Universitat Autònoma de Barcelona

Centre d'Estudis Demogràfics

MEMÒRIA DE RECERCA DEL DOCTORAT EN DEMOGRAFIA

DEPARTAMENT DE GEOGRAFIA / CENTRE D' ESTUDIS DEMOGRÀFICS

UNIVERSITAT AUTÒNOMA DE BARCELONA

OLD AGE MORTALITY A STATE OF THE ART

Núria Calduch Verdiell

Director: Dr. Jeroen Spijker

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Cover image: Ah Puch (The God of Death of the Maya People)

Agraïments

No puc dir que la redacció d'aquesta memòria hagi estat un procès llarg com acostuma a ser si no que tot el contrari, ha estat un procès curt però molt intens i l'he pogut dur a terme gràcies a l'ajut i recolzament que he rebut per part de tantes persones.

Voldria començar els meus agraïments per a la Dra. Anna Cabré, directora del Centre d' Estudis Demogràfics per haver-me donat l'oportunitat de realitzar aquesta memòria. Per a mi a significat molt més que un treball de recerca m'ha fet entrar en un nou mon, el de la investigació. Al Jeroen Spijker el director d'aquesta memòria, per tota la seva inesgotable dedicació, estic segura que sense el seu ajut el resultat d' aquesta memòria hagués estat un altre.

Agrair també a l'Amand Blanes, ja que d' ell vaig rebre els primers consells per a dur a terme aquesta memòria, al Daniel Devolder per facilitar-me tants articles que em van servir per fer madurar el tema d'investigació, a la Candi Abella qui sempre ha tingut a mà qualsevol dels llibres que he necessitat i a tots els companys del CED que em preguntaven constantment com anava aquesta tesina.

També voldria agrair a tots els amics i amigues per haver aguantat durant tots aquest temps només sentir-me parlar de la tesina *(per a després de la tesina...)*.

I molt especialment agrair als meus pares i germans que tot i la distància m'han donat tot el seu recolzament dia a dia, al meu avi per tots els seus savis discursos i per acabar voldria dedicar aquesta memòria a la meva àvia.

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"En España somos poquitos demógrafos, y la demografía no llega a ser una ciencia, es el arte de desvelar misterios y resolver paradojas"

Anna Cabré[1]

CHAPTER 1 INTRODUCTION

1.1. Introduction

Many demographers who analyze mortality changes over time in low-mortality countries share two common views. On the one hand, that most of the gain in life expectancy before the first half of the twentieth century was due to large reductions in mortality early in life and on the other hand that recent and future gains in human life durations are and will be mainly due to mortality decline among the elderly. As a consequence, in recent years more attention has been paid to adult mortality and especially to that of the very old. The subject is likely to be the scene of a lively renewed debate, because the growing number and proportion of elderly has very important implications for social policy.

This Phd research report (tesina) aims to obtain a better understanding of the recent changes in mortality of the elderly population in developed countries[2].It's essence will be a critical thinking, without performing empirical work, which means that the analysis and concepts of the actual demographic literature will form the main body of this work. It basically reviews and discusses the recent work on old age mortality that was published between 1980 and 2006. However, some older work is evoked

[1] Anna Cabré Pla (2000) El País Semanal, nº 1224.12 de marzo de 2000.
"In Spain we are few demographers, and the demography does not get to be a science, is the art to keep awake mysteries and to solve paradoxes"

[2] The developed countries include those of Europe and North America, as well as Australia, New Zealand and Japan.

given its continued importance today (e.g. Vincent 1951; Greenwood & Irwin 1939; Gompertz (1820, 1825, 1862,1872)).

The literature aims to list research issues examined recently and to identify still unanswered questions that should be investigated in the years to come. There are two central questions: "*Which age and cause specific mortality trends caused these recent changes in the life expectancy?" and "Did the increase in life expectancy of the elderly population accelerate, continue or decline in recent years?*

In order to answer the first question, the evolution of recent mortality developments at older ages are discussed in terms of the Epidemiological Transition, the Compression of Mortality, the Limit of Human Life Span and the Rectangularization of the Survival Curve. From a theoretical point of view the Biodemography, the Evolutionary Theory of Senescence and the Reliability Theory of Ageing are analysed and finally there is a review of the existing mathematical models of old age mortality in developed countries. Although the PhD Research Report is entirely dedicated to these issues, a brief introduction to each of them is provided in the following sub-section.

1.2. Theoretical background to old age mortality

Epidemiological Transition Theory

During the Epidemiologic Transition changes in the cause-of-death pattern from mainly infectious diseases and external causes of death to chronic diseases were accompanied by a shift in the age pattern of mortality from younger towards older ages. During the third stage mortality began to concentrate at the older ages and was mainly caused by chronic diseases. The objective of this theory's analysis is to study the trend of the last stage of the epidemiologic transition in developed countries, and to find out the extend to which this fourth stage, during which deaths by degenerative

diseases are postponed to older ages, has been experienced, and if we are heading to, or already in, new stages.

The study of the Epidemiological Transition Theory allows a better insight of the processes behind the trends in mortality and causes of death in developed countries.

Compression of mortality

Mortality is defined as being compressed when there is a decrease in the variability of the age of death. Regarding the history of compression of mortality there exist sufficient evidence to prove that the transition from high to low mortality was accompanied by large compression of mortality, with deaths occurring at older ages and in an increasingly narrower age range. Today in developed countries the middle half of the distribution of age-specific deaths is concentrated in a range of about 15 years, usually somewhere between ages 75 and 90.

Long-term changes in the variability of age at death had significant effects on the perceptions, attitudes, and behaviours of individuals and, in turn, on society in general. Thus, these broad consequences of the historical compression of mortality merit a fully detailed analysis to understand the continuous decline of old age mortality in developed countries.

The human life span limit

A fundamental question in ageing research is whether humans and other species possess an immutable life-span limit, as the world record of human life-span seems to continue moving upward over time (Oeppen & Vaupel 2002). Projections of duration of life for humans based on mathematical models have led some researchers to claim that there is no lower limit to death rates or upper limit to life expectancy, and that a life expectancy of 100 will be achieved in the 21st century (Carnes, Olshansky & Grahn 2003). Two important questions arise from these observations. First, has

this upward trend been stable over time, or has it changed pace in recent years? Second, what explains the increase in the maximum age at death?

Rectangularitzation of human survival curve

Rectangularization is defined as a trend toward a more rectangular shape of the survival curve due to increased survival and concentration of deaths around the mean age at death. That is, the variability in the age at death declines and deaths are being compressed into upper years of life. After Fries introduced the concept of rectangularization of the survival curve (Fries 1980), the question of whether there is or not rectangularization and how to measure it has been addressed in several studies (Fries (2002,1984); Cheung 2001; Yashin et al. 2001; Coale 1996; Manton & Stallard 1996; Eakin & Witten 1995; Go, Brustrom, Lynch & Aldwin 1995; Rothenberg, Lentzner, Parker 1991; Myers & Manton 1984).

Because rectangularization is necessary for mortality decline on old age, the development of a better understanding of the rectangularization of the survival curve at older ages in developed countries plays an important role in this research.

Biodemography

Biodemography proposes explanations for life table patterns in human populations that are not evident in the absence of broader biological concepts. For example, it provides explanations of sex differentials in life expectancies, whether life span limits exists, and also of the deceleration of mortality at older ages that has been observed in virtually every large-scale life-table study on insects. Hence, there are strong biological arguments for the analysis of the law of mortality and the search for better explanatory models.

The emerging field of biodemography will provide the context, perspective and a frame of reference for the interpretation and modelling of human

mortality data and it will set a new light on those factors correlated with the survival history of our species and those factors that explain demographic variations between populations.

Evolutionary Theories of Ageing

After twenty years of experiments testing the evolutionary theories of ageing, we could accept them because they currently offer the most plausible explanation of ageing (Le Bourg 2001), as they try to explain the remarkable differences in observed ageing rates and longevity records of different biological species (compare, for example, mice and humans). Evolutionary explanations of ageing are based on three major evolutionary theories: the theory of programmed death (Weismann 1882), the theory of the accumulation of mutations at old age (Medawar 1951), and the theory of antagonistic pleiotropy (Williams 1957).

It would seem that the evolutionary theories of ageing, currently offer the most plausible explanations of ageing.

Reliability Theory of Ageing

Reliability theory is a body of ideas, mathematical models, and methods directed to predict, estimate, understand, and optimize the lifespan distribution of systems and their components (adapted from Barlow & Proschan 1965). The theory explains why the age-related increase in mortality rates declines at older ages.

The Reliability theory seems to be a promising approach for developing a comprehensive biodemographic theory of ageing and longevity integrating mathematical methods with specific biological knowledge.

1.3. A chronology of the earliest and recent models of old age mortality

In the distant past, the age pattern of mortality of the oldest old (80 years old and over) was not well-known. Knowledge of mortality among persons approaching the age of 100 (or more), in particular, was mainly based on unreliable statistics and theoretical concepts. In recent decades, this situation has gradually changed. Efforts have been made to improve past as well as recent mortality statistics and to develop methods of estimating mortality age patterns in cases where data are inaccurate. The reason that underlies these new developments is quite simple. As a result of a 50-year persistent decline in mortality, the number of persons surviving to very old ages has increased dramatically. As a result, institutions involved in social policies that strive to balance the needs of, and resources for the elderly, have become interested in reliable and detailed population projections.

Although Chapter Three provides more in depth information on how old age mortality is estimated , below is a brief historical review of the most important mathematical models that have been developed to describe and explain human mortality, starting with the first detailed investigation of mortality by Graunt (1661).

- In **1661 Graunt** carries out the first detailed investigation of mortality of Londoners ("Natural and Political Observations made upon the Bills of Mortality").

- In **1693 Edmond Halley** published an article with following title in the Philosophical Transactions of the Royal Society of London: "An Estimate of the Degrees of the Mortality of Mankind, drawn from curious Tables of the Births and Funerals at the City of Breslaw; with an attempt to ascertain the Price of Annuities upon Lives" .This important work contains the first ever known mortality table and had a fundamental influence on mortality statistics and on social statistics in general.

- In **1725 De Moivre** published *"Annuities on Lives"* where proposed a piecewise linear survivor function $S(x)-S(x+t)=a\ t$ assuming that the probability of remaining alive decreased with age in an arithmetic progression.

- In **1746 Deparcieux** used a life table method to show increases in life expectancies and his results were published in the 1746 treatise '*Essai sur les probabilités de la durée de la vie humaine*'.

- **In 1766 Bernoulli** first studies the properties of mortality intensity $\mu(x)$.

- **In 1772 Lambert** calls the inverse of this the 'force of vitality' and develops the earliest mathematical models for the survivor function $S(x)$. He was the first expresses the gradual 'exhaustion of man's power' in a mathematical formula.

- **In 1798 Malthus** predicted the exponential increase of human population as a consequence of the misery and famine that was suffered at the time unless family size was regulated. The model was later adjusted by Verhulst in 1838, to limit the population growth predictions.

- **In 1825 Benjamin Gompertz** proposed the first explanatory model, and the most influential parametric mortality model in the literature. He recognised that an exponential pattern in age captured the behaviour of human mortality for large portions of the life table. Later was found that at advanced ages mortality rates increase less rapidly than an exponential function. However, the close fit of the Gompertz function to empirical data suggested that a "law of mortality" may exist to explain the age patterns at death for individuals and populations.

- **In 1860 Makeham** noticed that the Gompertz equation failed to capture the behaviour of mortality at higher ages and added a constant term in order to correct for this deficiency. The constant can be thought of representing the risk of death by causes that are independent of age.

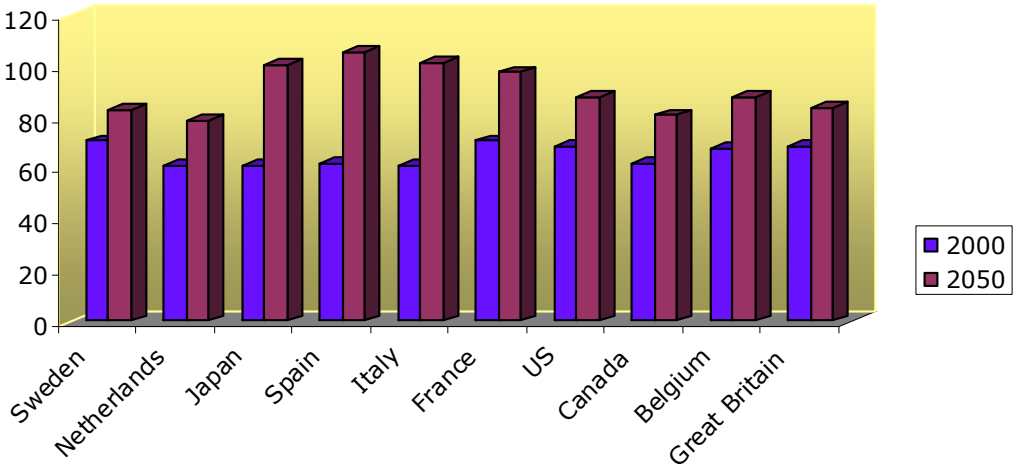
- **In 1871 Thiele** develops the first complex model that attempts to accurately model m*(x)* over the whole life span.

1.4. Financial implications of the Ageing Population

Population in developed countries is aging rapidly. Both the number and proportion of people aged 65 years and older are increasing, although at different rates in different parts of the world. Figure 1 shows that the age-dependency ratio[3] will increase by around ¼ over to 2050, this change in the age-dependency ratio will be the result of the population trend. For example, Europe has the highest proportion of elderly and will probably remain the oldest region for decades, while Japan is the only non-European country with a similar ageing population as the European countries. As part of the same process, the older population itself will age, with large increases in the number of people aged 85 and older. As a consequence, the number of oldest-old persons in developed countries is increasing at a much faster rate than any other age group. This also means that the proportion of the population that is very old will rise, which will place increased financial pressure on the social security system.

