149	0,032	0,149	0,1914	0,00019978	0,00068143
2000	0,00165	0,00039	0,161	0,021	0,245	0,1328	0,00000000	0,00909384
2001	0,00169	0,00044	0,144	0,023	0,179	0,1410	0,00000003	0,00208758
2002	0,00144	0,00034	0,159	0,021	0,261	0,1342	0,00000001	0,00327586
2003	0,00141	0,00041	0,143	0,024	0,102	0,1508	0,00000003	0,00202416
2004	0,00089	0,00036	0,153	0,034	0,094	0,2058	0,00042679	0,00042096
2005	0,00112	0,00040	0,131	0,029	0,094	0,1810	0,00002289	0,00044022
2006	0,00099	0,00044	0,133	0,036	0,121	0,2323	0,00000002	0,00245833
2007	0,00118	0,00054	0,113	0,035	0,045	0,2154	0,00000004	0,00217271
2008	0,00059	0,00026	0,161	0,038	0,357	0,2635	0,00025165	0,00030480
2009	0,00053	0,00024	0,176	0,038	0,492	0,2748	0,00043125	0,00028722

Source: WHOMD and HMD 2014, own elaboration

Table A. 15: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for digestive system diseases of mortality in Finland

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00016	0,00006	0,212	0,033	0,756	0,2748	0,00021330	0,00009313
1997	0,00028	0,00012	0,162	0,035	0,340	0,2783	0,00005435	0,00014961
1998	0,00023	0,00011	0,174	0,037	0,511	0,3025	0,00014345	0,00014172
1999	0,00040	0,00021	0,123	0,036	0,043	0,2778	0,00000005	0,00074489
2000	0,00037	0,00014	0,137	0,027	0,264	0,2284	0,00000000	0,00365064
2001	0,00020	0,00011	0,169	0,042	0,457	0,3564	0,00023676	0,00014503
2002	0,00021	0,00012	0,164	0,044	0,422	0,3725	0,00017983	0,00015479
2003	0,00005	0,00003	0,259	0,045	1,151	0,3983	0,00039444	0,00006951
2004	0,00036	0,00021	0,129	0,040	0,233	0,3377	0,00000062	0,00023735
2005	0,00024	0,00018	0,138	0,053	0,239	0,4576	0,00026754	0,00020799
2006	0,00030	0,00016	0,130	0,038	0,190	0,3355	0,00011189	0,00019022
2007	0,00020	0,00014	0,137	0,050	0,000	0,4523	0,00029188	0,00016838
2008	0,00020	0,00010	0,160	0,039	0,568	0,3856	0,00013955	0,00013220
2009	0,00018	0,00010	0,158	0,041	0,483	0,4145	0,00023590	0,00013170
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00012	0,00009	0,248	0,068	0,725	0,4235	0,00072499	0,00014092
1997	0,00011	0,00009	0,258	0,080	0,864	0,4924	0,00078404	0,00015884
1998	0,00021	0,00020	0,186	0,089	0,503	0,5476	0,00070778	0,00025900
1999	0,00021	0,00027	0,163	0,111	0,171	0,6285	0,00070273	0,00033481
2000	0,00011	0,00012	0,234	0,097	0,696	0,5811	0,00085647	0,00019347
2001	0,00030	0,00033	0,137	0,090	0,076	0,5137	0,00050376	0,00037592
2002	0,00013	0,00019	0,169	0,123	0,127	0,6967	0,00094494	0,00026337
2003	0,00005	0,00006	0,279	0,099	0,874	0,5981	0,00081382	0,00012905
2004	0,00016	0,00023	0,155	0,117	0,102	0,6724	0,00086266	0,00029357
2005	0,00006	0,00007	0,227	0,092	0,441	0,5640	0,00107593	0,00013233
2006	0,00006	0,00008	0,243	0,125	0,671	0,7937	0,00095740	0,00014827
2007	0,00015	0,00015	0,174	0,086	0,362	0,5621	0,00090853	0,00020523
2008	0,00004	0,00008	0,206	0,153	0,275	0,9987	0,00102952	0,00013146
2009	0,00004	0,00006	0,224	0,125	0,412	0,8350	0,00126865	0,00012819

Source: WHOMD and HMD 2014, own elaboration

Table A. 16: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for external causes of mortality in Finland

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00016	0,00012	0,160	0,056	0,094	0,4154	0,00035218	0,00015604
1997	0,00025	0,00015	0,143	0,044	0,145	0,3388	0,00020717	0,00018143
1998	0,00023	0,00016	0,141	0,051	0,000	0,3841	0,00034347	0,00019139
1999	0,00006	0,00003	0,255	0,045	1,045	0,3842	0,00033046	0,00006916
2000	0,00021	0,00014	0,137	0,050	0,000	0,3897	0,00025230	0,00017046
2001	0,00014	0,00008	0,183	0,046	0,582	0,3907	0,00032102	0,00012262
2002	0,00009	0,00006	0,212	0,048	0,677	0,4088	0,00038285	0,00009678
2003	0,00028	0,00019	0,127	0,049	0,000	0,3851	0,00015879	0,00022675
2004	0,00019	0,00014	0,139	0,053	0,000	0,4400	0,00030963	0,00017006
2005	0,00017	0,00014	0,141	0,059	0,000	0,4981	0,00027595	0,00017073
2006	0,00013	0,00010	0,159	0,055	0,107	0,4963	0,00036752	0,00012864
2007	0,00003	0,00004	0,253	0,087	1,024	0,8243	0,00047862	0,00008283
2008	0,00016	0,00011	0,145	0,051	0,084	0,4881	0,00028629	0,00013633
2009	0,00004	0,00002	0,258	0,045	1,314	0,4764	0,00044239	0,00005778
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00000	0,00000	0,532	0,066	2,098	0,4136	0,00178794	0,00009312
1997	0,00024	0,00030	0,150	0,107	0,000	0,5881	0,00154010	0,00037615
1998	0,00014	0,00012	0,255	0,081	0,783	0,4907	0,00155233	0,00020469
1999	0,00009	0,00012	0,237	0,110	0,537	0,6395	0,00153778	0,00020420
2000	0,00014	0,00016	0,199	0,097	0,286	0,5567	0,00147474	0,00024044
2001	0,00038	0,00051	0,126	0,105	0,000	0,5630	0,00119191	0,00059529
2002	0,00016	0,00021	0,179	0,111	0,186	0,6345	0,00159978	0,00029998
2003	0,00004	0,00004	0,317	0,089	1,019	0,5356	0,00159490	0,00013373
2004	0,00020	0,00029	0,150	0,114	0,000	0,6320	0,00163116	0,00037332
2005	0,00011	0,00010	0,230	0,081	0,546	0,4942	0,00158741	0,00018691
2006	0,00000	0,00000	0,533	0,049	2,414	0,3713	0,00191723	0,00008675
2007	0,00011	0,00013	0,187	0,095	0,161	0,5934	0,00162058	0,00019724
2008	0,00014	0,00016	0,186	0,092	0,215	0,5926	0,00144383	0,00022459
2009	0,00007	0,00006	0,273	0,074	0,933	0,5103	0,00144386	0,00013532

Source: WHOMD and HMD 2014, own elaboration

Table A. 17: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for the remaining causes of death of mortality in Finland

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00101	0,00021	0,172	0,016	0,227	0,1305	0,00024723	0,00026261
1997	0,00100	0,00019	0,178	0,015	0,301	0,1226	0,00023950	0,00024476
1998	0,00110	0,00021	0,165	0,015	0,168	0,1210	0,00000005	0,00080013
1999	0,00116	0,00022	0,165	0,015	0,230	0,1218	0,00000001	0,00173407
2000	0,00103	0,00020	0,170	0,015	0,219	0,1260	0,00004948	0,00024649
2001	0,00102	0,00017	0,169	0,013	0,202	0,1126	0,00000000	0,00675175
2002	0,00113	0,00021	0,168	0,015	0,204	0,1238	0,00000007	0,00068899
2003	0,00122	0,00022	0,156	0,014	0,061	0,1186	0,00000004	0,00093358
2004	0,00100	0,00018	0,168	0,014	0,185	0,1248	0,00000001	0,00133997
2005	0,00076	0,00014	0,189	0,015	0,389	0,1363	0,00025667	0,00019192
2006	0,00101	0,00016	0,168	0,012	0,185	0,1138	0,00000002	0,00097220
2007	0,00096	0,00016	0,172	0,013	0,202	0,1274	0,00000004	0,00070655
2008	0,00105	0,00016	0,164	0,012	0,111	0,1166	0,00000001	0,00133118
2009	0,00099	0,00014	0,172	0,010	0,205	0,1099	0,00000004	0,00063820
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00142	0,00059	0,145	0,037	0,075	0,2168	0,00046384	0,00065842
1997	0,00099	0,00033	0,192	0,031	0,437	0,1929	0,00084215	0,00040201
1998	0,00135	0,00049	0,164	0,034	0,228	0,2050	0,00028934	0,00054489
1999	0,00166	0,00060	0,145	0,032	0,125	0,1876	0,00016870	0,00067162
2000	0,00081	0,00026	0,208	0,030	0,496	0,1890	0,00081657	0,00033987
2001	0,00058	0,00018	0,239	0,029	0,674	0,1826	0,00135505	0,00027172
2002	0,00110	0,00032	0,187	0,027	0,364	0,1703	0,00079491	0,00039697
2003	0,00114	0,00033	0,178	0,026	0,295	0,1643	0,00045913	0,00040072
2004	0,00115	0,00034	0,170	0,026	0,232	0,1647	0,00054826	0,00040695
2005	0,00130	0,00036	0,167	0,024	0,240	0,1552	0,00055468	0,00042687
2006	0,00170	0,00051	0,142	0,025	0,049	0,1599	0,00000008	0,00143173
2007	0,00119	0,00031	0,180	0,023	0,340	0,1559	0,00053069	0,00037479
2008	0,00148	0,00040	0,160	0,023	0,196	0,1577	0,00008606	0,00046481
2009	0,00087	0,00021	0,209	0,022	0,534	0,1566	0,00080556	0,00028496

Source: WHOMD and HMD 2014, own elaboration

Table A. 18: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for overall mortality in France

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00747	0,00018	0,128	0,002	0,003	0,0195	0,00070134	0,00021242
2001	0,00707	0,00016	0,129	0,002	0,000	0,0192	0,00096048	0,00019648
2002	0,00708	0,00016	0,129	0,002	0,000	0,0191	0,00078990	0,00019278
2003	0,00691	0,00014	0,132	0,001	0,000	0,0180	0,00093440	0,00017599
2004	0,00625	0,00014	0,130	0,002	0,000	0,0188	0,00102167	0,00017026
2005	0,00589	0,00011	0,134	0,001	0,000	0,0168	0,00140542	0,00014819
2006	0,00550	0,00010	0,134	0,001	0,000	0,0164	0,00164684	0,00013747
2007	0,00508	0,00009	0,137	0,001	0,000	0,0165	0,00210556	0,00013044
2008	0,00518	0,00008	0,136	0,001	0,000	0,0161	0,00186926	0,00012375
2009	0,00518	0,00008	0,136	0,001	0,000	0,0159	0,00164954	0,00011976

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,01199	0,00053	0,131	0,003	0,148	0,0232	0,00963124	0,00060303
2001	0,01207	0,00055	0,130	0,004	0,136	0,0237	0,00866848	0,00061879
2002	0,01235	0,00059	0,126	0,004	0,101	0,0248	0,00782392	0,00066665
2003	0,01241	0,00059	0,128	0,004	0,111	0,0255	0,00718608	0,00066656
2004	0,01287	0,00066	0,120	0,004	0,087	0,0265	0,00514006	0,00072273
2005	0,01385	0,00068	0,112	0,004	0,000	0,0247	0,00417267	0,00074675
2006	0,01283	0,00063	0,113	0,004	0,000	0,0252	0,00435097	0,00068920
2007	0,01214	0,00055	0,115	0,003	0,000	0,0242	0,00462099	0,00060548
2008	0,01211	0,00055	0,116	0,003	0,006	0,0247	0,00408154	0,00060484
2009	0,00902	0,00037	0,136	0,003	0,162	0,0253	0,00698778	0,00043031

Source: WHOMD and HMD 2014, own elaboration

Table A. 19: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for neoplasms mortality in France

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00401	0,00043	0,069	0,006	0,175	0,0529	0,00000002	0,00200979
2001	0,00409	0,00040	0,066	0,005	0,113	0,0461	0,00000002	0,00208661
2002	0,00397	0,00037	0,070	0,005	0,200	0,0485	0,00000044	0,00041404
2003	0,00396	0,00035	0,068	0,004	0,136	0,0444	0,00000178	0,00036240
2004	0,00385	0,00033	0,069	0,004	0,162	0,0448	0,00000006	0,00098884
2005	0,00385	0,00029	0,068	0,004	0,146	0,0408	0,00004543	0,00031172
2006	0,00386	0,00026	0,065	0,003	0,087	0,0367	0,00002290	0,00027857
2007	0,00379	0,00026	0,067	0,003	0,150	0,0405	0,00000001	0,00167520
2008	0,00393	0,00027	0,064	0,003	0,109	0,0397	0,00000005	0,00087090
2009	0,00381	0,00023	0,065	0,003	0,085	0,0393	0,00000276	0,00025048

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00995	0,00792	0,063	0,041	0,083	0,2152	0,00001519	0,00806628
2001	0,00954	0,00202	0,067	0,011	0,125	0,0636	0,00017309	0,00206348
2002	0,00949	0,00181	0,064	0,010	0,078	0,0537	0,00022940	0,00184822
2003	0,00917	0,00133	0,069	0,008	0,128	0,0463	0,00000031	0,00174819
2004	0,00879	0,00569	0,072	0,036	0,160	0,2084	0,00000396	0,00584371
2005	0,00883	0,00103	0,069	0,006	0,123	0,0385	0,00000000	0,01106944
2006	0,00859	0,00111	0,069	0,007	0,127	0,0435	0,00002921	0,00114801
2007	0,00840	0,00114	0,069	0,007	0,111	0,0475	0,00003606	0,00117500
2008	0,00807	0,00108	0,073	0,007	0,174	0,0511	0,00000015	0,00206570
2009	0,00768	0,00106	0,075	0,008	0,177	0,0544	0,00031323	0,00109135