[3] Age-dependency ratios are a measure of the age structure of the population. They relate the number of individuals that are likely to be "dependent" on the support of others for their daily living – youths and the elderly – to the number of those individuals who are capable of providing such support. The key indicator of age dependency used relates the number of individuals aged less than 20 and of those aged 65 and over to the population aged 20 to 64.

Figure1: The age-dependency ratio 2000-2050



Source: OECD Demographic and Labour Force database

Under pay-as-you-go[4] social security systems most developed countries have made promises they can't keep, as in their current forms they are not financially sustainable, while some already face financial crises. For example, member countries of the Organization for Economic Cooperation and Development[5] are projected to experience a roughly 50% increase in the share of GDP that is devoted to old age pension expenditures over the next fifty years, from 7.4% of GDP to 10.8% of GDP (Thai-Thanh, Pablo & Howard 2001).

It has been commonly assumed that the aging population is the main cause of the financial constraints for current, as well as future, old age pension schemes. The number of older persons is increasing very rapidly relative to the number of younger persons, a trend which will continue as was shown earlier in Figure 1. As a result, the proportion of retirees has

[4] PAYG systems collect contributions on workers through a tax bearing on labour earnings and share the collected amount among the retirees in function of their past contributions according to some fixed benefit rule (for instance, it is shared in proportion to individuals' contribution)(Gabrielle Demange (2006))

[5] The OECD member countries are: Australia, Austria, Belgium, Canada, Czech Republic, Denmark, Finland, France, Germany, Greece, Hungary, Iceland, Ireland, Italy, Japan, Korea, Luxembourg, Mexico, Netherlands, New Zealand, Norway, Poland, Portugal, Slovak Republic, Spain, Sweden, Switzerland, Turkey, United Kingdom, United States.

increased relative to the number of employed persons who must pay for the benefits of those who are retired. In addition, persons are living longer so that those who reach retirement age are receiving benefits longer than they used to.

1.5. Structure of the PhD Research Report (tesina)

The tesina includes five chapters, starting with an introduction to the topic (Chapter 1). This is followed by a theoretical background on old age mortality (Chapter 2) that reviews concepts and modern theories of mortality. I then address strictly empirical considerations related to the estimation of old age mortality (Chapter 3). In Chapter 4 I try to incorporate Biodemography into the modelling of old age mortality and the final part intents to synthesize the earlier chapters and discuss its usefulness for the proposed thesis (Chapter 5).

"La vida no es sino una sucesión de transiciones. En ellas se expresan los procesos profundos de cambio que van conduciendo de un estadio de existencia a otro. Lo mismo se aplica a las personas. En ambos casos lo único que no cambia, es el cambio mismo"

Julio Frenk[6]

CHAPTER 2 THEORETICAL BACKGROUND TO OLD AGE MORTALITY

2.1. Introduction

It is a fact of life that some people die in childhood, some during their adult years, and others in old age. Obviously, age at death is variable in all human populations, although the extent of this variability changes over time and differs across societies, and does so for a variety of reasons. Today in the more developed countries (e.g. Japan, France and Switzerland) life expectancy at birth continues to increase by three months every year although infant mortality does not exceed five per 1000 and which cannot decline much further. Instead, this increase of life expectancy is caused by adult mortality, in particular old age mortality. Mortality of old and even very old people is falling drastically, and paradoxically, this fall in itself has not been studied greatly beyond statistical descriptions, and little is still known of its causes and mechanisms (Catalano 2002; Myers 1996; Manton & Vaupel 1995; Wilmoth 1995; Kannisto et al. 1994; Vaupel & Lundstrom 1994; Horiuchi 1991; Kannisto 1988).

In this part I examine the decline in old people's mortality by cause of death within the context of the epidemiological transition theory, the variability of mortality in terms of the distribution of ages at death (rectangularization of survival curve and the compression of mortality) and

[6] Julio Frenk (1997): "Transiciones: vidas, instituciones, ideas". Conferencia Magistral "José Luis Bobadilla" dictada en el VII Congreso Nacional de Investigación en Salud Pública, conmemorativo del X Aniversario del Instituto Nacional de Salud Pública y del LXXV Aniversario de la Escuela de Salud Pública de México; 1997 marzo 4; Cuernavaca, Morelos.
"The life is not but one sucesion of transitions. In them the deep processes of change are expressed that are leading of an existence stage to another one. The same it is applied to the people. In both cases the nico that it does not change, is the same change"

the modern theories of mortality (evolutionary theory of senescence, reliability theory of ageing, and biodemography).

Several interesting questions are related to this part. For example, Why did the recent transition in mortality took place? How long can it continue and what factors might contribute to future gains in life expectancy? How has the variability of age at death changed historically? What relationships, if any, exist between the level and the variability of human mortality? Can we assume that sometime in the future everyone has the potential to die from senescence related mortality? If so, are biological limits in the variability of human mortality related to limits in its level?

2.2. The Epidemiological Transition Theory

The impressive increase in length of life has been accompanied by substantial changes in the age at death and cause of death patterns. These shifts in age and cause–specific mortality are described by the Epidemiological Transition Theory that establishes a general framework of the description and interpretation of changes in the patterns of mortality and health during the decline of mortality, and its determinants and consequences (Omran 1971).

According to **Omran** (1971), the epidemiological transition is a shift from the predominance of infectious diseases to degenerative disease and coincides with the mortality component of the (first) Demographic Transition Theory[7]. During the epidemiological transition the most profound changes in health and disease patterns occur among children and young women. Omran distinguished three stages:

[7] The demographic transition theory explains how changes in mortality and fertility influences population growth. The basic demographic transition model describes the process during which slow population growth gives way to a period of rapid population growth, before reverting back to slow growth (Notestein et al., 1944). There is a fifth stage to the demographic transition. When fertility falls to very low levels and stays there for a protracted period, a slow rate of population growth can turn into a negative one. Many countries in Europe now have TFRs below the replacement level of about two children per woman. In the late 1990s, the TFRs of Bulgaria, Italy, Spain, the Czech Republic, Latvia and Russia- all 1.2 – were among the world's (Haupt, A. 2004). Lesthaeghe and Van de Kaa (1986) in their introductory paper to *Bevolking:groei en Krimp* considered for the first time a Second Demographic Transition (or Fifht Stage to the demographic transition.

1. "*The Age of Pestilence and Famine"* lasted until the middle of the 19th century, was characterized by high and fluctuating mortality and its peaks coincided with the years of epidemics, famine, crop failure, disasters and wars. A continuous population growth was impossible. In this stage the average life expectancy at birth was low and variable, oscillating between 20 and 40 years, while infant mortality exceeded 200 deaths per 1000 live births. An early epidemiological study based on the *Bill of Mortality* in 17th century London, reported that 3/4ths of death were attributed to infectious diseases, malnutrition and maternal complications. In contrast, heart diseases and cancer were responsible for less than 6% of deaths (United Nations. World Population Prospects, 1966 Revision. New York, United Nations, 1998).

2. "*The Age of Receding Pandemics"* ended in the middle of the 20th century for most developed countries which was distinguished by the onset of the shift from infectious to chronic diseases. The average life expectancy at birth increased substantially from about 30 to 50 years, while infant mortality declined gradually to below 200/1000 live births. Population growth was sustained and began to describe an exponential curve. Communicable diseases (especially tuberculosis) continued to lead the causes of death. Towards the end of the second stage however, heart diseases, stroke and cancer started to take higher positions. Mortality from tuberculosis began to decline towards the end of this stage and was responsible for part of the decline in general mortality.

3. *'The Age of Degenerative and Man-made Diseases'* is characterized by the stabilization of mortality at a low level. Life expectancy at birth rose gradually from about 50 to 75 years or more, one began to observe a relative ageing of the population. In terms of specific diseases, characteristic of this stage was the increasing prevalence of heart diseases, cerebrovascular accidents (strokes), cancer at various sites, diabetes, chronic obstructive, pulmonary disease and metabolic

disorders, as well as those introduced by man (so-called man-made diseases). These include, for instance, diseases or external cause mortality related to radiation, industrial hazards, traffic accidents. Stress-related diseases such as depression and other mental illness, violence and drug dependency also increased. Although conceding their leading position, infectious diseases and gross malnutrition continued to be important causes of morbidity and mortality.

Omran's third stage marked the end of the epidemiological transition, with an end to the mortality decline. However, shortly afterwards, it was observed that mortality continued to decline throughout the 1970s in developed countries and for women this decline actually never completely stopped. In light of this, Olshansky and Ault (1986) proposed a fourth stage for the epidemiological transition in which mortality fell because the onset of and eventual mortality from chronic diseases was delayed to older ages.

S. J. Olshansky and B. Ault (1986) were the first ones to propose a fourth stage after observing an uninterrupted increase in life expectancy. They thought this trend was significant enough to distinguish it from Omran's three previous stages. This fourth stage is referred to as the "*age of delayed degenerative diseases*", since the probability of death from these causes is shifted toward older ages, and it is summarized by three general characteristics:

1. Rapidly declining death rates that are concentrated mostly in advanced ages and which occur at nearly the same pace for males and females.

2. The age pattern of mortality by cause remains largely the same as in the third stage, but the age distribution of deaths for degenerative causes is shifted progressively toward older ages.

3. Relatively rapid movements in survival are concentrated among the population in advanced ages.

Rogers and Hackenberg (1987) also put forward a fourth stage of the epidemiological transition. They agree with Olshansky and Ault that the major causes of death are still due to degenerative and man-made diseases, each is becoming increasingly influenced by individual behaviours and new life-styles, influences not concretely addressed in the present theory. The point they stress the most is the fact that Omran did not include violent deaths in his theory, or deaths due to social pathologies (such as accidents, suicides and homicides).

In an article published in 1998, **Omran** recognizes the existence of one and possibly two additional stages to his initial epidemiological transition theory. According to him, the fourth stage that he called the "*age of declining cardiovascular mortality, ageing, lifestyles modification, emerging and resurgent diseases*", is characterized by an ongoing rise in life expectancy until it reaches 80 to 85 years or longer, especially for females; by a stabilization followed by a decrease of cardiovascular diseases as a cause of death; as well as by the emergence of new diseases (HIV, Hepatitis B and C, Ebola, Lyme disease, Hantaan virus, New forms of E.Coli, etc.) and by the revival of former diseases (Cholera, Malaria, Dengue, Diphtheria, Tuberculosis, Plague and Chagas disease). The Fifth stage, the "*Age of aspired quality of life with paradoxical longevity and persistent inequities*" for the mid-21st century and beyond, is expected to be one of great human achievements in disease control, health promotion, and further prolongation of healthy life. Inevitably this stage will include, paradoxically, longevity and the emergence of new morbidity and persistent inadequacies. There will be disparities between people because of the polarization of socio-economic status within and between countries (Omran 1998).

Robine (2001b) is even more austere than the previous researchers on his critique of the epidemiological transition theory. After studying the trend in

the dispersion of life spans Robine doubted of the existence of the last two stages of the theory (Omran's third stage and Olshansky and Ault's fourth stage) and he provided support for the existence of only three stages:

1. The mythical reference era that precedes the fall in mortality, i.e. Omran's "*Age of Pestilence and Famine*", that came to an end between the 18th and 19th centuries depending on the country.

2. A first stage of transition, when the level of mortality fell and tended to stabilize as a consequence of the decline in infectious diseases, in particular those affecting women and children, resulting in a very large reduction in the disparities of life spans. This "*Age of Receding Pandemics*" came to an end in the 1950s in the countries which had gone furthest in the transition, such as Northern and Western Europe, North America and Japan.

3. A subsequent transition in which the mortality decline at adult ages, including among the very old, becomes relatively larger than at young ages and where the increase in life expectancy is no longer associated with a reduction in the dispersion of life spans - or with only a very small reduction. This new age corresponds less to Omran's third stage - which today appears to have little basis in reality - and more to the fourth stage proposed by Olshansky and Ault. Robine labelled this new stage as the *"Age of the Conquest of the Extent of Life"*. This may be the age when man, having been finally liberated from the great epidemics, is able to explore the full extent of his life.

Robine says that there is no doubt that this stage will come to an end one day due to the start of a new stage that he labelled as the *"Age of Limits"*, even though at present man is making unexpected discoveries in its exploration of the potential of human longevity, whereby some consider that it may be possible to live for between 110 and 120 years (Robine 2001b).

As for **Meslé and Vallin** (2002), they revised the Epidemiological theory into two stages only. They consider Omran's first three stages, during which the improvement in survival is mostly due to the collapse of infectious diseases and the rise of chronic diseases, as just one stage. The end of this first stage would lead directly to the decline of cardiovascular diseases, which would be the main factor underlying the growth of life expectancy during the second stage. They refer to this stage as the "*cardiovascular revolution*".

Most of the declines in mortality and gains in life expectancy during this recent mortality transition have been achieved in the elderly population- a phenomenon so unexpected and unexplained that it has been referred to as a new stage in the epidemiologic history of developed nations. Consequently, this emergence of ninety-year old and centenarian populations throws a new doubt on the now traditional theory of epidemiological transition and its explanations. A theory that explains how we reached the current stage still remains to be found, with age-specific mortality trends, the emergence of the oldest-old, factors determining their states of health, in particular their functional state, and the causes of their death. Thus, a theory that can be used to improve forecasts for the future still remains to be found. *Where do the current transitions lead us?*

2.3. Variability of mortality in terms of the distribution of ages at death

Biologists were the first to become interested in the shapes and transformation of the survival curve, long before demographers showed any concern (Comfort (1956, 1964); Deevey 1947; Pearl & Miner 1935; Greenwood 1928; Pearl & Doering 1923). At this time demographers were undoubtedly more interested in fertility and in the fall of infant mortality than in life durations and population ageing. In 1964 Comfort introduced the concept of the rectangularization of the survival curve, and the concept was subsequently discussed by others (Gordon 1980; Vaupel et al. 1979;

Strehler 1975). In 1980 James Fries popularized the concept by publishing his theory on the compression of morbidity in the *New England Journal of Medicine*. Drawing on the work of the biologist Comfort (1964), Fries observed that the historical increase in life expectancy was accompanied by a gradual "rectangularization" of the survival curve. He argued that this change was responsible for a shift to an "ideal" survival curve, highly rectangularized, corresponding to a life expectancy at birth of 85 years and with a high concentration of individual life spans around this average value: 66% of life spans would be between 81 and 89 years and 95% between 77 and 93 years. Fries justified this prediction by the existence of a fixed biological maximum limiting the human life span to roughly 100 years and the average life span to 85 years. After that, the rectangularization of the survival curve appeared in the heated debates followed for demographers (Fries (2002, 1989, 1984); Olshansky et al. 1991; Rothenberg et al. 1991; Myers & Manton 1984; Schneider & Brody 1983).

This part of the research analyzes the change of the variability in the age at death and in the life expectancy to evaluate the rectangularization of survival curve. Thus, there is a review of different discourses about whether mortality is becoming more or less compressed at present, if there is or not a limit of human life span and when rectangularization has or has not occurred.

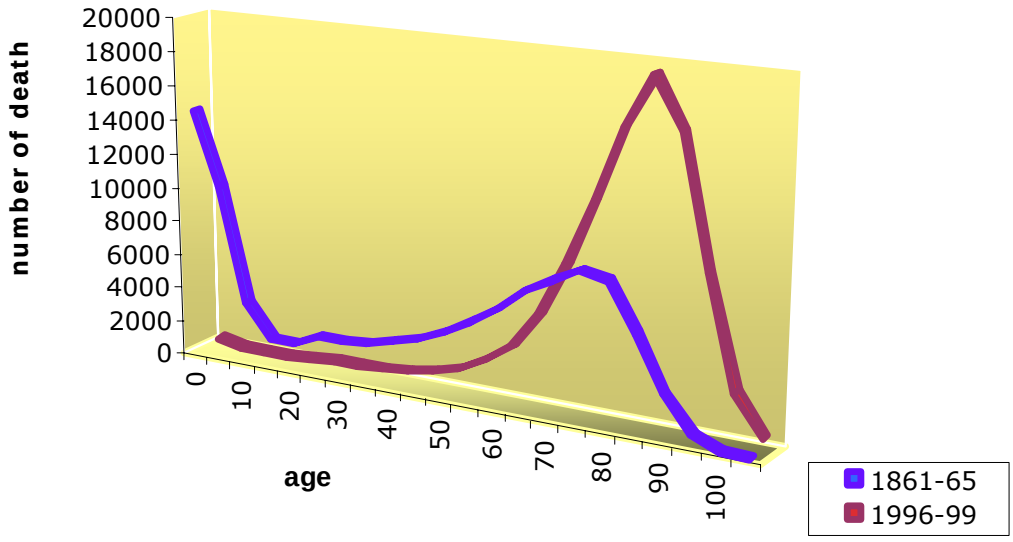
2.3.1. Compression of mortality

Since 1980 researchers have questioned whether mortality compression is occurring (e.g., Fries 1980, 1984), where compression generally refers to the diminishing of the variance in the distribution of age at death over time. This question is now more than 20 years old, and evidence exists both for and against compression (Lynch & Brown 2001; Kannisto 2000b; Nusselder & Mackenbach 1996; Manton & Singer 1994; Olshansky &

Carnes 1994; Carnes & Olshansky 1993 Manton & Tolley 1991; Rothenberg, Lentzner & Parker 1991;Olshansky, Carnes & Cassel 1990).

Regarding the history of mortality compression, there is existing evidence sufficient for proving that the transition from high to low mortality- and, thereby, to modern society- has been accompanied by general and massive compression of the mortality to ever narrower age intervals.

Figure 2: Distribution of deaths by age. Sweden male 1861-65 and 1991-95



Source: Data available through the Berkeley Mortality Data Base.
http:// demog.berkeley.edu

Figure 2 shows the distribution of the age at death of Swedish males before and after the mortality transition and we can see that the compression of mortality actually includes two separate processes. On one hand, the lives saved through the progressive elimination of premature deaths have been redistributed to older ages and this has been the motor of the compression of mortality throughout most of the transition. On the other hand, as people survive to ever higher ages, they enter an area where the lengthening of life meets increasing resistance, demonstrated by the fact that the decline in old age mortality in recent decades has been very much faster near age 80 than 100 (Kannisto 1996).

The decline of human mortality during the industrial era is typically described in terms of increased life expectancy or decreased risks of death across the age range. In contrast, the decreased variability of age at death has received relatively little attention. Similarly, although some studies have been conducted on social and behavioural effects of the historical mortality decline (e.g. several articles in United Nations 1986), little research has focused on the consequences of reduced variability in age at death. Indeed, most analyses of the compression of mortality have been motivated by questions about biological limits to the human life span, with little discussion of the broader historical significance of this phenomenon.

Finally, long-term changes in the variability of age at death may have had significant effects on perceptions, attitudes, and behaviours of individuals and, in turn, on society in general. *What should we expect about future trends in the variability of age at death in low mortality populations?* If the current decline in mortality ceases, the distribution of ages at death (including levels of variability) will stabilize. Yet if mortality rates continue to decline rapidly at both younger and older adult ages, we should expect a continuing pattern of stability in the variability of ages at death, as the entire distribution shifts upward. These broad consequences of the historical compression of mortality merit a more fully detailed analysis.

2.3.2. Limit of human life span[8]

Estimates of limits to human life span (defined as the "biologically" maximum length of life) and of population life expectancy are made by demographers, biologist and actuaries. Despite the significance of a life expectancy limit for private and public pension programs and for private and public short and long-term health insurance, there is little consensus

[8] One of the earliest suggestions that the human life span is fixed appears in the Old Testament: " My spirit hill not abide in man forever, for he is mortal; his days will be a hundred and twenty years" (Genesis 6:3)

on the value at which the limit is set (or if a limit should be considered at all).

Perspectives on the issue can be roughly divided into three groups:

1. The *"traditionalists":* they suggest that the limit is not significantly greater than current life expectancy, namely about 85 years (Fries 1990; Olshansky, Carnes & Cassel 1990 ;).The limit is viewed as being due to biological senescence, which is not affected by the changing patterns in mortality by specific causes.

2. The *"visionary"*: they argue that, while life expectancy limits are due to senescence, advances in biomedical research will raise those limits in the future (in some 25 to 50 years). Since senescence itself is thereby modified, life expectancies of 100 to 125 years (Strehler 1975) or more 150 to 200 years, (Walford 1985; Rosenberg et al. 1973) might be achievable. It is suggested that altering senescence has implications for age-dependent diseases (Strehler 1975).

3. The *"empiricist"*: they contend that we are not currently near a life expectancy limit, because mortality is declining and progress is being made in the treatment and management of the chronic diseases and disabilities that dominate mortality at later ages. It is observed that, if recent mortality declines of about 2 percent per year are continued, life expectancies of 95 to 100 years could be achieved by the year 2080 (Ahlburg & Vaupel 1990; Schneider & Guralnik 1990; Guralnik, Yanagishita & Schneider 1988; Schneider & Guralnik 1987).

In spite of many attempts, an absolute limit to human life has never been conclusively proven, and recent extensive studies of the very oldest have not found evidence of any limit (Vaupel 2004; Thatcher et al.1998). Nevertheless, as the probability of dying keeps rising in modern populations to at least the age of 110, it reduces the dispersion

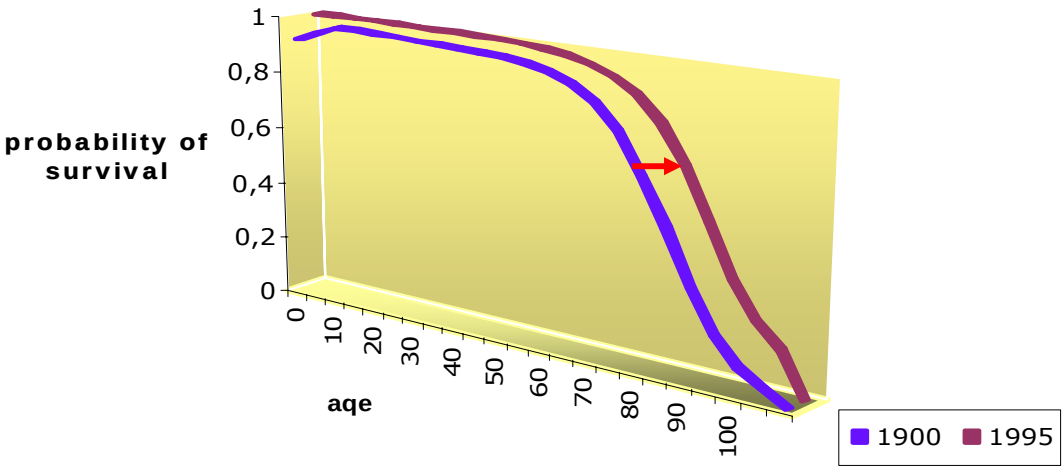
progressively. In the words of Keyfitz (Keyfitz 1978), there is "an uphill road ahead" for extending human life.

2.3.3. Rectangularization of human survival curve

Rectangularization is defined as a trend towards a more rectangular shape of the survival curve due an increase of survivals to advanced ages, an upper boundary to human life expectancy determined by fixed genetic limits and to an increase of the concentration of deaths around the mean age at death of the population (Nusselder 1998). That is, the variability in the age at death declines and deaths are being compressed into the upper years of life.

This rectangularity of the survival curve is visible in Figure 3, which shows changes in the survival curve for the population of the Sweden in 1900 and 1995 and illustrates how the survival curve has become more rectangular as more individuals survive through childhood and young adulthood, but then die off rapidly in old age.

Figure 3: Survival curve for the total population of the Sweden 1900 and 1995



Source: Data available through the Berkeley Mortality DataBase. http:// demog.berkeley.edu

More than 20 years after Fries's (1980) reintroduction of the concept, there appears to be no consensus on a definition of this phenomenon and of how to assess it in a continued debate on the rectangularitzation of the survival curve (Martel & Bourbeau 2003; Fries (2002,1989,1984); Cheung 2001; Yashin et al. 2001; Coale 1996; Manton & Stallard 1996; Eakin & Witten 1995; Manton & Singer 1994; Manton & Tolley 1991; Olshansky et al. 1991; Rothenberg et al. 1991; Myers & Manton 1984; Schneider & Brody 1983). Many demographers who have studied the transformation of survival curves in different countries have found evidence of rectangularization during the epidemiological transition and they agree with Fries that rectangularization has been an important characteristic of historical mortality change in humans, although sometimes they observe that rectangularization has become slower in recent decades (Martel & Bourbeau 2003; Cheung 2001; Robine 2001a; Wilmoth & Horiuchi 1999; Paccaud et al. 1998; Nagnur 1997, 1986; Pelletier et al. 1997 Levy 1996; Manton & Stallard 1996; Nusselder & Mackenbach 1996; Eakin & Witten 1995; Go et al. 1995; Hill 1993; Rothenberg et al. 1991).

Others suggest that the concept of rectangularization has been poorly defined and subjectively judged in visual terms on occasion. For example, Manton & Tolley (1991) proposed distinguishing between "hard" and "soft" rectangularization to distinguish the degree of rectangularization. Coale (1996) described the survival curve in low-mortality populations as being nearly flat from age 5 to 55, then starting to fall, later becoming steeper, and even following a vertical fall as an innate maximum age is approached. And for others rectangularitzation of the survival curve is “a myth” (Myers & Manton (1984)) or an “ill-posed question” (Manton & Tolley 1991), or even that mortality is now undergoing an expansion, rather than a compression, at the oldest ages (Rothenberg, Lentzner & Parker 1991).

The literature also shows little agreement about how to measure rectangularity and variability. Fries and other early researchers depended essentially on visual inspection. Since that time, several different measures

have been used (Nusselder & Mackenbach 1996; Eakin & Witten 1995; Myers & Manton 1984), but no systematic effort has been made to list and compare the various possible measures.

2.4. Biodemographic theories of mortality

Research on human mortality, particularly in the field of ageing, has become an important part of biodemographic studies because of a growing interest in the scientific explanations of ageing and in the search for a general theory that can explain what ageing is and why and how it happens (Carey & Judge 2001).

In particular, many researchers consider that a comprehensive biodemographic theory of species ageing and longevity should provide answers to a number of important questions (Gavrilov & Gavrilova 2001) that include:

- Why do most biological species deteriorate with age (i.e., die more often as they grow older) while some primitive organisms do not demonstrate such a clear age dependence for mortality increase? (Haranghy & Balázs 1980; Finch 1990; Martinez1998).

- Specifically, why do mortality rates increase exponentially (Gompertz law) with age in many biological species? (Strehler 1978; Finch 1990) How should we handle cases when the Gompertzian mortality law is not applicable? (reviews in Strehler 1978).

- Why does the age-related increase in mortality rates disappear at older ages? Why do mortality rates eventually decelerate when compared to predictions of the Gompertz law, occasionally demonstrate leveling-off (late-life mortality plateau), or may even show a paradoxical decrease at extreme ages? (Gavrilov & Gavrilova 2002b).

- How do we explain the so-called compensation law of mortality? (Gavrilov & Gavrilova 1991). The Compensation law of mortality (late-life mortality convergence) states that the relative differences in death rates between different populations of the same biological species are decreasing with age, because the higher initial death rates are compensated by lower pace of their increase with age.