Source: WHOMD and HMD 2014, own elaboration

Table A. 20: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for ischaemic heart diseases mortality in France

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00053	0,00003	0,156	0,005	0,482	0,0580	0,00000000	0,00116172
2001	0,00051	0,00003	0,153	0,004	0,403	0,0561	0,00000000	0,00050981
2002	0,00048	0,00003	0,151	0,004	0,308	0,0550	0,00000000	0,00150121
2003	0,00046	0,00002	0,148	0,004	0,194	0,0545	0,00000000	0,00062926
2004	0,00041	0,00002	0,149	0,004	0,240	0,0564	0,00000000	0,00067828
2005	0,00041	0,00002	0,141	0,004	0,025	0,0511	0,00000000	0,00059266
2006	0,00038	0,00002	0,140	0,003	0,000	0,0514	0,00000000	0,00055835
2007	0,00035	0,00002	0,142	0,003	0,000	0,0531	0,00000000	0,00062167
2008	0,00034	0,00002	0,142	0,003	0,000	0,0551	0,00000000	0,00061457
2009	0,00030	0,00001	0,147	0,003	0,069	0,0559	0,00000000	0,00043880

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00166	0,00021	0,129	0,010	0,304	0,0685	0,00038148	0,00023158
2001	0,00153	0,00021	0,132	0,011	0,318	0,0741	0,00032669	0,00023132
2002	0,00143	0,00018	0,133	0,010	0,313	0,0695	0,00033574	0,00020061
2003	0,00163	0,00024	0,119	0,011	0,199	0,0775	0,00003769	0,00026078
2004	0,00126	0,00016	0,135	0,010	0,307	0,0744	0,00029444	0,00018104
2005	0,00144	0,00021	0,118	0,011	0,161	0,0762	0,00003605	0,00022348
2006	0,00103	0,00013	0,137	0,010	0,264	0,0743	0,00032515	0,00014361
2007	0,00094	0,00012	0,138	0,010	0,252	0,0789	0,00037635	0,00013731
2008	0,00097	0,00012	0,134	0,010	0,229	0,0779	0,00023323	0,00013826
2009	0,00093	0,00013	0,131	0,011	0,193	0,0908	0,00018693	0,00014998

Source: WHOMD and HMD 2014, own elaboration

Table A. 21: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for cerebrovascular diseases mortality in France

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00052	0,00003	0,167	0,004	0,566	0,0526	0,00000000	0,00076115
2001	0,00050	0,00002	0,164	0,004	0,515	0,0517	0,00000000	0,00158793
2002	0,00049	0,00002	0,157	0,004	0,395	0,0520	0,00000000	0,00056283
2003	0,00049	0,00002	0,158	0,004	0,422	0,0511	0,00000000	0,00244776
2004	0,00046	0,00002	0,147	0,003	0,192	0,0485	0,00000000	0,00059179
2005	0,00045	0,00002	0,147	0,003	0,202	0,0482	0,00000000	0,00064959
2006	0,00044	0,00002	0,139	0,003	0,062	0,0477	0,00000000	0,00033267
2007	0,00042	0,00002	0,139	0,003	0,038	0,0496	0,00000000	0,00053441
2008	0,00042	0,00002	0,138	0,003	0,003	0,0501	0,00000000	0,00180081
2009	0,00040	0,00002	0,142	0,003	0,113	0,0514	0,00000000	0,00040110

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00092	0,00011	0,153	0,010	0,361	0,0713	0,00003176	0,00012211
2001	0,00071	0,00008	0,170	0,010	0,470	0,0712	0,00023335	0,00009472
2002	0,00087	0,00009	0,148	0,009	0,329	0,0654	0,00000003	0,00046366
2003	0,00068	0,00008	0,173	0,010	0,541	0,0755	0,00017193	0,00009298
2004	0,00068	0,00009	0,157	0,011	0,404	0,0816	0,00008070	0,00010073
2005	0,00058	0,00007	0,167	0,011	0,450	0,0815	0,00016842	0,00008602
2006	0,00066	0,00008	0,150	0,010	0,335	0,0773	0,00000000	0,00234448
2007	0,00063	0,00008	0,148	0,010	0,300	0,0819	0,00000002	0,00049061
2008	0,00061	0,00007	0,149	0,010	0,342	0,0793	0,00000001	0,00077741
2009	0,00058	0,00007	0,148	0,010	0,305	0,0836	0,00000004	0,00026962

Source: WHOMD and HMD 2014, own elaboration

Table A. 22: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for remaining circulatory system diseases mortality in France

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00081	0,00003	0,166	0,003	0,196	0,0381	0,00000001	0,00031636
2001	0,00085	0,00003	0,157	0,003	0,044	0,0367	0,00000001	0,00038631
2002	0,00080	0,00003	0,159	0,003	0,059	0,0362	0,00000001	0,00029660
2003	0,00078	0,00003	0,161	0,003	0,058	0,0354	0,00000001	0,00029313
2004	0,00072	0,00003	0,158	0,003	0,037	0,0366	0,00000000	0,00076051
2005	0,00069	0,00002	0,159	0,002	0,000	0,0337	0,00000002	0,00019708
2006	0,00064	0,00002	0,161	0,002	0,000	0,0334	0,00002332	0,00003336
2007	0,00061	0,00002	0,162	0,002	0,000	0,0330	0,00000000	0,00115242
2008	0,00060	0,00002	0,163	0,002	0,000	0,0326	0,00000063	0,00002972
2009	0,00058	0,00002	0,163	0,002	0,000	0,0324	0,00000000	0,00069309

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00157	0,00014	0,150	0,007	0,152	0,0509	0,00055500	0,00016117
2001	0,00132	0,00012	0,159	0,008	0,193	0,0528	0,00072400	0,00014169
2002	0,00123	0,00011	0,165	0,007	0,231	0,0528	0,00067536	0,00013074
2003	0,00114	0,00010	0,172	0,008	0,289	0,0552	0,00073426	0,00012358
2004	0,00138	0,00013	0,149	0,008	0,137	0,0572	0,00029340	0,00014913
2005	0,00155	0,00015	0,135	0,008	0,000	0,0546	0,00006366	0,00016343
2006	0,00120	0,00011	0,151	0,007	0,103	0,0555	0,00034825	0,00012516
2007	0,00130	0,00011	0,141	0,007	0,000	0,0545	0,00015376	0,00013278
2008	0,00118	0,00010	0,146	0,007	0,009	0,0547	0,00020645	0,00011730
2009	0,00071	0,00006	0,188	0,007	0,372	0,0567	0,00071539	0,00007549

Source: WHOMD and HMD 2014, own elaboration

Table A. 23: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for respiratory system diseases mortality in France

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00034	0,00002	0,155	0,005	0,000	0,0639	0,00000002	0,00015892
2001	0,00028	0,00002	0,157	0,006	0,025	0,0686	0,00000001	0,00022633
2002	0,00029	0,00002	0,159	0,005	0,052	0,0620	0,00000000	0,00027683
2003	0,00031	0,00002	0,157	0,005	0,006	0,0601	0,00000001	0,00016709
2004	0,00026	0,00002	0,154	0,005	0,000	0,0714	0,00000002	0,00011959
2005	0,00027	0,00002	0,160	0,004	0,000	0,0555	0,00000001	0,00018315
2006	0,00023	0,00001	0,157	0,004	0,000	0,0589	0,00001584	0,00002091
2007	0,00025	0,00001	0,154	0,004	0,000	0,0570	0,00000003	0,00008816
2008	0,00025	0,00001	0,154	0,004	0,000	0,0572	0,00000001	0,00017465
2009	0,00025	0,00001	0,156	0,003	0,000	0,0551	0,00000000	0,00024022

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00084	0,00010	0,150	0,010	0,096	0,0673	0,00028466	0,00011548
2001	0,00094	0,00012	0,134	0,010	0,010	0,0705	0,00003062	0,00013488
2002	0,00089	0,00010	0,142	0,009	0,075	0,0639	0,00000398	0,00011263
2003	0,00092	0,00011	0,140	0,010	0,017	0,0712	0,00000819	0,00012709
2004	0,00075	0,00010	0,147	0,011	0,124	0,0779	0,00002426	0,00011092
2005	0,00087	0,00010	0,137	0,009	0,000	0,0686	0,00000006	0,00033604
2006	0,00076	0,00008	0,136	0,008	0,000	0,0638	0,00000001	0,00063962
2007	0,00076	0,00008	0,137	0,009	0,004	0,0665	0,00000004	0,00032824
2008	0,00073	0,00008	0,140	0,009	0,036	0,0709	0,00000030	0,00012100
2009	0,00072	0,00008	0,140	0,009	0,018	0,0717	0,00000001	0,00072643

Source: WHOMD and HMD 2014, own elaboration

Table A. 24: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for digestive system diseases of mortality in France

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00028	0,00004	0,128	0,009	0,000	0,1042	0,00013912	0,00004403
2001	0,00026	0,00003	0,130	0,008	0,000	0,0984	0,00013270	0,00003797
2002	0,00031	0,00004	0,123	0,008	0,000	0,0996	0,00009801	0,00004409
2003	0,00028	0,00003	0,127	0,008	0,000	0,0988	0,00013658	0,00004049
2004	0,00028	0,00003	0,123	0,008	0,000	0,1004	0,00008346	0,00004075
2005	0,00027	0,00003	0,124	0,007	0,000	0,0866	0,00006564	0,00003606
2006	0,00025	0,00002	0,126	0,006	0,000	0,0842	0,00008405	0,00003217
2007	0,00024	0,00002	0,126	0,006	0,000	0,1060	0,00011576	0,00003181
2008	0,00027	0,00003	0,123	0,006	0,000	0,0862	0,00004408	0,00003280
2009	0,00028	0,00002	0,120	0,005	0,000	0,0832	0,00004417	0,00003176

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00019	0,00005	0,178	0,023	0,380	0,1541	0,00100588	0,00007056
2001	0,00020	0,00006	0,172	0,025	0,352	0,1695	0,00098813	0,00008340
2002	0,00025	0,00008	0,152	0,025	0,206	0,1689	0,00083523	0,00009732
2003	0,00024	0,00007	0,150	0,022	0,139	0,1532	0,00090958	0,00008208
2004	0,00044	0,00015	0,111	0,025	0,000	0,1672	0,00054571	0,00016759
2005	0,00030	0,00009	0,137	0,023	0,138	0,1605	0,00070284	0,00010355
2006	0,00034	0,00011	0,122	0,023	0,000	0,1629	0,00058625	0,00012225
2007	0,00052	0,00017	0,102	0,022	0,000	0,1542	0,00032313	0,00018546
2008	0,00038	0,00010	0,121	0,020	0,066	0,1481	0,00049782	0,00011588
2009	0,00037	0,00010	0,120	0,020	0,056	0,1501	0,00045487	0,00011341

Source: WHOMD and HMD 2014, own elaboration

Table A. 25: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for external causes of mortality in France

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00028	0,00003	0,142	0,007	0,007	0,0866	0,00015735	0,00003692
2001	0,00028	0,00003	0,141	0,007	0,000	0,0847	0,00012469	0,00003508
2002	0,00025	0,00002	0,146	0,007	0,000	0,0826	0,00017037	0,00003113
2003	0,00029	0,00002	0,144	0,006	0,000	0,0768	0,00011745	0,00003187
2004	0,00024	0,00002	0,141	0,007	0,000	0,0856	0,00010934	0,00003016
2005	0,00021	0,00002	0,148	0,006	0,000	0,0769	0,00017174	0,00002550
2006	0,00019	0,00002	0,147	0,006	0,000	0,0760	0,00018698	0,00002465
2007	0,00019	0,00001	0,146	0,005	0,000	0,0748	0,00015922	0,00002350
2008	0,00019	0,00001	0,147	0,005	0,000	0,0740	0,00015295	0,00002238
2009	0,00020	0,00001	0,143	0,005	0,000	0,0728	0,00013540	0,00002273

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00034	0,00006	0,161	0,016	0,187	0,1069	0,00076780	0,00008279
2001	0,00024	0,00005	0,184	0,017	0,323	0,1207	0,00082128	0,00006753
2002	0,00029	0,00006	0,168	0,017	0,209	0,1192	0,00073590	0,00007898
2003	0,00039	0,00008	0,148	0,017	0,082	0,1188	0,00064068	0,00009837
2004	0,00044	0,00010	0,130	0,019	0,000	0,1282	0,00048749	0,00011975
2005	0,00039	0,00008	0,144	0,016	0,104	0,1136	0,00054098	0,00009154
2006	0,00037	0,00008	0,136	0,017	0,000	0,1208	0,00056613	0,00009433
2007	0,00039	0,00008	0,134	0,016	0,000	0,1173	0,00046428	0,00009396
2008	0,00035	0,00007	0,143	0,015	0,079	0,1161	0,00052893	0,00008342
2009	0,00027	0,00005	0,167	0,014	0,274	0,1145	0,00057304	0,00006184

Source: WHOMD and HMD 2014, own elaboration

Table A. 26: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for the remaining causes of death of mortality in France

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00142	0,00006	0,148	0,003	0,035	0,0347	0,00005406	0,00007018
2001	0,00135	0,00005	0,152	0,003	0,063	0,0337	0,00006688	0,00006424
2002	0,00141	0,00005	0,151	0,003	0,050	0,0326	0,00003526	0,00006410
2003	0,00152	0,00005	0,151	0,002	0,047	0,0298	0,00000001	0,00036801
2004	0,00123	0,00004	0,151	0,002	0,041	0,0324	0,00005182	0,00005609
2005	0,00128	0,00004	0,150	0,002	0,000	0,0289	0,00001926	0,00005378
2006	0,00119	0,00003	0,151	0,002	0,000	0,0280	0,00007012	0,00005018
2007	0,00116	0,00003	0,153	0,002	0,000	0,0273	0,00003573	0,00004711
2008	0,00115	0,00003	0,155	0,002	0,000	0,0268	0,00011309	0,00004561
2009	0,00116	0,00003	0,154	0,002	0,000	0,0262	0,00007565	0,00004377

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
2000	0,00119	0,00009	0,187	0,006	0,375	0,0447	0,00189770	0,00011822
2001	0,00161	0,00013	0,163	0,007	0,215	0,0462	0,00130290	0,00015417
2002	0,00158	0,00012	0,164	0,007	0,215	0,0472	0,00132320	0,00015328
2003	0,00143	0,00011	0,179	0,007	0,318	0,0476	0,00152906	0,00013764
2004	0,00147	0,00012	0,166	0,007	0,251	0,0521	0,00111307	0,00015028
2005	0,00152	0,00012	0,162	0,006	0,191	0,0475	0,00116753	0,00014243
2006	0,00172	0,00013	0,147	0,006	0,092	0,0465	0,00075524	0,00015342
2007	0,00154	0,00011	0,155	0,006	0,142	0,0462	0,00095672	0,00013496
2008	0,00170	0,00011	0,151	0,005	0,106	0,0423	0,00074451	0,00013330
2009	0,00124	0,00008	0,177	0,005	0,315	0,0461	0,00121650	0,00010631

Source: WHOMD and HMD 2014, own elaboration

Table A. 27: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for overall mortality in Netherlands

Females								
Year	$\hat{\alpha}(y)$	s.e. $\hat{\alpha}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00944	0,00057	0,134	0,004	0,152	0,0416	0,00332719	0,00067055
1997	0,00969	0,00060	0,131	0,005	0,127	0,0429	0,00241294	0,00069633
1998	0,00904	0,00054	0,135	0,004	0,159	0,0425	0,00302259	0,00063621
1999	0,01025	0,00061	0,127	0,004	0,069	0,0399	0,00183904	0,00070251
2000	0,00975	0,00057	0,131	0,004	0,134	0,0409	0,00205020	0,00066431
2001	0,01061	0,00063	0,124	0,004	0,057	0,0412	0,00091658	0,00072047
2002	0,01135	0,00066	0,119	0,004	0,000	0,0395	0,00001168	0,00074408
2003	0,00939	0,00054	0,130	0,004	0,080	0,0407	0,00182153	0,00062902
2004	0,00977	0,00056	0,124	0,004	0,039	0,0401	0,00082191	0,00064552
2005	0,01000	0,00056	0,121	0,004	0,004	0,0392	0,00000015	0,00117763
2006	0,00925	0,00051	0,125	0,004	0,022	0,0398	0,00052871	0,00058130
2007	0,00862	0,00047	0,125	0,004	0,028	0,0400	0,00102452	0,00053893
2008	0,00843	0,00045	0,126	0,004	0,004	0,0399	0,00103496	0,00051835
2009	0,00746	0,00038	0,133	0,004	0,078	0,0411	0,00165147	0,00045476
Males								
Year	$\hat{\alpha}(y)$	s.e. $\hat{\alpha}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,02577	0,00225	0,110	0,007	0,154	0,0421	0,00008105	0,00237503
1997	0,02295	0,00228	0,115	0,008	0,169	0,0480	0,00203371	0,00241523
1998	0,02194	0,00224	0,115	0,008	0,143	0,0492	0,00305978	0,00237056
1999	0,02417	0,00230	0,107	0,007	0,079	0,0429	0,00000202	0,00242049
2000	0,02308	0,00209	0,112	0,007	0,132	0,0420	0,00002049	0,00221236
2001	0,02248	0,00207	0,109	0,007	0,095	0,0420	0,00003301	0,00219099
2002	0,02151	0,00185	0,114	0,007	0,133	0,0402	0,00025759	0,00196658
2003	0,02042	0,00188	0,116	0,007	0,110	0,0426	0,00021383	0,00200490
2004	0,01906	0,00179	0,115	0,007	0,112	0,0430	0,00072641	0,00191699
2005	0,01874	0,00153	0,116	0,006	0,108	0,0379	0,00000771	0,00163819
2006	0,01769	0,00151	0,116	0,006	0,096	0,0396	0,00000065	0,00162271
2007	0,01572	0,00128	0,122	0,006	0,119	0,0386	0,00140521	0,00138390
2008	0,01483	0,00119	0,123	0,006	0,114	0,0383	0,00142542	0,00129839
2009	0,01397	0,00113	0,126	0,006	0,148	0,0397	0,00217719	0,00123129

Source: WHOMD and HMD 2014, own elaboration

Table A. 28: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for neoplasms mortality in Netherlands

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00432	0,00164	0,071	0,020	0,120	0,1492	0,00132860	0,00169632
1997	0,00542	0,00154	0,062	0,014	0,084	0,1016	0,00000028	0,00213237
1998	0,00470	0,00148	0,068	0,016	0,090	0,1235	0,00064450	0,00152965
1999	0,00555	0,00135	0,059	0,011	0,048	0,0830	0,00000008	0,00348677
2000	0,00540	0,00127	0,060	0,011	0,046	0,0821	0,00001431	0,00130908
2001	0,00532	0,00172	0,064	0,016	0,155	0,1228	0,00006166	0,00176670
2002	0,00547	0,00160	0,062	0,014	0,147	0,1114	0,00000031	0,00210240
2003	0,00545	0,00120	0,062	0,011	0,145	0,0871	0,00000052	0,00124372
2004	0,00532	0,00167	0,066	0,016	0,217	0,1284	0,00000028	0,00232050
2005	0,00515	0,00116	0,067	0,011	0,187	0,0956	0,00002799	0,00120518
2006	0,00510	0,00115	0,071	0,012	0,295	0,1051	0,00013722	0,00119132
2007	0,00505	0,00142	0,071	0,014	0,336	0,1308	0,00000053	0,00146957
2008	0,00536	0,00148	0,064	0,013	0,210	0,1159	0,00000001	0,01499447
2009	0,00520	0,00183	0,065	0,017	0,213	0,1517	0,00000099	0,00188195
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,01050	0,00246	0,090	0,016	0,317	0,0964	0,00000175	0,00253729
1997	0,01033	0,00292	0,085	0,018	0,261	0,1078	0,00000936	0,00299963
1998	0,00848	0,00209	0,097	0,017	0,316	0,1061	0,00167729	0,00216552
1999	0,01008	0,00219	0,088	0,014	0,293	0,0861	0,00000819	0,00225524
2000	0,00982	0,00265	0,086	0,017	0,278	0,1030	0,00004947	0,00272928
2001	0,00961	0,00183	0,090	0,012	0,333	0,0772	0,00000130	0,00189915
2002	0,00939	0,00159	0,090	0,011	0,334	0,0709	0,00006048	0,00164742
2003	0,00897	0,00152	0,095	0,011	0,353	0,0719	0,00000029	0,00208144
2004	0,00893	0,00140	0,092	0,010	0,351	0,0661	0,00000035	0,00175385
2005	0,00862	0,00144	0,093	0,011	0,331	0,0698	0,00000030	0,00194925
2006	0,00835	0,00134	0,094	0,010	0,353	0,0685	0,00000022	0,00213296
2007	0,00825	0,00169	0,096	0,013	0,371	0,0870	0,00000004	0,00609331
2008	0,00816	0,00138	0,090	0,011	0,301	0,0684	0,00000046	0,00150237
2009	0,00817	0,00146	0,092	0,011	0,349	0,0755	0,00000018	0,00254784

Source: WHOMD and HMD 2014, own elaboration

Table A. 29: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for ischaemic heart diseases mortality in Netherlands

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00192	0,00029	0,127	0,011	0,484	0,1047	0,00000000	0,00345778
1997	0,00169	0,00026	0,131	0,011	0,527	0,1117	0,00000014	0,00055591
1998	0,00156	0,00027	0,132	0,013	0,484	0,1251	0,00014787	0,00030960
1999	0,00146	0,00020	0,135	0,010	0,536	0,1031	0,00000002	0,00126566
2000	0,00143	0,00020	0,133	0,010	0,524	0,1042	0,00000006	0,00069752
2001	0,00125	0,00018	0,137	0,011	0,503	0,1115	0,00000002	0,00110353
2002	0,00121	0,00018	0,136	0,011	0,502	0,1153	0,00000004	0,00077249
2003	0,00104	0,00017	0,142	0,013	0,522	0,1283	0,00000002	0,00102695
2004	0,00092	0,00012	0,141	0,010	0,444	0,1066	0,00000001	0,00090915
2005	0,00084	0,00011	0,143	0,010	0,508	0,1066	0,00000000	0,00151225
2006	0,00096	0,00011	0,143	0,010	0,508	0,1066	0,00000000	0,00151225
2007	0,00074	0,00012	0,135	0,012	0,338	0,1306	0,00000004	0,00046225
2008	0,00063	0,00009	0,143	0,011	0,381	0,1221	0,00000001	0,00103893
2009	0,00051	0,00008	0,155	0,012	0,580	0,1409	0,00000001	0,00059201
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00514	0,00089	0,109	0,013	0,366	0,0863	0,00000003	0,00362339
1997	0,00474	0,00138	0,108	0,022	0,342	0,1371	0,00021958	0,00144095
1998	0,00462	0,00126	0,106	0,021	0,312	0,1274	0,00000027	0,00178738
1999	0,00433	0,00110	0,105	0,019	0,271	0,1153	0,00002991	0,00115709
2000	0,00390	0,00085	0,110	0,016	0,329	0,1039	0,00000035	0,00107953
2001	0,00346	0,00060	0,112	0,013	0,265	0,0854	0,00016129	0,00064711
2002	0,00296	0,00077	0,119	0,020	0,305	0,1261	0,00032859	0,00081470
2003	0,00305	0,00048	0,112	0,012	0,246	0,0785	0,00000001	0,00490967
2004	0,00190	0,00044	0,144	0,019	0,451	0,1246	0,00076906	0,00048575
2005	0,00178	0,00032	0,143	0,015	0,445	0,1014	0,00060477	0,00036001
2006	0,00174	0,00043	0,132	0,020	0,317	0,1263	0,00044363	0,00046790
2007	0,00137	0,00030	0,144	0,018	0,346	0,1173	0,00048144	0,00033427
2008	0,00126	0,00028	0,147	0,018	0,398	0,1210	0,00047859	0,00031371
2009	0,00157	0,00039	0,125	0,019	0,285	0,1272	0,00000011	0,00092229

Source: WHOMD and HMD 2014, own elaboration

Table A. 30: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for cerebrovascular diseases mortality in Netherlands

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00084	0,00009	0,180	0,009	0,735	0,0962	0,00000005	0,00038196
1997	0,00083	0,00011	0,177	0,011	0,685	0,1107	0,00000000	0,00149387
1998	0,00084	0,00010	0,173	0,010	0,647	0,1007	0,00000001	0,00090975
1999	0,00089	0,00011	0,170	0,010	0,648	0,1010	0,00000001	0,00078845
2000	0,00082	0,00010	0,175	0,009	0,700	0,0998	0,00000000	0,00166318
2001	0,00078	0,00009	0,171	0,009	0,576	0,0986	0,00000000	0,00148398
2002	0,00075	0,00008	0,175	0,009	0,641	0,0960	0,00000000	0,00115377
2003	0,00065	0,00007	0,182	0,009	0,694	0,0963	0,00000000	0,00126467
2004	0,00063	0,00007	0,182	0,009	0,746	0,1021	0,00000003	0,00036782
2005	0,00061	0,00007	0,174	0,009	0,616	0,1041	0,00000000	0,00140005
2006	0,00058	0,00007	0,168	0,009	0,483	0,1067	0,00000001	0,00072091
2007	0,00054	0,00006	0,169	0,010	0,543	0,1110	0,00000001	0,00058129
2008	0,00047	0,00005	0,173	0,009	0,526	0,1052	0,00000000	0,00682799
2009	0,00046	0,00005	0,177	0,009	0,635	0,1118	0,00000000	0,00070201
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00104	0,00023	0,190	0,021	0,668	0,1465	0,00051529	0,00026529
1997	0,00116	0,00024	0,181	0,020	0,621	0,1371	0,00023513	0,00027579
1998	0,00128	0,00029	0,163	0,021	0,455	0,1419	0,00013940	0,00032537
1999	0,00132	0,00025	0,160	0,018	0,406	0,1178	0,00000005	0,00089800
2000	0,00125	0,00021	0,164	0,015	0,482	0,1050	0,00000000	0,00347701
2001	0,00122	0,00026	0,160	0,019	0,441	0,1284	0,00000017	0,00048943
2002	0,00111	0,00023	0,168	0,019	0,479	0,1281	0,00017698	0,00026722
2003	0,00109	0,00022	0,161	0,018	0,423	0,1176	0,00000001	0,00226828
2004	0,00092	0,00021	0,165	0,020	0,406	0,1315	0,00015082	0,00023547
2005	0,00086	0,00017	0,168	0,018	0,458	0,1207	0,00000004	0,00071348
2006	0,00082	0,00016	0,164	0,017	0,426	0,1191	0,00000000	0,00218245
2007	0,00068	0,00015	0,169	0,019	0,427	0,1323	0,00013321	0,00017702
2008	0,00067	0,00015	0,160	0,019	0,344	0,1290	0,00009296	0,00017552
2009	0,00062	0,00013	0,167	0,018	0,436	0,1284	0,00000001	0,00146705