According to Gavrilov & Gavrilova (2001), the search for a general biological theory to explain such questions on ageing and longevity has been made mainly in terms of two theories:

1.-Evolutionary Theory of Ageing: Important advocates and contributors to evolutionary theory of ageing include: Le Bourg (1998,2001); Keller & Genoud (1999,1997); Kirkwood (1999, 1993); Charlesworth 1994;Carnes & Olshansky 1993; Rose 1991; Hamilton 1966; Williams 1957; Medawar (1951, 1946); Weismann 1882.

2.-Reliability Theory of Ageing: The most important contributions to reliability theory of ageing are coming from Lloyd & Lipow 1962; Barlow & Proshan 1975; Barlow, Proschan & Hunter 1965, Kaufmann *et al*. 1977; Crowder *et al*. 1991; Aven & Jensen 1999; Rigdon & Basu 2000. This theory allows researchers to understand many puzzling features of mortality and lifespan (Bains 2000; Gavrilov & Gavrilova (2001, 1993, 1991); Gavrilov (1987, 1978); Doubal 1982; Abernethy 1979; Gavrilov *et al*. 1978).

The purpose of this part is to review the literature within the field of ageing with a balanced scientific discussion of the evolutionary theories of ageing and the relative theory of ageing.

2.4.1. Evolutionary theory of ageing[9]

According to Weismann (1882), Medawar (1951) and Williams (1957) the classic evolutionary theory of ageing seeks to explain why mortality rises with age as health and function decline. In this theory, as individual age, their continued survival contributes less and less to reproductive fitness, because less of their lifetime fertility remains. Consequently, natural selection acts more weakly to reduce mortality at older ages. Although this theory has been extended and qualified, it is still the dominant paradigm for the evolution of senescence (Rose 1991).

The following three major evolutionary theories of ageing are discussed:

1.- The Theory of Programmed Death.

This theory was suggested by August Weismann (1882), one of the first biologists to use evolutionary arguments to explain ageing and one of the forerunners of the evolutionary theory of ageing, argued that *"there is no reason to expect life to be prolonged beyond the reproductive period; so that the end of this period is usually more or less coincident with death*" (quoted in Rose, 1991).

His initial idea was that there exists a specific death-mechanism designed by natural selection to eliminate the old, and therefore worn-out, members of a population (Weismann 1882). The purpose of this programmed death of the old is to clean up the living space and to free up resources for younger generations[10].

[9]Biologists suggest the evolutionary theory of aging as "the intellectual core of gerontology" (Rose 1991). The evolutionary theory of aging may be considered as part of a more general life history theory Stearns 1992). The evolutionary theory of aging is considered to be the intellectual core of the biodemography of aging and longevity (Carnes & Olshansky 1993). The evolutionary theory of aging is a subset of the life history theory (*Le Bourg 2001*).

[10] Weismann probably came to this idea while reading the following notes of Alfred Russell Wallace (one of Darwin's contemporaries and a codiscoverer of natural selection), which he later cited in his essay "The Duration of Life" (1889): *"…when one or more individuals have provided a sufficient number of successors they themselves, as consumers of nourishment in a constantly increasing degree, are an injury to those successors. Natural selection therefore weeds them out, and in many cases favours such races as die almost immediately after they have left successors."*

While discussing Weismann's evolutionary theory of ageing, it is important to know that his scientific views evolved significantly over the course of his life. Later Weismann stopped writing about the "injuriousness" of the old and changed his evolutionary views, considering old organisms not as harmful but simply neutral for the biological species:

"…in regulating duration of life, the advantage to the species, and not to the individual, is alone of any importance. This must be obvious to any one who has once thoroughly thought out the process of natural selection. It is of no importance to the species whether the individual lives longer or shorter, but it is of importance that the individual should be enabled to do its work towards the maintenance of the species.…The unlimited existence of individuals would be a luxury without any corresponding advantage" (Weismann 1892).

But he did not receive as much attention as his famous earlier theory of programmed death. After his death in 1914, his name became forever associated with the earlier, not the later, version of his theory[11].

2.- The Mutation Accumulation Theory of Ageing

This evolutionary theory, suggested by Peter Medawar (1946) considers ageing as a product of natural selection. For example, a mutant gene that kills young children will be strongly selected against (will not be passed to the next generation) while a lethal mutation with effects confined to people over the age of 80 will experience no selection because people with this mutation will have already passed it to their offspring by that age. Over successive generations, late-acting deleterious mutations will accumulate, leading to an increase in mortality rates late in life.

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[11] This can be seen in the 1922 edition of the book The Biology of Death written by Raymond Pearl: "*In his famous address of 1881 on the duration of life, Weisman propounded the thesis that death was an adaptation, advantageous to the race, and had arisen and was preserved by natural selection. Probably no more perverse extension of the theory of natural selection than this was ever made*" (p. 43)

According to this theory, persons loaded with a mutant gene have fewer chances to reproduce if the deleterious effect of this mutation is expressed earlier in life. By contrast, people expressing a mutation at older ages can reproduce before the illness occurs. In other words, the mutation accumulation theory predicts that the frequency of genetic diseases should increase at older ages.

Obviously, the most important conceptual problems with the theory of the accumulation of mutations at old age are that it predicts that diseases are more common at old age than at young age, which is a mere tautology, and that the risk of dying increases with age. However, the fact that mortality rates can decelerate at old ages is a difference with the theory (Pletcher & Curtsinger 1998).

If this theory is considered as the main or unique cause of ageing, it may be ruled out. By contrast, if one considers that it describes one cause of ageing among others, the theory may be valuable. Indeed, some features of ageing could be due to such alleles with a late expression while other ones could be best explained by different mechanisms. If the mutations at old age explain only a part of the ageing process, "plateaus far below 100%" could be explained (Gavrilov & Gavrilova 2002).

3.- The Antagonistic Pleiotropy Theory of Ageing

The Antagonistic Pleiotropy Theory was proposed by George Williams (1957), who noticed that natural selection may be said to be biased in favour of youth over old age whenever a conflict of interests arises. According to him, this conflict arises from:

"*pleiotropic genes that have opposite effects on fitnesses at different ages.…Selection of a gene that confers an advantage at one age and a disadvantage at another will depend not only on the magnitudes of the effects themselves, but also on the times of the effects. An advantage during the period of maximum reproductive probability would increase the*

total reproductive probability more than a proportionately similar disadvantage later on would decrease it. So natural selection will frequently maximize vigour in youth at the expense of vigour later on and thereby produce a declining vigour (ageing) during adult life" (Williams 1957).

In other words, Williams suggested the existence of so-called pleiotropic genes (demonstrating favourable effects on fitness at young ages and deleterious ones at old age) that could explain the ageing process. Such genes will be maintained in the population due to their positive effect on reproduction at young ages despite their negative effects at old post-reproductive age (their negative effects in later life will look exactly like the ageing process) (Gavrilov & Gavrilova 2002).

Another prediction of the trade-offs between reproductive capacity and longevity was made by George Williams in the following way: "*Successful selection for increased longevity should result in decreased vigour in youth*" (Williams 1957).

Since the 1960s, no fundamentally new evolutionary theories of ageing have been proposed. There were, however, attempts to find a better name for the antagonistic pleiotropy theory and to specify in more detail how one and the same gene could have both deleterious and beneficial effects. In particular, **the disposable soma theory** was proposed (Kirkwood & Holliday1979; Kirkwood 1977), which postulated a special class of gene mutations with the following antagonistic pleiotropic effects: these hypothetical mutations save energy for reproduction (positive effect) by partially dissabling molecular proofreading and other accuracy promoting devices in somatic cells (negative effect). The authors of the disposable soma theory argued that "*it may be selectively advantageous for higher organisms to adopt an energy saving strategy of reduced accuracy in somatic cells to accelerate development and reproduction, but the consequence will be eventual deterioration and death*" (Kirkwood & Holliday 1979).

When applied at the evolutionary level, the soma theory is not really different from the antagonistic pleiotropy theory. As emphasized by Kirkwood & Rose (1991), the disposable soma theory is a causal subset of the antagonistic pleiotropy theory. In fact, "the difference between the disposable soma theory and the antagonistic pleiotropy is partly a difference between an optimality approach… and a quantitative genetics approach", but the two theories do not differ in their predictions regarding trade-offs between reproduction and longevity.

These evolutionary theories offer a convincing explanation of the ageing process and the question now is to know whether they may be considered as validated or not. During the last twenty years, some gerontologists have tried to confirm or invalidate evolutionary theories of ageing[12]. We may wonder whether it is useful to spend the next twenty years to test them again, since it is clear that they explain, at least partly, the ageing process. These theories probably still need to be refined. New tests of them are surely useful. However, testing them is probably no longer a top priority. This priority could be to find new means to modulate the ageing process in animal models (worms, flies, rodents or primates), using genetic or environmental manipulations, to finally improve everyday life of elderly (Le Bourg 2001).

2.4.2. Reliability Theory of Ageing

It looks like the evolutionary theory is more appropriate to explain early successes of biological species (e.g., reproductive success), rather than their later failures (ageing and death). Thus, there seems to be a missing piece in the theoretical body of evolutionary biologists trying to explain

[12] In 1989, Rose and Graves wrote, "*if other biologists could accept the sufficiency of the evolutionary theory as the general theory of ageing, then there might be a relaxation of efforts to find general physiological theories of ageing*". Keller and Genoud (1997) showed "*that evolutionary theories of ageing are consistent with the high longevities of queens*". In 2001, Le Bourg E conclude saying, "*To be perfectly clear, the evolutionary theories give a plausible explanation of the aging process, but in many occasions they do not fit to the results: since better theories are not available, we could accept them provisionally*".

ageing, and this missing piece is about the general theory of system failures known as the theory of reliability.

The Reliability Theory of Ageing is a body of ideas, mathematical models, and methods directed to predict, estimate, understand, and optimize the lifespan distribution of systems and their components (Barlow & Proschan 1965).

Reliability theory explains why most biological species deteriorate with age (i.e., die more often as they grow older) while some primitive organisms do not demonstrate such a clear age dependence for mortality increase. The theory predicts that even those systems that are entirely composed of non-ageing elements (with a constant failure rate) will nevertheless deteriorate with age, if these systems are *redundant* in irreplaceable elements. Ageing, therefore, is a direct consequence of systems redundancy. The 'apparent ageing rate' (the relative rate of age-related mortality acceleration corresponding to parameter in the Gompertz[13] law) increases, according to reliability theory, with higher redundancy levels. Therefore, a negligible 'apparent ageing rate' in primitive organisms (Martinez 1998; Finch 1990; Haranghy & Balázs 1980) with little redundancy is a predicted observation for reliability theory (Gavrilov & Gavrilova 2002).

The reliability theory explains why mortality rates increase exponentially with age in many adult species (Gompertz law) by taking into account the initial flaws (defects) in newly formed systems. It also explains why organisms 'prefer' to die according to the Gompertz law, while technical

[13] Gompertz (1825) recognised that an exponential pattern in age captured the behaviour of human mortality for large portions of the life table. He proposed the function: $\mu_x = A\, c^x$
Where *A* and *c* are constants. The left hand equation (1) is simply the definition of the force of mortality (μ_x,) as the instantaneous rate of mortality at age x. The right hand equation(1) is the actual 'law'. The constant A reflects the general level of mortality in the population concerned. The constant c (the Gompertz constant) reflects the rate at which the force of mortality increases with age.

devices usually fail according to the Weibull[14] (power) law. Moreover, the theory provides a sound strategy for handling those cases when the Gompertzian mortality law is not applicable. In this case, the second best choice would be the Weibull law, which is also fundamentally grounded in reliability theory. Theoretical conditions are specified when organisms die according to the Weibull law: organisms should be relatively free of initial flaws and defects (Gavrilov & Gavrilova 2002).

In those cases when none of these two mortality laws is appropriate, reliability theory offers a more general failure law that is applicable to adult and extreme old ages. The Gompertz and the Weibull laws are just special cases of this unifying more general law.

Reliability theory also explains why the age-related increase in mortality rates vanishes at older ages. It predicts the late-life mortality deceleration with subsequent levelling off, as well as the late-life mortality plateaus, as an inevitable consequence of *redundancy exhaustion* at extreme old ages. This is a very general prediction of reliability theory: it holds true for systems built of elements connected in parallel, for hierarchical systems of serial blocks with parallel elements, for highly interconnected networks of elements (Bains 2000), and for systems with avalanche-like random failures (Gavrilov & Gavrilova 1991).

The reliability theory also predicts that the late-life mortality plateaus will be observed at any level of initial damage: for initially ideal systems, for

[14]Weibull proposed a model in 1951 which represents the failure of a technical system due to wear and tear: $\mu(x)=A\,x^{\theta}$
If we consider the distribution of the times till failure of human organs, and suppose that death occurs when the first failure occurs, we have an analogy with mortality. A distinguishing feature of parametric hazard models is the assumption concerning the shape of the risk function. Rather than modelling the hazard function directly, one can model a function of the hazard which may be easier to estimate from empirical data. For example, the probability that an individual alive at age 0 will survive to age x can be written in terms of the hazard function: $_xp = exp\ -\int \mu\,(s)\,ds$ (Higgins T 2003)

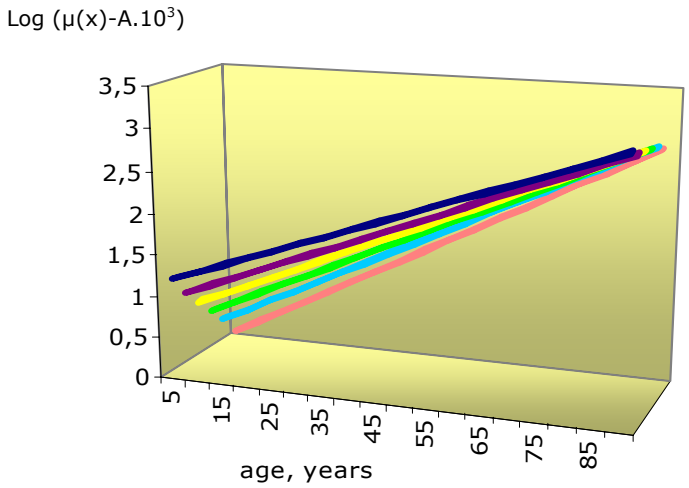
highly redundant systems saturated with defects and for partially damaged redundant systems with an arbitrary number of initial defects.