Source: WHOMD and HMD 2014, own elaboration

Table A. 31: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for remaining circulatory system diseases mortality in Netherlands

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00132	0,00017	0,145	0,010	0,151	0,0929	0,00000006	0,00054802
1997	0,00126	0,00017	0,147	0,010	0,172	0,0987	0,00005074	0,00019894
1998	0,00121	0,00016	0,148	0,010	0,146	0,0986	0,00001372	0,00018747
1999	0,00125	0,00015	0,147	0,010	0,153	0,0929	0,00002080	0,00018385
2000	0,00118	0,00014	0,156	0,009	0,264	0,0922	0,00008297	0,00016949
2001	0,00122	0,00013	0,151	0,009	0,204	0,0882	0,00000001	0,00108480
2002	0,00117	0,00012	0,154	0,008	0,213	0,0845	0,00000001	0,00143822
2003	0,00112	0,00011	0,157	0,008	0,248	0,0810	0,00000000	0,00228273
2004	0,00110	0,00011	0,154	0,008	0,207	0,0833	0,00000002	0,00076547
2005	0,00106	0,00010	0,159	0,007	0,283	0,0796	0,00000000	0,00280564
2006	0,00093	0,00009	0,165	0,008	0,288	0,0830	0,00000001	0,00112212
2007	0,00096	0,00009	0,160	0,007	0,216	0,0825	0,00000002	0,00052777
2008	0,00087	0,00008	0,167	0,007	0,285	0,0835	0,00000000	0,00345226
2009	0,00080	0,00007	0,169	0,007	0,298	0,0793	0,00000000	0,00108478
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00298	0,00070	0,124	0,020	0,144	0,1222	0,00020138	0,00074572
1997	0,00280	0,00053	0,126	0,016	0,145	0,1005	0,00000001	0,00343837
1998	0,00236	0,00053	0,132	0,019	0,145	0,1178	0,00088483	0,00057964
1999	0,00259	0,00057	0,127	0,018	0,122	0,1125	0,00040931	0,00061571
2000	0,00280	0,00056	0,125	0,017	0,128	0,1015	0,00001158	0,00060785
2001	0,00268	0,00057	0,121	0,017	0,062	0,1048	0,00000018	0,00102742
2002	0,00263	0,00052	0,128	0,016	0,142	0,1004	0,00000103	0,00056281
2003	0,00228	0,00045	0,134	0,016	0,112	0,1015	0,00025620	0,00049696
2004	0,00232	0,00048	0,131	0,017	0,104	0,1035	0,00008506	0,00052401
2005	0,00229	0,00040	0,132	0,014	0,106	0,0883	0,00000044	0,00046434
2006	0,00202	0,00035	0,139	0,014	0,166	0,0909	0,00000710	0,00038620
2007	0,00189	0,00033	0,139	0,014	0,125	0,0919	0,00009246	0,00037125
2008	0,00171	0,00026	0,146	0,013	0,178	0,0829	0,00000002	0,00165012
2009	0,00139	0,00023	0,156	0,014	0,185	0,0922	0,00032924	0,00026645

Source: WHOMD and HMD 2014, own elaboration

Table A. 32: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for respiratory system diseases mortality in Netherlands

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00053	0,00008	0,171	0,013	0,298	0,1232	0,00027188	0,00011079
1997	0,00064	0,00010	0,159	0,013	0,205	0,1240	0,00016660	0,00012656
1998	0,00066	0,00010	0,161	0,012	0,243	0,1227	0,00018575	0,00012963
1999	0,00069	0,00011	0,156	0,012	0,132	0,1183	0,00028918	0,00013980
2000	0,00081	0,00012	0,147	0,012	0,124	0,1143	0,00011077	0,00014999
2001	0,00082	0,00012	0,140	0,012	0,087	0,1154	0,00000001	0,00108577
2002	0,00086	0,00014	0,134	0,012	0,000	0,1203	0,00000001	0,00122956
2003	0,00089	0,00013	0,135	0,011	0,010	0,1100	0,00000909	0,00015822
2004	0,00077	0,00012	0,140	0,012	0,113	0,1223	0,00000048	0,00015142
2005	0,00083	0,00011	0,139	0,010	0,034	0,1059	0,00000002	0,00075935
2006	0,00080	0,00010	0,140	0,010	0,099	0,1031	0,00000001	0,00096377
2007	0,00078	0,00011	0,134	0,010	0,035	0,1068	0,00000000	0,00271565
2008	0,00077	0,00009	0,141	0,009	0,155	0,1015	0,00000001	0,00074483
2009	0,00072	0,00009	0,146	0,010	0,192	0,1092	0,00000000	0,00136579
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00192	0,00027	0,176	0,014	0,484	0,0940	0,00000005	0,00097122
1997	0,00192	0,00029	0,168	0,015	0,394	0,0977	0,00000010	0,00075537
1998	0,00199	0,00030	0,169	0,014	0,430	0,0958	0,00000008	0,00081754
1999	0,00175	0,00023	0,175	0,013	0,400	0,0852	0,00000012	0,00055197
2000	0,00175	0,00021	0,183	0,012	0,512	0,0799	0,00000004	0,00086046
2001	0,00144	0,00021	0,188	0,014	0,537	0,0964	0,00022093	0,00025479
2002	0,00153	0,00020	0,179	0,012	0,453	0,0818	0,00000000	0,00244445
2003	0,00123	0,00018	0,199	0,014	0,555	0,0920	0,00028398	0,00021666
2004	0,00128	0,00017	0,184	0,012	0,473	0,0845	0,00000000	0,00243048
2005	0,00142	0,00019	0,176	0,012	0,384	0,0814	0,00000001	0,00176044
2006	0,00121	0,00015	0,182	0,011	0,406	0,0771	0,00000000	0,00272135
2007	0,00122	0,00016	0,175	0,012	0,336	0,0792	0,00000003	0,00078659
2008	0,00108	0,00013	0,184	0,010	0,407	0,0722	0,00000001	0,00091371
2009	0,00116	0,00015	0,174	0,011	0,350	0,0787	0,00000010	0,00040129

Source: WHOMD and HMD 2014, own elaboration

Table A. 33: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for digestive system diseases of mortality in Netherlands

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00032	0,00008	0,152	0,020	0,188	0,1860	0,00011879	0,00009976
1997	0,00032	0,00008	0,156	0,021	0,319	0,2023	0,00007921	0,00010277
1998	0,00040	0,00010	0,133	0,019	0,050	0,1812	0,00000000	0,00453155
1999	0,00029	0,00007	0,166	0,019	0,420	0,1856	0,00005439	0,00008397
2000	0,00039	0,00008	0,142	0,016	0,201	0,1642	0,00000000	0,00207128
2001	0,00038	0,00008	0,145	0,016	0,232	0,1662	0,00000002	0,00048580
2002	0,00039	0,00008	0,145	0,016	0,232	0,1662	0,00000002	0,00048580
2003	0,00031	0,00007	0,156	0,018	0,246	0,1821	0,00012971	0,00008926
2004	0,00038	0,00008	0,145	0,016	0,238	0,1670	0,00000000	0,00190752
2005	0,00035	0,00008	0,146	0,016	0,285	0,1714	0,00000000	0,00101589
2006	0,00031	0,00007	0,155	0,017	0,365	0,1832	0,00005878	0,00008236
2007	0,00036	0,00006	0,140	0,012	0,223	0,1405	0,00000001	0,00048152
2008	0,00032	0,00006	0,154	0,016	0,428	0,1786	0,00000000	0,00112785
2009	0,00029	0,00006	0,150	0,015	0,342	0,1740	0,00000000	0,00122869
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00071	0,00037	0,109	0,043	0,000	0,2564	0,00000001	0,00254221
1997	0,00037	0,00016	0,164	0,040	0,385	0,2630	0,00034616	0,00018983
1998	0,00059	0,00030	0,115	0,042	0,001	0,2486	0,00012085	0,00032457
1999	0,00053	0,00026	0,131	0,041	0,139	0,2509	0,00012655	0,00028025
2000	0,00048	0,00019	0,150	0,035	0,309	0,2260	0,00016212	0,00021293
2001	0,00041	0,00015	0,155	0,033	0,279	0,2147	0,00020688	0,00017416
2002	0,00043	0,00017	0,158	0,035	0,319	0,2229	0,00015659	0,00019669
2003	0,00054	0,00023	0,129	0,035	0,058	0,2148	0,00013940	0,00025319
2004	0,00040	0,00017	0,157	0,035	0,339	0,2263	0,00030253	0,00019153
2005	0,00019	0,00007	0,214	0,031	0,641	0,2097	0,00043765	0,00009364
2006	0,00028	0,00010	0,178	0,032	0,420	0,2102	0,00030105	0,00012508
2007	0,00051	0,00017	0,131	0,026	0,147	0,1660	0,00000003	0,00082363
2008	0,00026	0,00010	0,171	0,032	0,358	0,2113	0,00032945	0,00012096
2009	0,00025	0,00010	0,167	0,033	0,300	0,2178	0,00030169	0,00011967

Source: WHOMD and HMD 2014, own elaboration

Table A. 34: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for external causes of mortality in Netherlands

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00015	0,00005	0,149	0,028	0,000	0,2594	0,00009582	0,00006758
1997	0,00012	0,00004	0,155	0,028	0,000	0,2719	0,00012999	0,00005670
1998	0,00011	0,00004	0,162	0,030	0,092	0,2869	0,00014328	0,00005441
1999	0,00008	0,00003	0,193	0,026	0,324	0,2541	0,00013961	0,00004106
2000	0,00012	0,00005	0,153	0,030	0,000	0,2853	0,00015115	0,00006056
2001	0,00015	0,00004	0,154	0,023	0,000	0,2292	0,00002511	0,00005523
2002	0,00015	0,00005	0,149	0,025	0,000	0,2526	0,00005362	0,00006037
2003	0,00012	0,00004	0,159	0,025	0,000	0,2491	0,00011373	0,00005043
2004	0,00012	0,00004	0,155	0,025	0,000	0,2485	0,00006864	0,00005160
2005	0,00013	0,00004	0,155	0,023	0,000	0,2379	0,00008073	0,00004996
2006	0,00009	0,00002	0,186	0,021	0,282	0,2301	0,00009680	0,00003585
2007	0,00014	0,00004	0,154	0,022	0,029	0,2330	0,00004582	0,00004862
2008	0,00013	0,00003	0,165	0,019	0,114	0,2156	0,00005539	0,00004201
2009	0,00013	0,00003	0,173	0,020	0,308	0,2255	0,00006701	0,00004216
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00029	0,00017	0,130	0,051	0,000	0,3154	0,00013839	0,00018941
1997	0,00006	0,00003	0,264	0,050	0,768	0,3303	0,00036736	0,00005540
1998	0,00008	0,00005	0,226	0,053	0,563	0,3518	0,00034401	0,00006839
1999	0,00012	0,00009	0,167	0,064	0,054	0,4007	0,00037092	0,00010669
2000	0,00010	0,00005	0,214	0,048	0,437	0,3116	0,00030656	0,00007451
2001	0,00010	0,00005	0,219	0,047	0,447	0,3030	0,00035245	0,00007117
2002	0,00019	0,00011	0,160	0,050	0,139	0,3124	0,00019933	0,00012739
2003	0,00012	0,00007	0,190	0,053	0,231	0,3282	0,00030461	0,00009487
2004	0,00006	0,00003	0,269	0,043	0,813	0,2829	0,00035901	0,00005167
2005	0,00018	0,00009	0,159	0,045	0,023	0,2775	0,00023713	0,00011361
2006	0,00013	0,00006	0,188	0,039	0,225	0,2512	0,00034539	0,00008157
2007	0,00018	0,00009	0,162	0,043	0,075	0,2737	0,00020580	0,00010895
2008	0,00013	0,00006	0,172	0,040	0,030	0,2496	0,00029959	0,00008537
2009	0,00009	0,00004	0,223	0,039	0,456	0,2559	0,00031079	0,00006348

Source: WHOMD and HMD 2014, own elaboration

Table A. 35: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for the remaining causes of death of mortality in Netherlands

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00130	0,00014	0,160	0,008	0,102	0,0811	0,00038308	0,00017468
1997	0,00140	0,00015	0,157	0,008	0,065	0,0810	0,00012638	0,00018009
1998	0,00131	0,00013	0,164	0,008	0,163	0,0806	0,00022843	0,00016749
1999	0,00161	0,00016	0,150	0,008	0,003	0,0732	0,00001653	0,00018956
2000	0,00121	0,00011	0,175	0,007	0,274	0,0745	0,00036773	0,00014526
2001	0,00152	0,00014	0,161	0,007	0,164	0,0741	0,00031023	0,00017719
2002	0,00173	0,00015	0,154	0,007	0,075	0,0681	0,00000003	0,00078368
2003	0,00138	0,00012	0,166	0,007	0,145	0,0710	0,00028417	0,00015637
2004	0,00133	0,00012	0,166	0,007	0,194	0,0725	0,00023270	0,00014981
2005	0,00134	0,00012	0,160	0,007	0,092	0,0715	0,00000005	0,00046233
2006	0,00140	0,00011	0,157	0,006	0,057	0,0660	0,00000001	0,00088225
2007	0,00121	0,00010	0,162	0,006	0,094	0,0709	0,00006698	0,00012710
2008	0,00122	0,00009	0,162	0,006	0,052	0,0678	0,00000003	0,00053529
2009	0,00103	0,00008	0,172	0,006	0,152	0,0717	0,00021551	0,00010692
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00224	0,00047	0,131	0,019	0,000	0,1156	0,00030341	0,00051703
1997	0,00236	0,00052	0,126	0,019	0,000	0,1148	0,00015789	0,00055916
1998	0,00196	0,00038	0,138	0,017	0,000	0,1085	0,00056931	0,00042780
1999	0,00202	0,00033	0,138	0,014	0,007	0,0895	0,00062301	0,00036941
2000	0,00182	0,00033	0,148	0,016	0,070	0,0988	0,00089805	0,00038137
2001	0,00147	0,00026	0,171	0,016	0,193	0,1018	0,00143937	0,00031343
2002	0,00211	0,00037	0,147	0,015	0,094	0,0940	0,00066948	0,00041592
2003	0,00162	0,00028	0,165	0,015	0,155	0,0952	0,00110020	0,00032641
2004	0,00156	0,00029	0,156	0,016	0,079	0,0985	0,00104565	0,00033757
2005	0,00131	0,00021	0,175	0,014	0,227	0,0892	0,00117162	0,00025764
2006	0,00184	0,00030	0,144	0,014	0,016	0,0865	0,00041385	0,00034560
2007	0,00129	0,00020	0,168	0,013	0,174	0,0870	0,00099393	0,00024938
2008	0,00114	0,00017	0,183	0,013	0,260	0,0823	0,00087773	0,00020951
2009	0,00122	0,00017	0,177	0,012	0,273	0,0821	0,00087505	0,00021553

Source: WHOMD and HMD 2014, own elaboration

Table A. 36: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for overall mortality in Norway

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00881	0,00090	0,136	0,008	0,152	0,0679	0,00266912	0,00107351
1997	0,01093	0,00119	0,121	0,008	0,031	0,0691	0,00021404	0,00135892
1998	0,01025	0,00110	0,126	0,008	0,096	0,0722	0,00021249	0,00126362
1999	0,00864	0,00090	0,136	0,008	0,120	0,0726	0,00270497	0,00107508
2000	0,00886	0,00092	0,136	0,008	0,169	0,0716	0,00147772	0,00109593
2001	0,01022	0,00106	0,122	0,007	0,019	0,0679	0,00000698	0,00122757
2002	0,00951	0,00097	0,127	0,007	0,046	0,0682	0,00083136	0,00114481
2003	0,00880	0,00093	0,128	0,007	0,049	0,0706	0,00117644	0,00109508
2004	0,00927	0,00098	0,122	0,007	0,009	0,0705	0,00000076	0,00112480
2005	0,00812	0,00082	0,127	0,007	0,003	0,0695	0,00130307	0,00097432
2006	0,00876	0,00082	0,125	0,007	0,000	0,0657	0,00000004	0,00336644
2007	0,00754	0,00069	0,135	0,007	0,111	0,0682	0,00169783	0,00084635
2008	0,00804	0,00074	0,127	0,006	0,018	0,0677	0,00126808	0,00087570
2009	0,00688	0,00059	0,139	0,006	0,150	0,0692	0,00201003	0,00072970
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,02334	0,00335	0,108	0,011	0,111	0,0664	0,00000196	0,00358126
1997	0,02262	0,00354	0,109	0,012	0,094	0,0721	0,00001162	0,00378977
1998	0,02194	0,00354	0,112	0,012	0,145	0,0756	0,00000726	0,00379367
1999	0,01865	0,00282	0,123	0,012	0,161	0,0738	0,00337257	0,00308416
2000	0,01983	0,00320	0,115	0,012	0,119	0,0761	0,00034607	0,00343662
2001	0,01813	0,00280	0,122	0,012	0,152	0,0747	0,00151361	0,00305356
2002	0,01647	0,00250	0,124	0,012	0,136	0,0732	0,00354592	0,00275653
2003	0,01228	0,00186	0,141	0,012	0,216	0,0774	0,00671989	0,00213895
2004	0,01194	0,00181	0,141	0,012	0,230	0,0758	0,00589189	0,00208293
2005	0,01485	0,00217	0,125	0,011	0,144	0,0681	0,00172692	0,00239970
2006	0,01010	0,00136	0,148	0,011	0,253	0,0687	0,00624682	0,00160502
2007	0,01104	0,00152	0,142	0,011	0,202	0,0695	0,00551579	0,00175577
2008	0,01275	0,00179	0,130	0,011	0,122	0,0687	0,00299523	0,00200193
2009	0,00895	0,00127	0,153	0,011	0,289	0,0751	0,00649733	0,00149327

Source: WHOMD and HMD 2014, own elaboration

Table A. 37: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for neoplasms mortality in Norway

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00357	0,00250	0,075	0,037	0,163	0,2734	0,00205167	0,00262826
1997	0,00540	0,00260	0,059	0,022	0,186	0,1723	0,00000335	0,00267287
1998	0,00485	0,00203	0,068	0,021	0,312	0,1808	0,00000001	0,01507222
1999	0,00532	0,00276	0,054	0,022	0,093	0,1585	0,00000009	0,00686251
2000	0,00499	0,00247	0,068	0,024	0,256	0,1989	0,00000039	0,00291336
2001	0,00538	0,00252	0,056	0,021	0,068	0,1504	0,00000044	0,00277177
2002	0,00533	0,00264	0,059	0,022	0,141	0,1670	0,00000010	0,00617627
2003	0,00512	0,00198	0,063	0,018	0,227	0,1486	0,00000001	0,01378304
2004	0,00528	0,00182	0,055	0,015	0,063	0,1197	0,00000780	0,00189434
2005	0,00390	0,00200	0,077	0,027	0,323	0,2359	0,00109989	0,00211585
2006	0,00469	0,00154	0,072	0,017	0,308	0,1562	0,00000020	0,00260413
2007	0,00517	0,00215	0,062	0,019	0,234	0,1703	0,00000007	0,00591698
2008	0,00503	0,00188	0,071	0,019	0,517	0,1956	0,00002979	0,00196097
2009	0,00475	0,00294	0,073	0,031	0,421	0,3006	0,00000027	0,00418784
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00855	0,00250	0,092	0,019	0,381	0,1284	0,00000030	0,00339779
1997	0,00795	0,00364	0,092	0,030	0,312	0,1872	0,00033521	0,00378042
1998	0,00682	0,00295	0,101	0,030	0,377	0,1921	0,00141205	0,00310012
1999	0,00855	0,00310	0,079	0,022	0,188	0,1261	0,00000042	0,00351660
2000	0,00783	0,00303	0,090	0,025	0,342	0,1577	0,00000010	0,00715058
2001	0,00767	0,00217	0,095	0,019	0,364	0,1236	0,00000104	0,00228682
2002	0,00665	0,00309	0,099	0,031	0,325	0,1936	0,00122956	0,00324779
2003	0,00761	0,00361	0,084	0,029	0,188	0,1702	0,00000041	0,00415424
2004	0,00757	0,00221	0,090	0,018	0,298	0,1162	0,00000015	0,00421201
2005	0,00701	0,00199	0,100	0,019	0,393	0,1234	0,00000008	0,00515188
2006	0,00558	0,00241	0,101	0,028	0,306	0,1747	0,00163995	0,00256015
2007	0,00519	0,00180	0,113	0,024	0,419	0,1586	0,00191295	0,00194080
2008	0,00669	0,00214	0,096	0,020	0,311	0,1335	0,00000065	0,00225292
2009	0,00611	0,00200	0,100	0,021	0,351	0,1440	0,00048974	0,00211288

Source: WHOMD and HMD 2014, own elaboration

Table A. 38: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for ischaemic heart diseases mortality in Norway

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00191	0,00035	0,140	0,014	0,345	0,1295	0,00000001	0,00365558
1997	0,00189	0,00032	0,142	0,013	0,358	0,1240	0,00000003	0,00167856
1998	0,00157	0,00024	0,157	0,012	0,569	0,1240	0,00005106	0,00030190
1999	0,00153	0,00031	0,156	0,016	0,472	0,1589	0,00012714	0,00037679
2000	0,00149	0,00028	0,157	0,014	0,532	0,1441	0,00000840	0,00034963
2001	0,00132	0,00021	0,156	0,012	0,432	0,1275	0,00000000	0,00522747
2002	0,00124	0,00022	0,157	0,013	0,376	0,1371	0,00000001	0,00191549
2003	0,00111	0,00019	0,161	0,013	0,403	0,1404	0,00000000	0,00546314
2004	0,00088	0,00014	0,177	0,013	0,589	0,1399	0,00000001	0,00181345
2005	0,00082	0,00015	0,162	0,014	0,368	0,1522	0,00000000	0,00494946
2006	0,00089	0,00017	0,149	0,014	0,166	0,1530	0,00000001	0,00199818
2007	0,00074	0,00013	0,166	0,013	0,396	0,1496	0,00000002	0,00094838
2008	0,00078	0,00015	0,154	0,015	0,240	0,1671	0,00000001	0,00112860
2009	0,00071	0,00014	0,162	0,015	0,395	0,1800	0,00000002	0,00086937
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00522	0,00181	0,118	0,027	0,296	0,1721	0,00134471	0,00195342
1997	0,00596	0,00139	0,114	0,018	0,288	0,1166	0,00002024	0,00150503
1998	0,00543	0,00163	0,122	0,024	0,369	0,1535	0,00002560	0,00175857
1999	0,00442	0,00131	0,130	0,024	0,358	0,1546	0,00089792	0,00143846
2000	0,00322	0,00093	0,147	0,024	0,402	0,1611	0,00131787	0,00106086
2001	0,00387	0,00119	0,129	0,025	0,262	0,1587	0,00026668	0,00129810
2002	0,00320	0,00106	0,135	0,027	0,254	0,1683	0,00102412	0,00118862
2003	0,00219	0,00063	0,164	0,024	0,461	0,1619	0,00152608	0,00074995
2004	0,00178	0,00055	0,168	0,026	0,457	0,1720	0,00151214	0,00067596
2005	0,00151	0,00043	0,177	0,024	0,525	0,1591	0,00114104	0,00053224
2006	0,00242	0,00084	0,117	0,025	0,032	0,1567	0,000000024	0,00131867
2007	0,00162	0,00056	0,145	0,027	0,163	0,1767	0,00090387	0,00065425
2008	0,00114	0,00036	0,175	0,026	0,416	0,1776	0,00130908	0,00045753
2009	0,00092	0,00030	0,186	0,027	0,501	0,1899	0,00135731	0,00039791

Source: WHOMD and HMD 2014, own elaboration

Table A. 39: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for cerebrovascular diseases mortality in Norway

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00094	0,00018	0,185	0,016	0,742	0,1551	0,00000002	0,00106875
1997	0,00081	0,00014	0,195	0,015	0,827	0,1487	0,00000000	0,00773824
1998	0,00090	0,00016	0,183	0,014	0,738	0,1502	0,00000001	0,00175296
1999	0,00075	0,00014	0,188	0,015	0,717	0,1612	0,00000001	0,00108672
2000	0,00063	0,00012	0,202	0,016	0,906	0,1661	0,00000001	0,00137626
2001	0,00063	0,00011	0,193	0,014	0,808	0,1522	0,00000000	0,00197153
2002	0,00071	0,00013	0,182	0,015	0,688	0,1571	0,00000001	0,00114349
2003	0,00066	0,00014	0,180	0,017	0,608	0,1767	0,00000000	0,00183835
2004	0,00073	0,00016	0,160	0,017	0,383	0,1784	0,00000000	0,00275198
2005	0,00054	0,00011	0,183	0,016	0,651	0,1732	0,00000001	0,00138988
2006	0,00059	0,00010	0,167	0,014	0,373	0,1565	0,00000000	0,01766902
2007	0,00052	0,00010	0,179	0,015	0,638	0,1773	0,00000000	0,00145810
2008	0,00050	0,00009	0,178	0,014	0,572	0,1666	0,00000000	0,00245288
2009	0,00040	0,00007	0,197	0,015	0,882	0,1849	0,00000000	0,00257300

Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00161	0,00038	0,167	0,022	0,461	0,1510	0,00000000	0,00513146
1997	0,00128	0,00033	0,189	0,024	0,645	0,1727	0,00000009	0,00091268
1998	0,00116	0,00031	0,203	0,025	0,794	0,1836	0,00020797	0,00037498
1999	0,00127	0,00036	0,177	0,026	0,504	0,1782	0,00000001	0,00276678
2000	0,00104	0,00033	0,186	0,029	0,575	0,2041	0,00030185	0,00040316
2001	0,00118	0,00032	0,169	0,024	0,477	0,1688	0,00000001	0,00314833
2002	0,00089	0,00033	0,185	0,032	0,508	0,2182	0,00032183	0,00040470
2003	0,00088	0,00034	0,176	0,033	0,454	0,2254	0,00013039	0,00040106
2004	0,00077	0,00027	0,190	0,030	0,615	0,2080	0,00021582	0,00033008
2005	0,00073	0,00026	0,178	0,030	0,427	0,2030	0,00037863	0,00032318
2006	0,00068	0,00018	0,181	0,023	0,467	0,1628	0,00000001	0,00178996
2007	0,00079	0,00027	0,164	0,028	0,381	0,1967	0,00000001	0,00183314
2008	0,00052	0,00017	0,202	0,028	0,635	0,2007	0,00026176	0,00021259
2009	0,00073	0,00030	0,158	0,033	0,282	0,2262	0,00021048	0,00034711

Source: WHOMD and HMD 2014, own elaboration

Table A. 40: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for remaining circulatory system diseases mortality in Norway