Furthermore, reliability theory predicts *paradoxical mortality decline in late life* (before eventual levelling-off to mortality plateau) if the system is redundant for *non-identical components* with different failure rates (Barlow & Proschan 1975; Barlow *et al*. 1965). Thus, in those cases when 'apparent rejuvenation' is observed (mortality decline among the oldest-old) there is no need to blame data quality or to postulate initial population heterogeneity and 'second breath' in centenarians. The late-life mortality decline is an inevitable consequence of *age-induced population heterogeneity* expected even among initially identical individuals, redundant in non-identical system components (Gavrilov & Gavrilova 2001). Recently this general explanation was also supported using computer simulations (Bains 2000). Late-life mortality decline was observed in many studies (Khazaeli *et al*. 1995; Carey *et al*. 1992; Barrett 1985) and stimulated interesting debates (Klemera & Doubal 1997; Olshansky 1998) because of the lack of reasonable explanation. Reliability theory predicts that the late-life mortality decline is an expected scenario of systems failure.

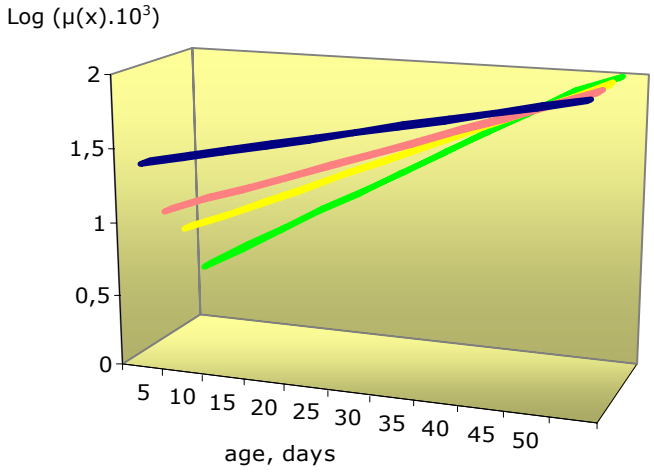
The theory explains the compensation law of mortality, when the relative differences in mortality rates of compared populations (within a given species) decrease with age, and mortality convergence is observed due to the exhaustion of initial differences in redundancy levels (see Figure 4).

Reliability theory also predicts that those experimental interventions that change 'true ageing rate' will also suppress mortality convergence, providing a useful approach on how to search for factors affecting ageing rate.

Figure 4: Compensation law of mortality



Graph 1: an example of fictitious human mortality from different populations.



Graph 2: an example of mortality of different fictitious species.

2.5. Conclusions

Causes of death form an integral part in the dynamic of the epidemiological transition, as each phase is typified by a particular cause-of-death structure. However, the transition from one phase to the net not only

signifies a change in mortality structure, it also implies a change in the prevalence and type of disease determinants.

Findings obtained in this study do not provide straightforward arguments in favour or against the rectangularization of the survival curve in developed countries. On the one hand, several results are compatible with Fries' theory. There is a sustained increase in age at death, with a shift in distribution toward the right hand side (Fig 3). And on the other hand, several results do not fit with any ongoing rectangularization.

The three major evolutionary theories of ageing (The Theory of Programmed Death, The Mutation Accumulation Theory of Ageing, The Antagonistic Pleiotropy Theory of Ageing) offer a convincing explanation of the ageing process but the question now is to know whether they may be considered as validated or not.

The reliability theory has an amazing predictive and explanatory power and requires only a few general and realistic assumptions. It offers a promising approach for developing a comprehensive theory of ageing and longevity that integrates mathematical methods with biological knowledge including cell biology (Abernethy 1998), evolutionary theory (Miller 1989; Charlesworth 1994) and systems repair principles (Rigdon & Basu 2000). I suggest, therefore, adding the reliability theory into the biodemographic methods to study aging and longevity.

"hendiendo las olas con dudoso rumbo, en débiles tablas navega tranquilo, mientras una linde demasiado estrecha separa las sendas de vida y muerte.

…

Ahora los mares se sienten vencidos y aceptan las leyes que dictan los hombres

…

Ya cualquier barquilla recorre el abismo"

Lucio Anneo Séneca[15]

CHAPTER 3 EMPIRICAL CONSIDERATIONS IN THE ESTIMATION OF OLD AGE MORTALITY

3.1. Introduction

In the distant past, the age pattern of mortality of the oldest old (80 and over) was not well-known. Knowledge of mortality among persons approaching the age of 100 (or more), in particular, was mainly based on unreliable statistics and theoretical concepts. However, in recent decades this situation has gradually changed, as efforts have been made to improve past as well as recent mortality statistics and to develop methods of estimating mortality age patterns in cases where data are inaccurate. The reason underlying these new developments is quite simple. Due to a 50 year persistent decline in mortality, the number of persons surviving to very old ages has increased dramatically. As a result, institutions involved in social policies that strive to balance the needs of, and resources for the elderly, have become interested in reliable and detailed population projections.

The objective of this chapter is therefore to describe and comment on the most common forecasting models for old age mortality that have been used in developed countries.

[15] *Second Act of Medea by Lucio Anneo Séneca*
"cleaving the waves with doubtful course, in weak tables it sails calm, while one is contiguous too much narrows separates the footpaths of life and death. … Now the seas feel won and accept the laws that the men dictate… Any log already crosses the abyss"

3.2. Mathematical models of old-age mortality in developed countries

Most discussions about old-age mortality have focused on its trends, levels and age patterns. Two aspects are most often discussed: age patterns and trends in mortality at selected ages over time. It is a fact that age- specific death rates have declined over time, even at ages as high as 107-109 years, the highest ages at the highest ages. However, there is little agreement among researchers with respect to the age patterns of old-age mortality. In this part I review the literature on Gompertz's law of mortality, mortality trajectories with age, modelling of old age mortality by cause of death, and mortality models incorporating theoretical concepts of ageing.

3.2.1. Age pattern of old age mortality: Gompertz's law[16]

Gompertz was an actuary who, like his contemporaries, was interested in the practical problem of estimating premiums for life annuities. But what separated Gompertz from the other actuaries of his time was that he saw the life table as more than just a working tool. He tried to go beyond the simple mathematics of insurance tables in an effort to understand why there were consistent age patterns of death among people. His motivation is perhaps best exemplified in a statement he made near the end of his career: *"The object of research is not only to give information of facts, but to draw beneficial and general views; and if generalisations lead to probable theories, they should be regarded as pleasing associates, to be entertained at the feast of knowledge ..."* (Gompertz (1872:330)).

Here I review the literature on Gompertz's law of mortality and discuss the importance of his observations. The literature related to the Gompertz equation and his proposed law of mortality is extensive so I will mainly focus on an article by Olshansky & Carnes (1997) that I believe best

[16] See Annex I and II for the main parameterization functions for mortality.

describes the important conceptual and methodological developments that have occurred since Gompertz's initial observations over 170 years ago.

Gompertz published only three articles. In the first one, published in 1820, he identified a consistent rate of increase in death rates for some nineteenth century European populations for a limited portion of the age range. In his second one, published in the Transactions of the Royal Society (1825), Gompertz set what is now recognized as the law of mortality. After observing similar patterns of geometrical progression in other tables of mortality, Gompertz believed he had discovered a general law of mortality that linked arithmetic increases in age with geometric increases in death rates. He translated his finding about the law of geometrical progression into the conclusion that "*if the law of mortality were accurately such that after a certain age the number of living corresponding to ages increasing in arithmetical progression, decreased in geometrical progression, it would follow that life annuities, for all ages beyond that period, were of equal value ...*" (Gompertz (1825:515). From 1825 to 1862 Gompertz was *"engaged on the subject of what is called Vital Statistics"* (Gompertz 1872:330) and published a paper in 1862 that focused primarily on revising his original notation. Gompertz presented a fourth article to the International Congress in 1860, but it was published after his death in the Journal of the Institute of Actuaries by his colleague, Dr.William Makeham (see Gompertz 1872). He noted that in his primary equation for geometric progression the parameters of his equation "*were supposed to represent constant quantities, or at least were shown to differ very little from constants, for a very long term of years, for instance, about 50 years…But in making the investigation, I did not pretend that [the parameters] were absolutely constant; were determined by a random selection from three distant periods of age, from a statement of the number of persons who will be living at different ages, out of a certain number of persons stated to have been born. And therefore as* $L_x = A \cdot B^{qx}$ *will not perfectly, during the whole term of life, express the facts ...*" (Gompertz 1872).

Olshansky & Carnes (1997) noted that Gompertz (1825:516) was somewhat vague on the issue of an age beyond which humans were incapable of living. At one point, he emphatically stated: *"though the limit to the possible duration of life is a subject not likely ever to be determined, even should it exist, still it appears interesting to dwell on a consequence which would follow, should the mortality of old age be as described above. The non-appearance on the page of history of a single circumstance of a person having arrived at a certain limited age, would not be the least proof of a limit of the age of man; and further, that neither profane history nor modern experience could contradict the possibility of the great age of the patriarchs of the scripture."* Later he qualified his position on a limit to life by stating: *"Such a law of mortality would indeed make it appear that there was no positive limit to a person's age; but it would be easy, even in the case of the hypothesis, to show that a very limited age might be assumed to which it would be extremely improbable that any one should have been known to attain".*

Moreover, as Olshansky & Carnes (1997) mention, Gompertz himself recognized that his original equation did not apply to the entire age range. In fact, Gompertz (1872) suggested in his last paper that there are four distinct periods in the life span between which separate laws of mortality apply: birth to 12 months, 12 months to 20 years, 20 years to 60 years, and 60 years to 100 years. His equation was intended to apply only to a limited age range for humans-between the ages of 20 and 60. Even within this age range he recognized that his formula worked best "*provided the intervals be not greater than certain limits*" (Gompertz (1825:514) quoted in Olshansky & Carnes (1997)). After that there has been a repeated historical recognition that a single Gompertz equation does not adequately describe mortality for humans over the entire life span. For example, Makeham (1867:346 in Olshansky & Carnes (1997) argued that even after he used his "partial forces of mortality" to characterize mortality schedules, the speed of the increase in the death rate decelerated beyond age 75. Similarly, Brownlee (1919:47 in Olshansky & Carnes (1997) suggested that the Gompertz equation does not apply equally throughout the age range;

"*the graduation is made in two sections, one section from the age of 15 to the age of 50, the second beginning at the latter age and extending upwards to the end of life*." In Perks' (1932:15 in Olshansky & Carnes, (1997)) development of the logistic equation to improve the graduation of death rates at older ages, he recognized that "*the ungraduated rates and the rates by adopted graduation show a curious peak in the rate of increase in qx round about age 80 ...it is thought that the sharpness of this peak may be due in some way to an element of neglected selection which would naturally rapidly wear itself away at about age 80 ...*". He further stated that "*the graduated curve [of mortality] starts to decline in the neighbourhood of age 84*" (p. 30). In Beard's (1959:303 in Olshansky & Carnes (1997)) discussion of mathematical mortality models, he recognized that "*what evidence is available tends to support the idea that the force of mortality does not continue to increase indefinitely with age*". Strehler & Mildvan (1960:311 in Olshansky & Carnes (1997)) argued that one of the four distinct phases of the human mortality curve was "*a period of departure from the Gompertzian relationship at great ages so that mortality rises more slowly than anticipated after age 85-90.*" He argued that this phenomenon would occur "*when the vitality has decreased to the point where it is similar to the average energy of fluctuations*" (p. 314) about a homeostatic mean. One of Strehler & Mildvan's (1960:217 in Olshansky & Carnes (1997)) "*observations that any mathematical theory of mortality must incorporate into its postulatory structure, explain, or at least not violate*" included the fact that "*at extremely advanced age, the mortality rate curves of several species rise at a rate progressively lower than exponential*" (p. 224). The limited applicability of the Gompertz function to only a specified range within the life span and a deceleration in death rates at older ages (including possible explanations for this phenomenon) have been recognized by many other researchers (Olshansky & Carnes 1997)[17].

[17] They cite the following references: Abernathy (1979); Brooks, Lithgow & Johnson 1994; Doubal 1982; Economos 1980; Gavrilov & Gavrilova 1991; Horiuchi & Coale 1990; Horiuchi & Wilmoth 1999; Pakin & Hrisanov 1984; Riggs 1993; Weiss 1989; Witten 1988).

Thus the current situation with applicability of the Gompertz law to extreme old ages is still a paradoxical one (Gavrilov and Gavrilova 2002). On the one hand, it has been well known for a long time that the Gompertz law is not applicable to mortality rates at advanced ages- the observed mortality rates are always lower than predicted by the Gompertz model, and not surprinsingly, the actual number of survivors to extreme ages is always higher than predicted by the Gompertz law. The problem now is to find out which of these theories is correct.

3.2.2. Mortality trajectories with age.

It has been known for some time that mortality increases with age, but is there an age at which death in the following year is certainty? And if yes, what is it? What determines the mortality ceiling? Can it drop significantly in the future? Explanations are put forward to explain the mortality trajectories with age.