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00080	0,00014	0,175	0,014	0,321	0,1413	0,00000000	0,00316404
1997	0,00072	0,00013	0,183	0,015	0,377	0,1460	0,00000000	0,00355302
1998	0,00078	0,00014	0,179	0,014	0,381	0,1457	0,00000000	0,00252353
1999	0,00087	0,00016	0,166	0,015	0,165	0,1503	0,00000000	0,00218243
2000	0,00073	0,00023	0,175	0,024	0,251	0,2332	0,00010651	0,00029735
2001	0,00065	0,00011	0,195	0,013	0,545	0,1409	0,00001782	0,00015777
2002	0,00067	0,00011	0,186	0,013	0,432	0,1415	0,00000000	0,00172665
2003	0,00078	0,00014	0,162	0,014	0,121	0,1459	0,00000000	0,00222883
2004	0,00068	0,00012	0,175	0,014	0,255	0,1474	0,00000001	0,00130596
2005	0,00068	0,00014	0,158	0,016	0,040	0,1677	0,00000003	0,00072659
2006	0,00056	0,00008	0,186	0,012	0,359	0,1364	0,00000000	0,00281942
2007	0,00059	0,00010	0,183	0,013	0,380	0,1463	0,00000001	0,00121742
2008	0,00050	0,00008	0,179	0,013	0,229	0,1547	0,00000001	0,00072565
2009	0,00049	0,00008	0,183	0,013	0,315	0,1594	0,00000003	0,00049224
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00198	0,00062	0,135	0,026	0,126	0,1701	0,00000000	0,01122930
1997	0,00194	0,00063	0,136	0,028	0,117	0,1753	0,00000005	0,00233384
1998	0,00196	0,00051	0,138	0,022	0,161	0,1452	0,00000001	0,00568806
1999	0,00144	0,00042	0,172	0,026	0,399	0,1753	0,00036778	0,00050585
2000	0,00133	0,00041	0,173	0,028	0,360	0,1851	0,00032429	0,00049710
2001	0,00100	0,00029	0,200	0,027	0,563	0,1834	0,00077302	0,00037846
2002	0,00169	0,00049	0,140	0,024	0,097	0,1549	0,00000000	0,00842673
2003	0,00097	0,00032	0,179	0,029	0,302	0,1906	0,00055048	0,00040590
2004	0,00092	0,00029	0,183	0,027	0,361	0,1777	0,00046747	0,00036482
2005	0,00078	0,00026	0,186	0,028	0,360	0,1854	0,00062902	0,00033801
2006	0,00062	0,00019	0,209	0,026	0,519	0,1783	0,00084245	0,00026628
2007	0,00055	0,00017	0,215	0,026	0,503	0,1798	0,00112097	0,00025726
2008	0,00093	0,00031	0,163	0,028	0,148	0,1850	0,00035095	0,00037759
2009	0,00105	0,00038	0,143	0,028	0,000	0,1887	0,00003602	0,00042626

Source: WHOMD and HMD 2014, own elaboration

Table A. 41: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for respiratory system diseases mortality in Norway

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00060	0,00017	0,156	0,021	0,050	0,1994	0,00008277	0,00021154
1997	0,00051	0,00015	0,160	0,022	0,000	0,2069	0,00046124	0,00020818
1998	0,00024	0,00007	0,222	0,022	0,616	0,2255	0,00070459	0,00012706
1999	0,00057	0,00016	0,160	0,022	0,081	0,2105	0,00044846	0,00022693
2000	0,00078	0,00022	0,142	0,021	0,000	0,1985	0,00013853	0,00027297
2001	0,00057	0,00018	0,153	0,023	0,000	0,2173	0,00047668	0,00024843
2002	0,00068	0,00017	0,156	0,019	0,109	0,1827	0,00032753	0,00023054
2003	0,00064	0,00019	0,141	0,022	0,005	0,2084	0,00026537	0,00025286
2004	0,00065	0,00020	0,145	0,022	0,258	0,2189	0,00008357	0,00024675
2005	0,00064	0,00019	0,141	0,021	0,025	0,2109	0,00030264	0,00025203
2006	0,00088	0,00028	0,124	0,021	0,000	0,2086	0,00004730	0,00033120
2007	0,00053	0,00013	0,175	0,018	0,605	0,2022	0,00032972	0,00018912
2008	0,00065	0,00019	0,138	0,020	0,091	0,2103	0,00044235	0,00024776
2009	0,00066	0,00017	0,146	0,019	0,272	0,2116	0,00037194	0,00022489
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00128	0,00037	0,163	0,026	0,288	0,1753	0,00000002	0,00215298
1997	0,00099	0,00033	0,174	0,030	0,259	0,2008	0,00044000	0,00040412
1998	0,00125	0,00037	0,156	0,027	0,215	0,1771	0,00000002	0,00205878
1999	0,00140	0,00042	0,151	0,026	0,116	0,1709	0,00000009	0,00116467
2000	0,00129	0,00046	0,156	0,031	0,169	0,2026	0,00024674	0,00053737
2001	0,00118	0,00028	0,171	0,021	0,286	0,1457	0,00000001	0,00242312
2002	0,00118	0,00029	0,174	0,022	0,330	0,1504	0,00005174	0,00035875
2003	0,00101	0,00034	0,165	0,029	0,205	0,1881	0,00029218	0,00041315
2004	0,00078	0,00029	0,169	0,031	0,204	0,2010	0,00042634	0,00036017
2005	0,00128	0,00037	0,154	0,023	0,206	0,1529	0,00000000	0,00560658
2006	0,00096	0,00030	0,175	0,026	0,384	0,1760	0,00027996	0,00036160
2007	0,00117	0,00025	0,163	0,018	0,320	0,1253	0,00000000	0,00571945
2008	0,00124	0,00029	0,155	0,019	0,246	0,1331	0,00000001	0,00242056
2009	0,00102	0,00032	0,163	0,025	0,305	0,1773	0,00010242	0,00037562

Source: WHOMD and HMD 2014, own elaboration

Table A. 42: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for digestive system diseases of mortality in Norway

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00016	0,00007	0,203	0,038	0,968	0,3803	0,00010024	0,00010378
1997	0,00014	0,00007	0,193	0,040	0,643	0,3924	0,00023580	0,00010541
1998	0,00018	0,00009	0,177	0,041	0,566	0,4081	0,00014838	0,00012447
1999	0,00025	0,00011	0,173	0,038	0,759	0,3886	0,00008380	0,00014331
2000	0,00036	0,00014	0,128	0,029	0,179	0,2873	0,00001805	0,00016442
2001	0,00029	0,00013	0,147	0,033	0,386	0,3279	0,00000001	0,00129238
2002	0,00035	0,00020	0,121	0,039	0,000	0,3635	0,00000000	0,00413969
2003	0,00028	0,00015	0,136	0,041	0,123	0,3995	0,00001615	0,00017888
2004	0,00033	0,00017	0,127	0,038	0,052	0,3691	0,00000002	0,00115130
2005	0,00032	0,00017	0,119	0,038	0,000	0,3751	0,00000000	0,01225809
2006	0,00023	0,00012	0,146	0,037	0,260	0,3834	0,00007149	0,00014631
2007	0,00028	0,00014	0,131	0,036	0,251	0,3706	0,00000001	0,00101036
2008	0,00026	0,00013	0,130	0,036	0,077	0,3852	0,00005017	0,00015754
2009	0,00030	0,00012	0,125	0,027	0,080	0,3108	0,00000000	0,00155251
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00056	0,00043	0,114	0,061	0,051	0,3726	0,00000856	0,00047244
1997	0,00055	0,00047	0,114	0,066	0,071	0,4083	0,00000001	0,00312223
1998	0,00032	0,00031	0,156	0,082	0,375	0,5332	0,00035987	0,00035762
1999	0,00026	0,00022	0,178	0,074	0,446	0,4789	0,00024623	0,00028337
2000	0,00039	0,00031	0,143	0,067	0,264	0,4330	0,00013494	0,00035738
2001	0,00050	0,00032	0,124	0,050	0,102	0,3161	0,00000001	0,00220681
2002	0,00038	0,00041	0,124	0,084	0,000	0,5047	0,00012612	0,00045252
2003	0,00030	0,00034	0,118	0,085	0,000	0,5046	0,00034147	0,00038395
2004	0,00008	0,00006	0,274	0,066	1,164	0,4587	0,00049293	0,00011819
2005	0,00011	0,00009	0,214	0,072	0,616	0,4762	0,00040190	0,00014694
2006	0,00046	0,00086	0,107	0,131	0,000	0,7653	0,00003485	0,00091538
2007	0,00027	0,00026	0,137	0,073	0,144	0,4661	0,00019675	0,00029897
2008	0,00025	0,00027	0,133	0,081	0,075	0,5195	0,00035754	0,00031233
2009	0,00018	0,00017	0,169	0,075	0,338	0,5042	0,00025386	0,00021562

Source: WHOMD and HMD 2014, own elaboration

Table A. 43: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for external causes of mortality in Norway

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00015	0,00007	0,171	0,038	0,112	0,3599	0,00014647	0,00010198
1997	0,00025	0,00014	0,141	0,040	0,000	0,3680	0,00006718	0,00016988
1998	0,00013	0,00006	0,183	0,039	0,258	0,3836	0,00017387	0,00009419
1999	0,00007	0,00003	0,241	0,034	0,815	0,3605	0,00022586	0,00006054
2000	0,00017	0,00008	0,167	0,040	0,181	0,3858	0,00009503	0,00011496
2001	0,00023	0,00011	0,145	0,038	0,000	0,7001	0,00000224	0,00014359
2002	0,00011	0,00005	0,197	0,040	0,518	0,4053	0,00016507	0,00009028
2003	0,00011	0,00005	0,202	0,036	0,667	0,3778	0,00007051	0,00007619
2004	0,00012	0,00006	0,168	0,041	0,135	0,4190	0,00017488	0,00009124
2005	0,00017	0,00007	0,155	0,034	0,000	0,3519	0,00000715	0,00009255
2006	0,00021	0,00009	0,142	0,033	0,000	0,3484	0,00000560	0,00010986
2007	0,00013	0,00006	0,178	0,032	0,345	0,3583	0,00015444	0,00008110
2008	0,00019	0,00008	0,149	0,031	0,029	0,3420	0,00000000	0,00223189
2009	0,00018	0,00007	0,155	0,029	0,058	0,3334	0,00005485	0,00009331
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
0,00023	0,00020	0,187	0,079	0,361	0,5051	0,00061977	0,00027060	0,00023
0,00036	0,00030	0,141	0,070	0,000	0,4293	0,00040208	0,00036446	0,00036
0,00028	0,00028	0,147	0,086	0,006	0,5261	0,00043949	0,00034444	0,00028
0,00021	0,00017	0,168	0,067	0,023	0,4242	0,00069011	0,00022840	0,00021
0,00014	0,00013	0,198	0,086	0,250	0,5497	0,00064127	0,00019725	0,00014
0,00024	0,00020	0,159	0,072	0,017	0,4588	0,00043195	0,00024418	0,00024
0,00013	0,00012	0,226	0,079	0,615	0,5085	0,00060922	0,00019997	0,00013
0,00017	0,00014	0,174	0,072	0,094	0,4519	0,00060080	0,00020301	0,00017
0,00017	0,00017	0,165	0,083	0,000	0,5127	0,00064171	0,00022936	0,00017
0,00027	0,00022	0,150	0,066	0,043	0,4135	0,00020864	0,00026467	0,00027
0,00020	0,00014	0,184	0,057	0,322	0,3760	0,00036340	0,00018174	0,00020
0,00007	0,00004	0,265	0,049	0,757	0,3389	0,00054189	0,00008930	0,00007
0,00009	0,00007	0,241	0,058	0,571	0,3940	0,00045003	0,00011988	0,00009
0,00007	0,00005	0,266	0,065	0,776	0,4436	0,00058366	0,00010966	0,00007

Source: WHOMD and HMD 2014, own elaboration

Table A. 44: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for the remaining causes of death of mortality in Norway

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00084	0,00021	0,153	0,019	0,045	0,1732	0,00047893	0,00027001
1997	0,00126	0,00027	0,137	0,016	0,000	0,1513	0,00000000	0,00506873
1998	0,00126	0,00085	0,140	0,050	0,002	0,4408	0,00004273	0,00099069
1999	0,00116	0,00027	0,142	0,018	0,000	0,1661	0,00027033	0,00034709
2000	0,00102	0,00023	0,149	0,017	0,012	0,1620	0,00027450	0,00028879
2001	0,00096	0,00020	0,161	0,016	0,183	0,1566	0,00049614	0,00027183
2002	0,00110	0,00022	0,154	0,015	0,095	0,1482	0,00028138	0,00028057
2003	0,00095	0,00019	0,165	0,015	0,187	0,1481	0,00051843	0,00026017
2004	0,00101	0,00018	0,164	0,013	0,235	0,1393	0,00000000	0,00979626
2005	0,00131	0,00023	0,146	0,013	0,000	0,1323	0,00000008	0,00072929
2006	0,00115	0,00018	0,162	0,012	0,190	0,1286	0,00000004	0,00086014
2007	0,00124	0,00019	0,151	0,011	0,000	0,1219	0,00000004	0,00086297
2008	0,00131	0,00019	0,152	0,011	0,084	0,1227	0,00000007	0,00064062
2009	0,00125	0,00017	0,153	0,010	0,033	0,1196	0,00000012	0,00044464
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1996	0,00144	0,00063	0,132	0,036	0,000	0,2220	0,00078289	0,00071740
1997	0,00136	0,00061	0,140	0,038	0,035	0,2360	0,00128729	0,00071552
1998	0,00158	0,00068	0,131	0,035	0,000	0,2129	0,00096524	0,00077903
1999	0,00109	0,00037	0,177	0,030	0,365	0,1972	0,00146525	0,00047284
2000	0,00226	0,00085	0,118	0,029	0,021	0,1803	0,00000001	0,00602204
2001	0,00053	0,00023	0,223	0,038	0,572	0,2482	0,00251095	0,00037139
2002	0,00178	0,00081	0,129	0,036	0,005	0,2199	0,00091118	0,00091028
2003	0,00111	0,00038	0,175	0,029	0,313	0,1915	0,00136686	0,00049262
2004	0,00090	0,00029	0,190	0,028	0,374	0,1820	0,00130286	0,00039699
2005	0,00212	0,00077	0,128	0,028	0,000	0,1695	0,00029261	0,00086453
2006	0,00160	0,00050	0,148	0,025	0,116	0,1602	0,00061053	0,00058444
2007	0,00131	0,00037	0,168	0,023	0,225	0,1531	0,00108556	0,00046489
2008	0,00157	0,00048	0,150	0,024	0,085	0,1593	0,00083993	0,00056665
2009	0,00120	0,00034	0,178	0,023	0,322	0,1607	0,00108952	0,00042789

Source: WHOMD and HMD 2014, own elaboration

Table A. 45: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for overall mortality in Sweden

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00854	0,00065	0,133	0,005	0,120	0,0506	0,00203255	0,00076384
1998	0,00887	0,00063	0,131	0,005	0,118	0,0489	0,00145373	0,00074356
1999	0,00883	0,00056	0,132	0,005	0,120	0,0452	0,00147394	0,00066438
2000	0,00853	0,00057	0,133	0,005	0,118	0,0473	0,00151881	0,00067522
2001	0,00908	0,00061	0,126	0,005	0,035	0,0464	0,00115910	0,00071294
2002	0,00922	0,00060	0,125	0,005	0,000	0,0453	0,00088076	0,00070169
2003	0,00824	0,00054	0,130	0,005	0,048	0,0474	0,00158176	0,00064729
2004	0,00872	0,00057	0,125	0,005	0,014	0,0463	0,00070247	0,00067169
2005	0,00825	0,00051	0,126	0,004	0,000	0,0444	0,00136907	0,00060330
2006	0,00807	0,00047	0,127	0,004	0,000	0,0438	0,00105595	0,00056878
2007	0,00772	0,00045	0,130	0,004	0,017	0,0452	0,00165860	0,00054007
2008	0,00734	0,00040	0,131	0,004	0,001	0,0436	0,00168082	0,00049024
2009	0,00749	0,00041	0,129	0,004	0,002	0,0438	0,00138124	0,00050104
2010	0,00744	0,00039	0,129	0,004	0,000	0,0434	0,00137350	0,00047486
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,01686	0,00176	0,121	0,0079	0,122	0,0481	0,00353164	0,00193730
1998	0,01524	0,00154	0,129	0,0078	0,169	0,0485	0,00497918	0,00171954
1999	0,01713	0,00169	0,120	0,0074	0,117	0,0453	0,00219142	0,00186046
2000	0,01631	0,00160	0,120	0,0073	0,096	0,0449	0,00216924	0,00177240
2001	0,01498	0,00154	0,122	0,0076	0,082	0,0469	0,00307946	0,00170842
2002	0,01395	0,00135	0,128	0,0072	0,118	0,0449	0,00393242	0,00152715
2003	0,01297	0,00121	0,133	0,0070	0,174	0,0440	0,00470033	0,00138862
2004	0,01154	0,00121	0,137	0,0079	0,181	0,0502	0,00542591	0,00139552
2005	0,01245	0,00114	0,132	0,0068	0,147	0,0440	0,00416955	0,00128912
2006	0,01176	0,00107	0,134	0,0068	0,164	0,0450	0,00411610	0,00122413
2007	0,01096	0,00096	0,136	0,0065	0,145	0,0441	0,00426993	0,00109383
2008	0,00960	0,00082	0,146	0,0065	0,227	0,0447	0,00546084	0,00096351
2009	0,00980	0,00083	0,142	0,0063	0,190	0,0447	0,00444081	0,00095449
2010	0,01042	0,00090	0,137	0,0064	0,164	0,0458	0,00359561	0,00101660

Source: WHOMD and HMD 2014, own elaboration

Table A. 46: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for neoplasms mortality in Sweden

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00518	0,00161	0,063	0,015	0,293	0,1240	0,00000788	0,00166624
1998	0,00504	0,00199	0,063	0,018	0,319	0,1576	0,00000003	0,00828110
1999	0,00505	0,00218	0,066	0,021	0,405	0,1888	0,00000057	0,00224966
2000	0,00483	0,00132	0,071	0,014	0,458	0,1306	0,00005149	0,00138133
2001	0,00534	0,00135	0,066	0,012	0,373	0,1126	0,00000002	0,00664475
2002	0,00520	0,00191	0,064	0,017	0,343	0,1522	0,00000008	0,00483858
2003	0,00506	0,00161	0,066	0,015	0,386	0,1417	0,00000020	0,00267317
2004	0,00503	0,00145	0,069	0,014	0,449	0,1387	0,00000019	0,00247821
2005	0,00516	0,00154	0,066	0,014	0,463	0,1425	0,00000008	0,00397783
2006	0,00511	0,00136	0,067	0,013	0,405	0,1271	0,00000105	0,00141407
2007	0,00516	0,00111	0,068	0,011	0,438	0,1130	0,00002738	0,00116364
2008	0,00497	0,00138	0,065	0,013	0,326	0,1320	0,00006674	0,00142314
2009	0,00498	0,00128	0,067	0,012	0,430	0,1310	0,00000002	0,00764316
2010	0,00481	0,00103	0,070	0,011	0,476	0,1212	0,00000002	0,00496152
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00723	0,00200	0,089	0,017	0,324	0,1090	0,00000558	0,00208448
1998	0,00712	0,00195	0,088	0,017	0,274	0,1049	0,00004003	0,00204144
1999	0,00708	0,00287	0,088	0,025	0,298	0,1502	0,00000859	0,00299230
2000	0,00660	0,00145	0,095	0,014	0,350	0,0909	0,00000004	0,00571131
2001	0,00678	0,00165	0,089	0,015	0,285	0,0937	0,00000053	0,00173595
2002	0,00673	0,00155	0,089	0,014	0,293	0,0880	0,00000002	0,00791282
2003	0,00674	0,00129	0,094	0,012	0,353	0,0791	0,00000347	0,00137358
2004	0,00664	0,00145	0,093	0,013	0,346	0,0887	0,00000018	0,00254747
2005	0,00685	0,00188	0,085	0,016	0,257	0,1005	0,00000104	0,00196418
2006	0,00619	0,00135	0,100	0,014	0,416	0,0966	0,00000002	0,00773164
2007	0,00427	0,00092	0,122	0,015	0,571	0,1117	0,00185159	0,00100980
2008	0,00584	0,00125	0,097	0,014	0,365	0,0945	0,00007262	0,00131930
2009	0,00565	0,00121	0,101	0,014	0,420	0,1000	0,00001886	0,00128222
2010	0,00582	0,00086	0,093	0,009	0,310	0,0695	0,00000002	0,00469778

Source: WHOMD and HMD 2014, own elaboration

Table A. 47: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for ischaemic heart diseases mortality in Sweden

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00192	0,00026	0,144	0,010	0,304	0,0966	0,00000004	0,00109399
1998	0,00176	0,00021	0,151	0,009	0,401	0,0898	0,00000004	0,00087019
1999	0,00167	0,00019	0,148	0,009	0,321	0,0897	0,00000169	0,00023556
2000	0,00155	0,00017	0,150	0,008	0,327	0,0888	0,00000003	0,00081846
2001	0,00155	0,00019	0,146	0,009	0,264	0,0963	0,00000001	0,00202730
2002	0,00143	0,00016	0,152	0,008	0,311	0,0908	0,00000003	0,00076167
2003	0,00140	0,00016	0,148	0,009	0,249	0,0951	0,00000005	0,00063965
2004	0,00133	0,00014	0,149	0,008	0,283	0,0899	0,00000001	0,00171419
2005	0,00115	0,00012	0,151	0,008	0,212	0,0868	0,00000001	0,00087869
2006	0,00117	0,00011	0,148	0,007	0,129	0,0846	0,00000001	0,00097217
2007	0,00104	0,00011	0,153	0,008	0,189	0,0943	0,00000157	0,00014098
2008	0,00104	0,00010	0,147	0,007	0,080	0,0890	0,00000004	0,00045654
2009	0,00094	0,00010	0,148	0,008	0,068	0,0938	0,00000002	0,00067277
2010	0,00090	0,00009	0,144	0,007	0,000	0,0940	0,00000000	0,00135948
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00610	0,00142	0,106	0,017	0,145	0,0982	0,00000204	0,00151729
1998	0,00422	0,00080	0,132	0,015	0,263	0,0939	0,00149286	0,00089767
1999	0,00483	0,00099	0,120	0,015	0,203	0,0942	0,00060272	0,00108764
2000	0,00364	0,00073	0,130	0,015	0,194	0,0953	0,00133448	0,00082342
2001	0,00321	0,00065	0,133	0,016	0,164	0,0976	0,00154217	0,00074896
2002	0,00255	0,00043	0,155	0,013	0,327	0,0880	0,00200684	0,00052681
2003	0,00275	0,00050	0,146	0,014	0,270	0,0895	0,00156509	0,00058364
2004	0,00279	0,00052	0,138	0,014	0,217	0,0910	0,00102001	0,00059272
2005	0,00273	0,00049	0,138	0,014	0,210	0,0901	0,00088541	0,00056531
2006	0,00288	0,00056	0,130	0,014	0,134	0,0947	0,00042559	0,00062603
2007	0,00196	0,00036	0,150	0,014	0,239	0,0972	0,00130386	0,00042705
2008	0,00165	0,00029	0,162	0,014	0,314	0,0952	0,00145971	0,00035141
2009	0,00204	0,00041	0,137	0,015	0,100	0,1034	0,00073720	0,00046616
2010	0,00183	0,00033	0,147	0,014	0,201	0,0992	0,00057550	0,00037671

Source: WHOMD and HMD 2014, own elaboration

Table A. 48: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for cerebrovascular diseases mortality in Sweden

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00076	0,00010	0,187	0,011	0,772	0,1112	0,00000000	0,00225641
1998	0,00081	0,00010	0,181	0,010	0,706	0,1104	0,00000002	0,00064822
1999	0,00076	0,00009	0,187	0,010	0,798	0,1093	0,00000000	0,00249302
2000	0,00072	0,00009	0,187	0,010	0,788	0,1077	0,00000000	0,00212608
2001	0,00074	0,00009	0,176	0,010	0,595	0,1080	0,00000023	0,00017883
2002	0,00082	0,00010	0,167	0,009	0,499	0,1059	0,00000000	0,00188845
2003	0,00070	0,00008	0,177	0,010	0,641	0,1102	0,00000000	0,00206098
2004	0,00064	0,00007	0,177	0,009	0,608	0,1069	0,00000000	0,00642890
2005	0,00062	0,00007	0,168	0,009	0,497	0,1103	0,00000000	0,00180448
2006	0,00060	0,00007	0,169	0,009	0,507	0,1060	0,00000000	0,00138609
2007	0,00057	0,00007	0,166	0,010	0,437	0,1190	0,00000001	0,00080683
2008	0,00057	0,00006	0,166	0,008	0,360	0,1051	0,00000000	0,01297483
2009	0,00057	0,00008	0,160	0,011	0,313	0,1295	0,00000034	0,00013076
2010	0,00047	0,00005	0,174	0,008	0,460	0,1078	0,00000000	0,00230813
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00108	0,00022	0,182	0,018	0,564	0,1251	0,00042825	0,00027542
1998	0,00112	0,00024	0,175	0,019	0,511	0,1281	0,00040227	0,00029415
1999	0,00130	0,00026	0,161	0,017	0,446	0,1146	0,00000000	0,00470864
2000	0,00120	0,00027	0,165	0,019	0,456	0,1267	0,00015357	0,00031513
2001	0,00087	0,00018	0,188	0,018	0,585	0,1236	0,00031409	0,00022495
2002	0,00074	0,00017	0,193	0,019	0,601	0,1298	0,00060169	0,00022009
2003	0,00104	0,00022	0,170	0,017	0,492	0,1180	0,00000041	0,00028129
2004	0,00098	0,00020	0,164	0,017	0,444	0,1184	0,00000002	0,00119448
2005	0,00083	0,00018	0,172	0,018	0,469	0,1262	0,00012483	0,00021770
2006	0,00079	0,00018	0,167	0,018	0,415	0,1312	0,00009911	0,00021313
2007	0,00088	0,00018	0,151	0,016	0,282	0,1185	0,00000001	0,00136088
2008	0,00076	0,00016	0,172	0,017	0,476	0,1227	0,00000015	0,00033789
2009	0,00072	0,00013	0,171	0,015	0,438	0,1163	0,00000001	0,00106374
2010	0,00073	0,00015	0,157	0,017	0,297	0,1270	0,00000001	0,00138260

Source: WHOMD and HMD 2014, own elaboration

Table A. 49: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for remaining circulatory system diseases mortality in Sweden