From 1950, the extinct generation method suggested by Vincent (1951) and its modern extensions (Meslé & Vallin 2000; Thatcher et al 2002) have been used to calculate the mortality rate for the oldest-olds accurately (Robine 2005). Creating a database covering several countries increased the number of observations (Vincent 1951; Dépoid 1973), but these remained limited until the K-Th Database[18] created by Kannisto and Thatcher that includes mortality data from 30 developed countries (Kannisto 1994). This database is the foundation for all modern work on mortality trajectories that suggest that mortality among the oldest old

[18] The recent progress in the demography of longevity is partly due to the constitution of international databases, such as the Oldest-Old Mortality Database, better known as the Kannisto-Thatcher Database (Kannisto, 1994), the Human Mortality Database (HMD), developed jointly by the University of California at Berkeley and the Max Planck Institute for Demographic Research at Rostock (www.mortality.org or www.humanmortality.de) and the International Database on Longevity (IDL), developed jointly by the University of Montpellier, the French National Institute for Demographic Studies (INED) and the Max Planck Institute for Demographic Research (Robine & Vaupel 2002). More of such databases in the fields of gerontology, epidemiology, biodemography and biology would surely contribute to significant progress.

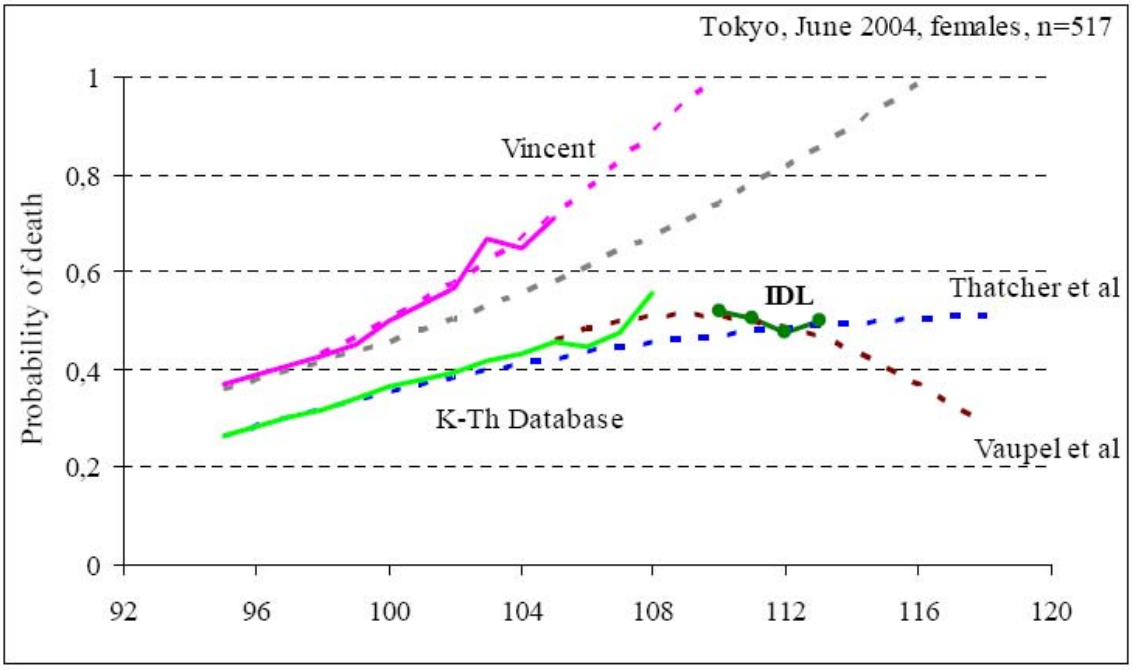
follow a logistic trajectory (Kannisto 1996; Thatcher et al. 1998; Thatcher 1999) or a quadratic trajectory (Vaupel et al. 1998).

Another database is the International Database on Longevity (IDL), which has objective to accurately calculate mortality rates between the ages 105 and 115 years (Robine & Vaupel 2002). There is no doubt today that mortality slows down after the age of 85 and that mortality rates observed at ages 95 or 100 are lower than expected (Robine 2005). He cites as well-known studies those by Barbi et al. (2003); Kestenbaum & Fergusson (2002) ; Lynch & Brown (2001); Weitz & Fraser (2001) ; Manton (1999); Horiuchi & Wilmoth (1999); Vanfleteren et al. (1998).

Figure 5 summarises the knowledge on old age mortality trajectories with age. Vincent (1951) was able to estimate for the first time the age at which the probability of dying in the same year was close to 110 years. Twenty years later, Dépoid (1973) would find a limit age of 117 years old based on same hypotheses and methods. The logistic trajectory with age of Thatcher et al. (1998) tends towards a mortality ceiling without ever reaching it. Vaupel et al. (1998) found a quadratic trajectory, where mortality diminishes with age, having passed a maximum at around 110 years old. The mortality rates between 110 and 114 years old, calculated extremely accurately thanks to the IDL (Robine & Vaupel 2002) show that mortality does not increase, or only very slightly, after 110 years of age.

These results nevertheless show that human longevity has no limits in terms of age and there are no biologically-controlled limits (clock genes or other mechanisms linked to natural selection). If there really is a limit, it is for mortality levels that do not reach an annual mortality rate of 60%, even in people of 110 years old or more, even for the most frailty, under mortality conditions currently found in the most developed countries.

Figure 5: Mortality trajectory beyond 95 years of age



Source: International Database on Longevity- IDL, 2004

3.2.3. Modelling of old age mortality by cause of death

A first step in improving mortality projections is to analyse cause-of-death patterns, as each disease has its own cause. It has the added advantage that it not only improves the projection of mortality, but also the explanation for mortality differences over time (Spijker 2004:6-7).

The cause-of-death approach is perhaps the most encouraging, particularly for short-term forecasting, because of the simplicity of the parameterisation functions for mortality-by-cause and by the fact that epidemiological knowledge can be used to formulate hypotheses. However, there are certain difficulties associated with this approach. For instance, bias may result from misclassifications of deaths that could result in cross-correlations between mortality trends from different but related causes. A more important implication for my future PhD research, however, is the

fact that the underlying cause of death is less easily identified with regard to deaths at the most advanced ages (Tabeau et al. 2001).

Differences in the outcomes and accuracy of overall mortality and cause of death-based forecasts of total mortality have been recently discussed by Tabeau et al. (2001), who reviewed studies conducted by Wilmoth (1995), Caselli (1996) and Alho (1991), which are briefly summarised below.

Wilmoth (1995) showed analytically for the proportional rate of change models which are linear in parameters that forecasts of morality obtained from the aggregation of mortality by cause must eventually exceed forecasts based on mortality data by all causes taken together. From Caselli's projections (Caselli 1996), another pattern of results has emerged. In general, projections of trends in mortality by cause in all countries reduced the advantage enjoyed by females and increased that for men, for whom the gains in life expectancy were larger when estimated by cause than those obtained from total mortality-based projections. Alho (1991) discussed the effects of aggregation on the estimation of trends in mortality. In some cases, Alho suggests that the aggregate forecasts appear to be more credible. He states that several factors are capable of influencing the potential for disaggregation-related gains in forecast accuracy. The effects of misclassification of deaths by cause, the cross-correlation between causes, the similarity of auto-correlations in different causes, modelling bias, and expert judgement are all examples of such factors. The results of different approaches are not similar if one or more causes serve as 'leading indicators' for the remaining causes, or outside information is incorporated into forecasting either through expert judgement or formal statistical modelling. Also, under highly non-linear models or in the presence of modelling errors the results may not be similar.

A difficulty of the cause-of-death approach is that the underlying cause of death cannot be easily identified for ages after approximately 90 years, which implies that cause-specific projections can only extend up to a

relatively low age. Thus, modelling mortality of the oldest-old cannot take place within the framework of the cause-specific approach (Tabeau et al. 2001). It must be stressed that old-age mortality is the key component of any projection model in the overall period and cohort forecasting approaches. The hypotheses chosen for this component are crucial to the outcome of the forecasts, but their formulation is difficult. The hypotheses can no longer be formulated on the basis of past experience, but should be based on a source other than mortality, such as research on longevity, as explanatory models of mortality are extremely relevant.

3.2.4. Mortality models incorporating theoretical concepts of ageing

Understanding why and how we age requires interdisciplinary efforts, and these include research in actuarial science, demography, gerontology, biology and so forth. There is an accumulated knowledge in different disciplines for the description of the ageing process, and the impact of these characteristics on mortality are represented in this models below.

Simms (1942) found that some important physiological characteristics connected with an individual's 'vitality' are different for individuals of the same age. This finding generated a new way of thinking about mortality as it is estimated in population studies and about biological and physiological characteristics of ageing.

Yashin (2001) reviewed several approaches to modelling the age pattern of mortality that incorporate theoretical concepts of ageing that have contributed to our understanding of the basic regularities of ageing and survival and shows how theories and types of observations influence the structure and scope of applications of mortality models.

A major characteristic of the earlier theories and models of mortality is that they associate the dramatic decline in the vitality of the organism.

However, reviews of physiological studies of ageing carried out by Shock (1960,1974) and later by Bafitis & Sargent (1977) showed that various functions characterising physiological capacities in humans decline more or less linearly with age.

Strehler & Mildvan (1960) combined in their mathematical model the Gompertz curve with the assumption that the death rate at a given age is proportional to "the frequency of stresses which surpass the ability of a subsystem to restore the initial conditions" at that age. They showed that, under such an assumption, the exponential increase in mortality is related to the linear decline in vitality.

Atlan (1968) modified Strehler & Mildvan's theory. He suggested expressing vitality in terms of entropy rather than of energy. According to Atlan, ageing is a process of progressively increasing entropy, meant as a decrease in the information content of the organism, until a level is reached that is incompatible with life. Atlan claimed that the linear portion of the curve corresponds to the exponential variation in the mortality rate (Gompertz curve) and that the subsequent portions correspond to the last part of the mortality curve, which deviates from Gompertz for old ages and moves towards a constant mortality rate.

Sacher & Trucco (1962) suggested a stochastic mechanism of ageing and mortality, in which the individual chances of death are regulated by random fluctuations of internal and external origin. In other words, they suggested a model of mortality that includes both homeostatic forces and random fluctuations of physiological variables. They also assumed a linear decline in the homeostatic capacity with age.

Simms (1948) viewed the stochastic nature of mortality as resulting from biological forces acting randomly within individuals who then become different as they grow older.

Szillard (1959) developed the genetic theory of mortality and ageing. According to his model, "the main reason why some adults live shorter lives and others live longer is the difference in the number of faults they have inherited".

Beard (1959) proposed a model of heterogeneous mortality which results in the logistic curve of the observed hazard. He claimed that the distribution of heterogeneity can be looked upon as an index of the genetic make-up of the population. He then assumed that the distribution of individuals by cause of death reflects this heterogeneity.

Vaupel et al. (1979) conceptualised the idea of population heterogeneity in mortality and introduced the notion of individual frailty as a measure of individual differences in the chances of survival in the proportional hazards model. His model of hidden heterogeneity in survival (frailty) was an important step towards a better understanding of the observed age patterns of mortality. In particular, the idea of heterogeneity was used to explain the deceleration and levelling off of mortality rates at late ages.

Charlesworth (1990) suggested a quantitative genetic model of senescence, which includes the effects of mutation pressure. The model predicts that the additive genetic variance for mortality rates should increase with age, and that, if deleterious mutations are completely age-specific in their effects, then a total collapse of survival would occur at later ages. This means that the mortality rate tends to infinity at a finite age interval.

Abrams & Ludwig (1995) explored the relationship between mortality and age that would hypothetically occur when individuals used the energy and resources normally spent on reproduction on the maintenance and repair of the organism. They predicted a marked deceleration of mortality rates and the death rate still tended to infinity with age.

Mueller & Rose (1996) developed a model for the calculation of mortality trajectories in a hypothetical population using the assumptions of either *antagonistic pleiotropy* or *mutation accumulation* (see Chapter 2 for their respective definitions). They concluded that the standard evolutionary theories explain late-life mortality plateaus.

3.3 Data types, sources and quality.

A frequently mentioned problem in studies dealing with mortality is the unreliability of age reporting at advanced ages. Ages of older persons tend to be misreported, and are more likely to be exaggerated rather than understated. This transfers some persons of relatively younger age in which mortality is lower to older age groups with higher mortality. This leads to the underestimation of mortality rates at old ages and the overestimation of the expectation of life unless the age at death is exaggerated considerably more than the ages of living persons, which would inflate mortality at those older ages that are reported (e.g. 85, 90, 95 etc.). To eliminate these errors, two steps are usually taken (Redfern 1974).

1. To improve the number of death at old ages that enables to reestimate old-age population numbers and deaths. To give an example, several developed countries use such adjusted death and population statistics as those that have been made available by the K-T database. The data from this collection meet the criteria of reliability and consistency for the 30 developed countries that are included, but much still remains to be done to improve the old-age population and mortality statistics of less developed countries.

2. To use mortality measures to solve problems of incomplete data (e.g. Preston, Elo & Stewart 1999).

With regard to the different types of population and vital statistics data sources available, that is, continuous population registers, death certificates and censuses, there are three principal advantages of using data from continuous population registers over data from death certificates and censuses. First, we should consider the validity of age recording. At birth a personal card based on the birth certificate is made and all changes in vital status, including death, are recorded on this card. Second, population registers also provide data on the population at risk by single year of age and sex. Finally, mortality data by single year of age at death and year of birth are available for all ages, including centenarians.

There seems to be need for a method of estimating the expectation of life at old ages directly from data on mortality in old age that is robust in the presence of substantial misreporting of ages (Horiuchi & Coale 1982).

3.4. Male-female differences

The important question in this context is whether the differential increase in the force of mortality is due to a differential ageing process between males and females, or whether this difference can be attributed to a stronger selection of the male population towards low-frailty individuals that is caused by the higher overall level of male mortality.

When comparing the 'male' and 'female' patterns of contribution, the pattern for women should be given more weight at the higher ages than the pattern for men. Because sex differentials in mortality are large at the older ages, the larger proportion of those surviving to older ages are women, leading to a "feminization" of the adult and elderly population. (Gomez & Boe 2005).