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00073	0,00009	0,180	0,010	0,300	0,1045	0,00000001	0,00077146
1998	0,00076	0,00009	0,178	0,010	0,350	0,1006	0,00000000	0,00193938
1999	0,00076	0,00008	0,178	0,009	0,319	0,0932	0,00000000	0,00125606
2000	0,00075	0,00008	0,179	0,009	0,358	0,0966	0,00000001	0,00074627
2001	0,00076	0,00008	0,171	0,009	0,224	0,0945	0,00000001	0,00108377
2002	0,00074	0,00008	0,168	0,008	0,143	0,0909	0,00000001	0,00098987
2003	0,00072	0,00008	0,166	0,009	0,091	0,0963	0,00000000	0,00115224
2004	0,00059	0,00006	0,180	0,009	0,261	0,0975	0,00000001	0,00053287
2005	0,00061	0,00005	0,182	0,007	0,348	0,0834	0,00000001	0,00071468
2006	0,00055	0,00005	0,189	0,008	0,402	0,0937	0,00000000	0,00278632
2007	0,00057	0,00005	0,187	0,007	0,304	0,0918	0,00000000	0,00162733
2008	0,00051	0,00005	0,188	0,007	0,277	0,0907	0,00000000	0,00088083
2009	0,00057	0,00005	0,180	0,007	0,258	0,0915	0,00000000	0,00097773
2010	0,00060	0,00005	0,176	0,007	0,139	0,0860	0,00000001	0,00058636
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00155	0,00031	0,153	0,017	0,150	0,1074	0,00000129	0,00035912
1998	0,00180	0,00042	0,139	0,019	0,093	0,1194	0,00002579	0,00047548
1999	0,00150	0,00027	0,156	0,015	0,188	0,1009	0,00000010	0,00071226
2000	0,00125	0,00025	0,162	0,016	0,165	0,1068	0,00045020	0,00030189
2001	0,00153	0,00033	0,146	0,017	0,097	0,1105	0,00000001	0,00222550
2002	0,00083	0,00016	0,192	0,016	0,367	0,1052	0,00061042	0,00020846
2003	0,00123	0,00025	0,155	0,016	0,126	0,1063	0,00022616	0,00030371
2004	0,00057	0,00011	0,215	0,016	0,498	0,1102	0,00076248	0,00016484
2005	0,00081	0,00015	0,192	0,015	0,375	0,1038	0,00043944	0,00019526
2006	0,00059	0,00011	0,212	0,015	0,478	0,1074	0,00073920	0,00016083
2007	0,00079	0,00014	0,191	0,015	0,358	0,1058	0,00044142	0,00018691
2008	0,00061	0,00011	0,205	0,015	0,437	0,1063	0,00059461	0,00015310
2009	0,00046	0,00008	0,226	0,015	0,565	0,1092	0,00077547	0,00012844
2010	0,00063	0,00011	0,199	0,015	0,349	0,1098	0,00056424	0,00015241

Source: WHOMD and HMD 2014, own elaboration

Table A. 50: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for respiratory system diseases mortality in Sweden

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00050	0,00012	0,147	0,017	0,000	0,1648	0,00009300	0,00014396
1998	0,00043	0,00011	0,148	0,018	0,066	0,1751	0,00025472	0,00013869
1999	0,00062	0,00015	0,133	0,017	0,000	0,1638	0,00004444	0,00017435
2000	0,00044	0,00010	0,146	0,017	0,046	0,1692	0,00032778	0,00013265
2001	0,00054	0,00014	0,130	0,018	0,000	0,1784	0,00006418	0,00016617
2002	0,00052	0,00012	0,135	0,017	0,001	0,1676	0,00022960	0,00015382
2003	0,00054	0,00013	0,130	0,017	0,002	0,1689	0,00013474	0,00015896
2004	0,00055	0,00015	0,125	0,019	0,000	0,2026	0,00011531	0,00018089
2005	0,00054	0,00012	0,130	0,016	0,000	0,1612	0,00014807	0,00014715
2006	0,00048	0,00011	0,131	0,016	0,034	0,1711	0,00016600	0,00013970
2007	0,00060	0,00014	0,122	0,016	0,030	0,1694	0,00003596	0,00016603
2008	0,00061	0,00013	0,120	0,015	0,000	0,1594	0,00000002	0,00083922
2009	0,00058	0,00012	0,129	0,015	0,184	0,1674	0,00000001	0,00098546
2010	0,00055	0,00014	0,114	0,017	0,057	0,1890	0,00010906	0,00016558
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00093	0,00021	0,159	0,019	0,175	0,1225	0,00000001	0,00176281
1998	0,00083	0,00018	0,160	0,018	0,178	0,1199	0,00000000	0,00383856
1999	0,00078	0,00018	0,170	0,020	0,253	0,1312	0,00016225	0,00022316
2000	0,00093	0,00022	0,151	0,019	0,155	0,1272	0,00000006	0,00075420
2001	0,00073	0,00021	0,154	0,023	0,128	0,1481	0,00016172	0,00024345
2002	0,00092	0,00020	0,144	0,018	0,090	0,1146	0,00000003	0,00097733
2003	0,00076	0,00019	0,162	0,020	0,276	0,1337	0,00007491	0,00023142
2004	0,00060	0,00016	0,170	0,021	0,308	0,1413	0,00015502	0,00019339
2005	0,00076	0,00019	0,152	0,020	0,165	0,1340	0,00001993	0,00022370
2006	0,00070	0,00016	0,151	0,018	0,180	0,1253	0,00000001	0,00111914
2007	0,00067	0,00015	0,149	0,017	0,127	0,1200	0,00000012	0,00035396
2008	0,00072	0,00018	0,149	0,020	0,173	0,1395	0,00000009	0,00049946
2009	0,00063	0,00016	0,159	0,020	0,279	0,1446	0,00000114	0,00018085
2010	0,00064	0,00017	0,152	0,020	0,304	0,1510	0,00000000	0,00221421

Source: WHOMD and HMD 2014, own elaboration

Table A. 51: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for digestive system diseases of mortality in Sweden

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00032	0,00014	0,120	0,031	0,003	0,2779	0,00000002	0,00084000
1998	0,00032	0,00011	0,122	0,025	0,020	0,2400	0,00000000	0,00133828
1999	0,00018	0,00007	0,162	0,028	0,386	0,2845	0,00016157	0,00008707
2000	0,00026	0,00010	0,136	0,029	0,173	0,2930	0,00009215	0,00012271
2001	0,00025	0,00010	0,128	0,029	0,000	0,2814	0,00013288	0,00012304
2002	0,00021	0,00007	0,155	0,024	0,296	0,2545	0,00014153	0,00009028
2003	0,00026	0,00009	0,133	0,025	0,107	0,2577	0,00007552	0,00011279
2004	0,00024	0,00009	0,134	0,026	0,084	0,2635	0,00011656	0,00010549
2005	0,00020	0,00008	0,144	0,028	0,358	0,2976	0,00016957	0,00009610
2006	0,00032	0,00011	0,115	0,023	0,000	0,2463	0,00000000	0,00303479
2007	0,00026	0,00010	0,122	0,026	0,000	0,2844	0,00015604	0,00011462
2008	0,00025	0,00008	0,137	0,022	0,260	0,2530	0,00004411	0,00009171
2009	0,00028	0,00010	0,117	0,024	0,010	0,2615	0,00009364	0,00011684
2010	0,00023	0,00008	0,119	0,025	0,000	0,2825	0,00011166	0,00010095
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00011	0,00006	0,239	0,049	0,750	0,3199	0,00052932	0,00009927
1998	0,00019	0,00011	0,175	0,047	0,295	0,3013	0,00044851	0,00014086
1999	0,00039	0,00027	0,123	0,053	0,019	0,3212	0,00021822	0,00029652
2000	0,00051	0,00039	0,103	0,054	0,000	0,3133	0,00009838	0,00041592
2001	0,00023	0,00013	0,160	0,044	0,274	0,2846	0,00037027	0,00016204
2002	0,00031	0,00021	0,132	0,050	0,000	0,3042	0,00033525	0,00024609
2003	0,00008	0,00005	0,234	0,054	0,648	0,3476	0,00069066	0,00009799
2004	0,00007	0,00005	0,231	0,059	0,622	0,3846	0,00060698	0,00009586
2005	0,00026	0,00015	0,155	0,044	0,336	0,2904	0,00029099	0,00017787
2006	0,00021	0,00015	0,149	0,055	0,173	0,3557	0,00053979	0,00018416
2007	0,00008	0,00005	0,215	0,050	0,559	0,3457	0,00056229	0,00008641
2008	0,00019	0,00011	0,161	0,044	0,286	0,3054	0,00049007	0,00014234
2009	0,00010	0,00006	0,201	0,045	0,514	0,3232	0,00047785	0,00008803
2010	0,00021	0,00017	0,130	0,059	0,000	0,3990	0,00040409	0,00019989

Source: WHOMD and HMD 2014, own elaboration

Table A. 52: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for external causes of mortality in Sweden

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00017	0,00008	0,144	0,034	0,084	0,3176	0,00006227	0,00009956
1998	0,00013	0,00006	0,159	0,038	0,235	0,3661	0,00017756	0,00008532
1999	0,00015	0,00006	0,147	0,032	0,111	0,3160	0,00014358	0,00008277
2000	0,00009	0,00004	0,173	0,031	0,222	0,3239	0,00020415	0,00005696
2001	0,00017	0,00007	0,139	0,030	0,000	0,3001	0,00007726	0,00009105
2002	0,00015	0,00006	0,146	0,028	0,000	0,2845	0,00010848	0,00007750
2003	0,00016	0,00006	0,147	0,026	0,000	0,2756	0,00005621	0,00007249
2004	0,00015	0,00006	0,153	0,027	0,210	0,2834	0,00012888	0,00007647
2005	0,00013	0,00004	0,152	0,023	0,000	0,2508	0,00020788	0,00006134
2006	0,00012	0,00004	0,153	0,026	0,000	0,2834	0,00014793	0,00005874
2007	0,00013	0,00004	0,156	0,024	0,081	0,2747	0,00012209	0,00005630
2008	0,00013	0,00004	0,152	0,022	0,001	0,2571	0,00013984	0,00005694
2009	0,00011	0,00003	0,157	0,022	0,000	0,2656	0,00018479	0,00005020
2010	0,00015	0,00004	0,145	0,022	0,000	0,2581	0,00014612	0,00006132
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00016	0,00010	0,176	0,052	0,172	0,3293	0,00063507	0,00013843
1998	0,00005	0,00003	0,288	0,055	1,046	0,3617	0,00082908	0,00008109
1999	0,00009	0,00005	0,229	0,050	0,626	0,3277	0,00069289	0,00009863
2000	0,00017	0,00012	0,164	0,059	0,106	0,3578	0,00066547	0,00017055
2001	0,00011	0,00006	0,207	0,049	0,406	0,3192	0,00071146	0,00010671
2002	0,00019	0,00012	0,161	0,050	0,000	0,3080	0,00059874	0,00016602
2003	0,00005	0,00003	0,259	0,048	0,641	0,3075	0,00078696	0,00007772
2004	0,00006	0,00004	0,249	0,049	0,554	0,3180	0,00080355	0,00008388
2005	0,00008	0,00004	0,230	0,039	0,504	0,2627	0,00082061	0,00007616
2006	0,00013	0,00007	0,197	0,044	0,354	0,2937	0,00070317	0,00010966
2007	0,00014	0,00007	0,193	0,040	0,316	0,2735	0,00057452	0,00010404
2008	0,00007	0,00004	0,229	0,044	0,460	0,2992	0,00073549	0,00008525
2009	0,00004	0,00002	0,272	0,049	0,728	0,3389	0,00070509	0,00006363
2010	0,00005	0,00003	0,267	0,042	0,835	0,3047	0,00072181	0,00006505

Source: WHOMD and HMD 2014, own elaboration

Table A. 53: Gamma-Gompertz-Makeham parameter estimates with associated standard errors for the remaining causes of death of mortality in Sweden

Females								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00078	0,00013	0,157	0,013	0,042	0,1192	0,00028107	0,00016807
1998	0,00078	0,00012	0,162	0,011	0,080	0,1129	0,00024454	0,00015341
1999	0,00090	0,00012	0,161	0,010	0,071	0,1031	0,00021362	0,00015544
2000	0,00084	0,00011	0,163	0,010	0,074	0,1023	0,00028163	0,00014563
2001	0,00078	0,00009	0,177	0,009	0,240	0,0971	0,00029554	0,00012961
2002	0,00105	0,00012	0,159	0,009	0,062	0,0925	0,00003501	0,00015595
2003	0,00081	0,00009	0,174	0,009	0,227	0,0969	0,00026725	0,00013174
2004	0,00091	0,00010	0,165	0,009	0,144	0,0936	0,00010794	0,00014004
2005	0,00105	0,00011	0,155	0,008	0,000	0,0864	0,00014268	0,00014741
2006	0,00105	0,00010	0,155	0,007	0,000	0,8395	0,00000000	0,00156225
2007	0,00111	0,00011	0,159	0,007	0,102	0,0862	0,00000000	0,00218333
2008	0,00107	0,00010	0,162	0,007	0,136	0,0833	0,00008245	0,00013244
2009	0,00106	0,00009	0,163	0,006	0,158	0,0792	0,00000001	0,00113051
2010	0,00126	0,00011	0,155	0,006	0,096	0,0792	0,00000013	0,00027490
Males								
Year	$\hat{a}(y)$	s.e. $\hat{a}(y)$	$\hat{b}(y)$	s.e. $\hat{b}(y)$	$\hat{\gamma}(y)$	s.e. $\hat{\gamma}(y)$	$\hat{c}(y)$	s.e. $\hat{c}(y)$
1997	0,00096	0,00028	0,151	0,024	0,036	0,1460	0,00071413	0,00033666
1998	0,00087	0,00020	0,176	0,020	0,255	0,1282	0,00086268	0,00026212
1999	0,00143	0,00036	0,138	0,020	0,000	0,1231	0,00034128	0,00041719
2000	0,00078	0,00017	0,191	0,018	0,351	0,1188	0,00097608	0,00023771
2001	0,00097	0,00020	0,172	0,017	0,199	0,1126	0,00075870	0,00026661
2002	0,00168	0,00036	0,136	0,017	0,000	0,1053	0,00000017	0,00070695
2003	0,00091	0,00020	0,177	0,017	0,240	0,1131	0,00089307	0,00026252
2004	0,00099	0,00022	0,168	0,018	0,170	0,1163	0,00104872	0,00028367
2005	0,00174	0,00038	0,134	0,017	0,000	0,1066	0,00000006	0,00122316
2006	0,00118	0,00023	0,161	0,016	0,162	0,1054	0,00076226	0,00028583
2007	0,00138	0,00029	0,150	0,016	0,034	0,1098	0,00045407	0,00034617
2008	0,00108	0,00020	0,173	0,014	0,257	0,1009	0,00084288	0,00024867
2009	0,00151	0,00027	0,151	0,014	0,117	0,0966	0,00038284	0,00031194
2010	0,00185	0,00032	0,142	0,013	0,101	0,0961	0,00020524	0,00036772

Source: WHOMD and HMD 2014, own elaboration