So differential mortality by sex continues to benefit women, they continue recovering from the century a lost biological advantage that, together with healthier behaviour and different life conditions, have also guaranteed a

social advantage in front of death (Vallin 2002; Waldron 1983). But in the future it is possible that in developed countries a different and regressive profile may arise since women have incorporated the habits and life styles characteristic of men. These factors, which regulate differential mortality by gender, are considered as undesirable outcomes in terms of both current female mortality patterns and future trends. For instance, in terms of life expectancy, many northern and western European countries have witnessed a decline in gender differences since the 1980s (Council of Europe 2003), which researchers have related to the significant changes that occurred during the same period in the life style, education, family roles and employment of women (Annandale & Hunt 2000; Waldron, 1983; Spijker et al. 2007).

3.5. Conclusions

Mortality at old age is a crucial component of any forecasting model for overall mortality. At the same time, more problems are inherent in the measurement and modelling of mortality at old ages than at lower ages. In effect, more uncertainty must be expected in the forecasts of old-age mortality.

Most discussions about old-age mortality have focused on its trends, levels and age patterns and there is no doubt today that mortality slows down over and beyond the age of 85 and that mortality rates observed at 95 or 100 years old are lower than expected. But among researchers there is little agreement with respect to the age patterns of old-age mortality. Some researches suggest that mortality increases exponentially with age, following the pattern of a Gompertz curve. Others state that human death rates increase exponentially with age from approximately 30-35 years to 80-85 years (e.g. Horiuchi & Coale 1990). Regarding the choice of preferable methods, the answer is clearly related to the type, the extent, and the quality of the data available at the moment of projection.

Mediterranean Fruit Fly

"My hunch is that much of the future growth of the field of demography will be in the direction of our legitimate but under explored territory in the biological sciences"

James W Vaupel (2004)[19]

CHAPTER 4 INCORPORATING BIODEMOGRAPHY INTO THE MODELLING OF OLD AGE MORTALITY

4.1. Introduction

The biodemography is a multidisciplinary approach that arose in the late of 20th century as a product of efforts to integrate biological knowledge (studies on human biology and animal models) with demographic research on human longevity and survival. Researchers in this emerging field have discovered attributes of ageing and death in humans that may very well change the way epidemiologists view and study the causes and of disease (Olshansky et al. 2005).

The earliest antecedents to the modern biodemography date back to the French zoologist Georges Buffon (1749), who demonstrated that "physical laws" regulate the duration of life in humans and other species. These physical laws, according to Buffon, link the biological clocks that he thought influenced the duration of life. He hypothesized that each species has a characteristic maximum life span and that this maximum life span is six or seven times the duration of the period of growth. The British actuary Benjamin Gompertz (1825) provided the first mathematical support for this view. Specifically, he found that the force of mortality increases in geometrical progression with the age of adult humans. According to him, human mortality rates double over about every eight years of adult age.

[19] Vaupel,J.W (2004) .The Biodemography of Aging *Population and Development Review*, Vol. 30, Supplement: Aging, Health, and Public Policy. pp. 48-62.

The linkage between Buffon's early life events and Gompertz's regularity in the timing of death appeared many years later with empirical evidence demonstrating that for some species, duration of life is calibrated to the onset and length of the reproductive window (i.e. at menopause) (Carnes et al. 1996). These observations serve as the foundation of the biodemography discipline.

The theoretical basis for the biodemography is derived principally from evolutionary biology (Kirkwood 1977; Hamilton 1966; Medawar 1951; Williams 1957), but it has roots in historical efforts of scientists who speculated on what was referred to as a "law of mortality" (Greenwood 1928; Brody 1924; Brownlee 1919; Makeham 1867; Gompertz 1825) - the observation that a consistent age pattern of death occurs across species (Carnes et al. 1996; Charlesworth 1994; Carey et al. 1992; Gavrilov & Gavrilova 1991; Finch 1990). The modern notion of the biodemography arose in the late 1980 and early 1990s, when scientists from a broad range of scientific backgrounds began speculating about the biological forces responsible for the similar age patterns of death that they observed among sexually-reproducing species. Including James R. Carey, James W. Curtsinger, Thomas E. Johnson, Vaino Kannisto, Pamela Larsen, Hans Lundström, A. Roger Thatcher, Anatoli I. Yashin, James Vaupel, and others — succeeded in peering into the remote regions of survival for five species — Homo sapiens, Drosophila melanogaster, Ceratitis capitata (better known as the medfly), Anastrepha ludens (the somewhat larger Mexican fruit fly), and Caenorhabditis elegans (a nematode worm) (Wachter & Finch 1997[20]). Thus, interest in integrating biology into demography in the context of ageing and longevity began to grow.

There are at least three general ways by which the inclusion of biodemographic concepts in demography coursework can strengthen the field (from Carey & Judge 2001).

[20] Demographer Kenneth Wachterk and molecular biologist Caleb Finch (1997),in the opening and ending chapters, respectively do explore the central questions at the core of biodemography - which are appropriately captured by2 the graphic title, *Between Zeus and the Salmon.* Zeus, the principal (immortal) god of the Greek pantheon, and salmon, genetically programmed to die shortly after they reproduce, represent the extremes that bound the study of ageing and longevity (Olashansky 1998).

1. Biodemographic principles provide a scientific knowledge that is lacking in conventional texts on demography and actuarial science.

2. Biodemographic principles can provide explanations for life table patterns in human populations that are not evident in the absence of broader biological concepts. For example, they suggest explanations of sex differentials in life expectancies, why older individuals may grow old more slowly, whether life span limits exists, whether post-reproductive life is common or rare and if how post-reproductive life spans in other species increase fitness.

3. Biodemographic principles can be used as a more secure foundation for qualitative *predictions* in the context of old-age mortality, upper limits of life span, and the magnitude and sign of the gender gap (Carey & Judge 2000; Wilmouth 1998). For example, it was not known until the results of animals studies were reported showing similar patterns whether slowing of mortality at older ages in humans was an artifact of small numbers of observations, a period effect, or real (Vaupel et al. 1998).

I believe that incorporating the field of biodemography will provide context, perspective and a frame of reference for the interpretation and modelling of human actuarial data (see Carnes & Olshansky 1993; Olshansky & Carnes 1997 for historical review) and will shed new light on those factors correlated with the life history of our species and those factors explaining demographic variations between populations.

4.2. Principles of Biodemography

The broad purpose of this section is to describe what Carey & Judge (2001) consider to be the most fundamental and important principles in biodemography that apply to a wide range of species including humans. I consider that these principles are important because they summarize and

synthesize many of the general demographic properties and concepts that apply to a wide range of species. The construction of this preliminary set of general principles encourages a greater communication between demographers and biologists by defining a common goal- identifying general demographic patterns (Carey 2001). Each of the principles listed above are general and therefore applies to virtually all species including humans of course.

1. *Principle of senescence:* senescence is a product of natural selection on survival versus reproduction. Medawar (1951) demonstrated that age-structured populations result from the chance application of mortality even in reproducing but non-senescing populations. In age-structured population, the force of natural selection decreases with age. Because more individuals of early age exist than individuals of older age, selection acts more forcefully among early expressing variable traits than among those traits that are only expressed later.

2. *Principles of mortality:* the single most important function of the life table is age-specific mortality, the fraction of individuals alive at age x that die prior to age x+1. Below I describe mortality concepts that Carey & Judge (2001) considered relevant to understanding mortality in humans.

    - *Death versus failure*: the term "death" is often used in metaphorical context such as automobile accident as the death of car. This distinction between literal and metaphorical is important in the context of mortality or death rate. Whereas cars can fail and therefore possess a failure rate, only living organisms can die and therefore possess a true death rate. This distinction is important in biodemography because of the argument that complexity may explain similarities in the age-specific patterns of death (or failure) in mechanical and biological systems (e.g. Abernethy 1979; Vaupel et al. 1998).

- *Mortality deceleration at advanced years*: mortality rates slow in virtually all organisms at older ages. This deceleration implies that there is no definite upper limit to life span.

- *Sex-specific mortality:* the mortality response of males and females of virtually all species will be different in similar environments. Female-longevity advantage is not universal across species.

- *Mortality trajectories:* The trajectory of mortality in all species including humans is "facultative": a term used in biology to describe life history traits with alternative conditions that vary with environmental conditions.

- *Demographic selection:* As population age, they become more selected because groups with higher death rates die out in greater numbers than those with lower death rates, thereby transforming the population into one consisting mostly of individuals with low death rates (Carey & Liedo 1995; Rogers 1995; Keyfitz 1985; Vaupel et al. 1979).

- *Subdetectable mortality:* a major concern of any study designed to estimate mortality rates at older ages in a cohort is sample size. The number of individuals at risk of dying becomes progressively less with age due to attrition. Consequently the sample size needed to measure mortality at older ages may be insufficient. Death rate cannot be measured below 1/n where n is the cohort size. In conclusion, this problem with "demographic sampling error" can bias estimates of rates of ageing, age at onset of senescence, costs of reproduction, and demographic tests of evolutionary models of ageing (Promislow et al. 1999).

- *Mortality variance increases with cohort age:* Human life tables at the younger ages are constructed from tens of thousands

or millions of deaths. Statistical properties such as the standard deviation and variance of mortality at each age are small and thus inconsequential. However, in biology, life tables are frequently based on a smaller population (either by design or because some animals live long and are expensive to maintain), making life table information more deficient as the number of individuals in a cohort decreases with age that causes the variance to increase. Such statistical problems are also becoming important in human populations, particularly at very old ages.

3. *Principles of longevity:* longevity refers to the period between birth and death of an individual.

- *Longevity is adaptive:* In evolutionary biology an "adaptation "is a characteristic of organisms whose properties are the result of selection in a particular functional context (West-Eberhard 1992). Thus, life span is evolved to solve particular ecological problems such as bridging unfavourable periods (droughts) or, for humans and other species, as a co-adaptation with sociality. This finding is important because it suggest that extended longevity should be considered along with features such as a large brain, bipedalism (standing on two appendages) and language as a key trait of our species (Carey & Judge 2001).

- *Maximal age is influenced by sample size:* The value of the parameter "maximal age" is dubious because the magnitude of the largest characteristic life span grows with an increase in the number of observations (Gavrilov & Gavrilova 1991). This is a general principle of statistics. The extent of the sample size affect is also affected by the shape of the survivorship curve. The more rectangular the survivorship curve the less variation in maximum observed life span with sample size (Calder 1996).

- *Life span is indeterminate:* maximal length of life remains as one of the most compelling concepts in demography. The validity of this concept is viewed by many as self-evident because different species exhibit different life expectancies; all individuals eventually die before the age of infinity; and therefore, each species must possess unique and finite maximal ages. Thus, life span is open-ended and dynamic; it is most usefully considered in the context of a species' ecology and evolutionary biology.

- *Reproduction as a longevity determinant:* Most organisms, from yeast and plants to invertebrates, birds and mammals, suspend reproduction during periods unfavourable for reproduction by entering a different physiological mode. Such waiting strategies for prolonging survival while maintaining reproductive potential have been extensively documented in the physiological, ecological and natural history literature (see Carey et al. 1998). In other words. a unit increase in reproduction at young ages causes a unit decrement in mortality at older ages. More generally, reproduction and mortality are inextricably linked (see MacInnes & Pérez Díaz 2005).

- *The heritability of individual life span is small:* life span heritability is defined as the proportion of the variance among individual ages of death that is attributable to differences in genotype (see Futyuma 1998). Contrary to popular myth, parental age at death appears to have minimal prognostic significance for offspring longevity (McGue et al. 1993).

Thus, these principles provide an important base to understand human birth and death rates, because the principles can help formulate questions being relevant to future directions and encourage biologists and demographers to identify other general demographic patterns.

4.3. The major biodemographic findings

My broad objective in this part is to use the results of studies in biological species to demonstrate the relevance and importance of biodemographic perspectives in human population studies such as the trajectory of mortality at older ages, sex mortality differentials or the concept of maximal life span.

Late-life mortality deceleration

Attempts to develop a fundamental quantitative theory of ageing, mortality and life span have deep historical roots. As we have seen in 1825, the British actuary Benjamin Gompertz found what he referred to as the *"law of mortality"*[21] (Gompertz (1872, 1862, 1825, 1820). Specifically, Gompertz model suggests that the number of living organisms decreases in geometrical (exponential) progression as age increases in arithmetical (linear) progression so this implies that the probability of dying with age increases exponentially after age 15 (Finch 1990; Pressat 1985).

The Gompertz model has been used widely in gerontology for fitting mortality schedules of nonhumans species (Finch 1990) and for comparing rates of actuarial aging between and among different species. (Comfort 1979; Gavrilov & Gavrilova 1991; Lamb 1977). It is also used in demography for smoothing the mortality functions of recorded life tables (Pressat 1985), for developing model life tables (Coale & Demeney 1983), and for projecting numbers of persons living to older ages (Bell, Wade &Goss 1992). On the other hand, Economos (1982) and others (e.g. Carey et al. 1992; Gavrilov & Gavrilova 1991) reported non-Gompertzian patterns of mortality at older ages in insects and humans.

[21] The Gompertz model is recognized by most demographers and gerontologists as an empirical model and not a "law" (Finch 1990), but not all gerentologists and medical researchers agree. For example, Fries (1983) stated that " Gompertz's law appears to hold in all populations and assures an exponentially increasing mortality rate and, therefore, death for the entire population within a decade or two past the age of 100".

This late-life mortality deceleration is a confuse observation. Gompertz himself, as well as numerous other scientists of the nineteenth and twentieth centuries, stated explicitly that his model did not apply, and in fact was never intended to apply, to older regions of the lifespan. For example, both Gompertz (1872, 1825) and Makeman (1867) recognized that the rise in human mortality decelerates at older ages. Makeham stated that for humans "the rapidity of the increase in the death rate decelerated beyond age 75". Perks (1932) identified a "curious peak in the rate of increase in death rate round about age 80" and observed that "the graduated curve of mortality starts to decline in the neighbourhood of age 84". Strehler & Mildvan (1960) argued that one of the four distinct phases of the human mortality curve was "a period of departure from the Gompertzian relationship at great ages so that mortality rises more slowly than anticipated after age 85-90". Strechler & Mildvan (1960) extended this to other species by noting that "at extremely advanced age, the mortality rate curves of several species rise at a rate progressively lower than exponential". More recently, Vaupel (1997) expressed that "until recently, it was impossible to determine whether this exponential rise continued to advanced ages". For humans, the scattered data available suggested that death rates keep on going up with age up to advanced ages but the rate of increase slows down, in other words mortality decelerate at the highest ages, but questions about data reliability precluded strong conclusions. For some species, virtually nothing was known about mortality at advanced ages because the populations studied had been too small to permit dependable estimates of death rates at ages that only a small fraction of the starting cohort reached (Carey et al. 1992) and for some other species, such as medflies, death rates reach a plateau and then fall dramatically. A number of other studies (Wang et al. 1997; Clark & Guadalupe 1995; Tatar & Carey (1994a, 1994b); Vaupel, Johnson, & Lithgow 1994; Curtsinger et al. 1992) reinforce this finding.

Although many attempts have been made to test the Gompertz model, the problem to find out when this model is correct still continues. Similarly, the reason why mortality decelerates at older ages in all the species (include

humans) for which large, careful studies have been conducted is also unclear. Thus, further research is warranted because as Vaupel (1997) asserted "at present it seems implausible that all of the observed deceleration of mortality at older ages is an artefact of heterogeneity".

Female longevity advantage

The prevailing wisdom in gerontology is that the female advantage in life expectancy is a universal law of nature. Unfortunately the scientific literature on sex longevity differences is ambiguous and it is still impossible to use the results of mortality studies on medflies or other species to predict the direction, rate of change, and extent of the human gender gap in the future. Some argue that females' greater longevity is essentially a universal law of nature (Hazzard 1990; Brody & Brock 1985; Hamilton & Mestler 1969) whereas others suggest that males of most species exhibit about the same life expectancy as females (Gavrilov & Gavrilova 1991; Smith 1989; Smith & Warner 1989; Lints et al. 1983)[22].

I share the view with Carey and Liedo (1995a) that there were at least three reasons why it is impossible to state unequivocally that either males or females live longer. On the basis of their medflies study they found that:

1. Longevity can be characterized in a number of different ways (e.g., life expectancy at eclosion [day 0], life expectancy at day 30, age when 90% of the original cohort is dead (life endurancy, maximal life span, etc.); Carey and Liedo discovered that one measure of longevity often favoured one sex, whereas another measure favoured the other sex.

2. The considerable variation among cohorts for a given longevity measure. For example, neither male nor female

[22] For additional perspectives on male-female differences in longevity see Hamilton 1948; Hazzard et al. 1991; Hazzard & Bowden 1990; Lopez & Ruzicka 1983; Manton et al 1995; Nathanson 1984.

longevity was greater in all of the groups regardless of the measure used.

3. Relative longevity for the two sexes was conditional on the environment in which they were maintained or the treatment to which they were subjected. For example, expectation of life for males and females was similar if flies were maintained in solitary confinement but favoured males if the flies were maintained in grouped cages.

I strongly believe that these same issues could arise in comparing other nonhuman species and that knowledge of whether the female advantage longevity or not is a general characteristic of nonhuman species would provide an important biological context for interpreting the current and predicting the future of the gender gap in humans.

Life span limits[23]

A fundamental question in population research is whether humans and other species possess a fixed life span limit and it remains as one of the most compelling concepts in human biology and demography. Although there has been considerable debate on whether the human life span is fixed, the evidence now appears to support the notion that no single number can be assigned as the life span limit of a species (Carey 2003).

Works since 1990 have been realized to try to understand this limits (Carnes et al 2003; Bongaarts & Feeney, 2002; Olshansky & Carnes 2002; Vaupel 2002; White 2002; Wilmoth (2002,2001,2000,1998); Olshansky et al. 2001; Riley 2001; Gavrilov & Gavrilova 1998; Vallin & Caselli 1997; Manton & Stallard 1996; Kannisto 1994; Olshansky & Carnes 1994; Carnes

[23] The oldest verifies age to which a human being has ever lived is 122 years and 167 days, a record held by the Frenchwoman Jeanne Calment, who was born on 21 February 1875 and died on 4 August 1997 (Allard et al.1998). Other verified long livers include Sarah Knauss, Mare Meiller, Chris Mortinson and Charlotte Hughs, who lived 118.1, 117.6,115.8 and 115.6 years (Robine & Vaupel 2001). Recently on 5 March 2004 died in Migjorn Gran (Spain) Joan Riudavets Moll who lived 114 years.

& Olshansky 1993; Vallin (1993,1992); Manton et al. 1991). Kannisto pointed out that the problem with this concept is that our knowledge of the nature of mortality makes it difficult to accept the notion that there is an age that some may reach but that no one has any chance to surviving. Manton and Stallard noted that declines in the age rate of increase of mortality for male and female cohorts in the U.S. are inconsistent with a fixed life span limit. Gavrilov and Gavrilova also rejected the notion of a human-specific upper age limit; they argued that people die before the age of infinity, not because they cannot pass some bounding age, but because the probability of a person avoiding the ever-present risks o death for that long is infinitesimal. On the other hand, in spite of the recent patterns of declines in mortality at the oldest ages and major increases in the prevalence of centenarians, there is still considerable support for the concept of a definite upper life span limit for humans (Olshansky et al. 2001; Vaupel et al. 1998; Manton, Stallard & Tolley 1991; Olshansky, Carnes & Cassel 1990; Fries 1980).

The ambiguity of the concept of life span limits is also evident from different species mortality data. It has been demonstrated that the results of large-scale life table studies in virtually all species that have been examined reveal a deceleration in mortality at older ages. If a single life span limit existed for all species, a pattern of records of the long lived should begin to appear for a least some of the species for which longevity information is available. However, this is not the case: there is no clustering of longevity records from independent sources for thousands of species for which life span data are available (Carey & Judge 2000). Moreover, the concept of a fixed species-specific life span is itself misleading because of issues such as species strains, biotype, sex, hybrids and seasonal variants. For example, no single life span exists for honeybees even in theory since queens live 4 to 6 years, workers 6 weeks and drones from a few days to several weeks (Wilson 1971). A similar problem exists for the domestic dog with its scores of breeds each with different life spans (Patronek et al. 1997).

I would like to share the view of Wilmoth & Lundstrom (1996) when he suggested "that the relevant limit of human life span may be in the pace of improvement rather than in the ultimate level to be attained".

4.5. Conclusions

My broad objective in this chapter was to develop a framework for the emerging field of biodemography. I started the chapter with an introduction of the discipline, then I described sets of general principles on mortality and longevity derived from Carey & Judge (2001) research on model systems and various longevity data sets on both human and non-human species.

The integration of biology into demography will provide a deeper understanding of demographic processes and insights into what patterns are common to a broad range of species and humans as well so, I think that now is an exciting time for those who are studying biodemography.

¿Que es lo que más le gusta de su deporte? la libertad y olor a vida

Jose Antonio Hermida [24]

CHAPTER 5 DISCUSSION

5.1. Discussion

This question of the art has been a review of the older as well as the recent literature about old age mortality in developed countries. The evolution in mortality among the oldest old has been discussed in terms of the Epidemiological Transition, the Compression of Mortality, the Limit of Human Life Span and the Rectangularization of the Survival Curve, in which one could affirm that the oldest old today is the fastest growing segment of the population in most developed countries. As old age mortality will be the key component responsible for changes in mortality in the future, forecasting survival, health or mortality for the oldest over the next decades has become an urgent political need, as developed societies aim to avoid a likely financially overstrained social insurance and medical systems.

What is uncertain, however, is whether this current trend in old age mortality will continue or not. Although there is a vast amount of accumulated knowledge that is specific to human mortality I believe that this is a good moment to review part of this literature, in particular that is

[24] *What is the most you like of your sport? The freedom and life's smell.* Solobici nº 191 pag.145.2007. José Hermida holds his position within the top of the world with impressive consistency: On world cup races he became two times runner-up, he finished three times on place four and was one of the hottest favourites in almost every race he started in. As his country's most prominent mountain biker, he is very experienced: From the junior ranks to the espoirs and elite, he celebrated significant successes in all categories, crowned so far by the Olympic silver medal in Athens. No matter if the course is technically simple and fast or extremely tricky and demanding, Hermida is always a candidate for the podium. It seems only a question of time until José goes for the rainbow jersey, and who knows, maybe he will be able to improve his best Olympic result in Peking. (http://www2.meridabikes.com/en_INT/Team.Hermida).

related to the slowing down of old age mortality, as this will form part of the theoretical framework of my PhD thesis.

When I was in my first year of the PhD course I got by chance an article by Dudley Lirk[25](1996) where he made the following observation about the Demographic Transition Theory: "*Demography is a science short on theory but rich in quantification. In spite of this it has produced one of the best-documented generalizations in the social sciences: the demographic transition."* Between demographers there is a debate if the demographic transition is or not a theory. Dudley denoted that "*For some, transition theory lies at the centre of modern scientific demography. Demeny (1972) has called it 'the central preoccupation of modern demography'. To others it is a non-theory to be dismissed as an unproven generalization unworthy of much discussion (Coleman and Schofield 1986)".*

I have made this reference because I saw a similar situation with Gompertz' law. Even though the Gompertz's law of mortality captured the attention of scientists for the past 170 years the current situation with regard to the applicability of his law as a general one is ambiguous. This is recognized by most demographers as an empirical model and not a "law" (Finch 1990), but not all researchers agree. For example, Fries (1983) stated that "*Gompertz's law appears to hold in all populations and assures an exponentially increasing mortality rate and, therefore, death for the entire population within a decade or two past the age of 100".* Willekens (2001) said that "*The Gompertz is without doubt the best known model, used in a range of disciplines from botany to sociology".*

As regards to my future research (i.e. the PhD thesis), after having reviewed the literature, I would like to go deeper into forecasting mortality of the oldest old. As we know, the oldest old population is growing. However, even though several leading forecasting mortality models (e.g. Makeham, Thiele, Perks, Helliman & Pollard) contain the Gompertz's model as one of their components, the Gompertz's law does not apply to the

[25] Kirk D (1996). Demographic Transition Theory. Population Studies, 50,361-387.

entire age range, including the population that I would like to study: the oldest old. Moreover, as was concluded in Chapter 3 of this PhD research report, there also exist a difficulty in applying the cause-of-death approach for the oldest old as the underlying cause of death cannot be easily identified, which implies that cause-specific projections cannot be performed for very old age groups. Thus, because the Gompertz model as a unique model seems to be limited, my idea for the PhD thesis is to start with Gompertz's law and linked it with a mortality model that incorporates theoretical concepts of ageing such as Strehler & Mildvan (1960), Sacher & Trucco (1962), as well as the fixed heterogeneity and debilitation models. They provide explanations for Gompertz (exponential) patterns and for the 'leveling off' pattern of mortality" (see Chapter 3 and Yashin et al. 1994 for a review).

On the other hand, I think that demography like other disciplines is faced with the continuous need to adapt new issues such as new concepts in life table analysis, advances in census techniques, new models for forecasting mortality, a better understanding of why and how we age and so forth, something which requires interdisciplinary efforts. In my opinion demographers who study mortality have much to gain by acquiring a basic understanding and awareness of developments in many different areas such as biostatistics, genetics, molecular biology and gerontology.

In Chapter 2 of this work I have seen that the Evolutionary theories of ageing have made an important contribution for the understanding of our evolution. But, even these theories still continue to be weak in explaining observed mortality patterns and I think that studies on the evolution of species may provide us with a better understanding of the role of the oldest old in nature. In my opinion, new models are needed to better understand the regularities of the evolution of life history traits among species that will aid in the understanding of our evolution as humans. Thus, for my PhD thesis I would like to go deeper into the field of biodemography, starting with the biological explanations of oldest old mortality.

Finally, I should mention than when modelling old-age mortality, it is vital to work with high-quality age-specific data. For example, data series truncated at age 75 are of little value, because mortality as a function of age increases at different rates in different age intervals. An important feature is the acceleration of the age-related increase in mortality at ages approaching 75 years and then a deceleration among those above the age of 85. Thus, methods that extrapolate mortality using data below age 75 or even 85 are likely to produce very unreliable results. For that reason for my thesis I am thinking of only studying developed countries using the Kannisto-Thatcher Database on Old Age Mortality that includes single-age mortality data up to age 110+, with time series of up to more than 250 years .

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Annex I : Main parameterization functions for mortality[26]

OLD NON-POLYNOMIALS

De Moivre	1725	$\mu(x) = \frac{1}{(\omega - x)}$
Gompertz	1825	$\mu(x) = Bc^x$
Makeham	1860	$\mu(x) = A + Bc^x$
		$\mu(x) = \alpha + \gamma x + \beta c^x$
Opperman	1870	$\mu(x) = \frac{a}{\sqrt{x}} + b + c\sqrt{x}$
Thiele	1872	$\mu(x) = a_1 e^{-b_1 x} + a_2 e^{-\frac{1}{2}b_2(x-c)^2} + a_3 e^{b_3 x}$
Wittstein	1883	$q(x) = \frac{1}{m} a^{-(mx)^n} + a^{-(M-x)^n}$
Steffenson	1930	$\log_{10} s(x) = 10^{-A\sqrt{x} - B} + C$
		$e(x) = \frac{1}{A + Bc^x}$
Perks	1932	$\mu(x) = \frac{A + Bc^x}{kc^{-x} + 1 + Dc^x}$
Harper	1936	$\log_{10} s(x) = A + 10^{B\sqrt{x} + Cx + D}$
Weibull	1939	$\mu(x) = \alpha x^{\beta - 1}$
Van der Maen	1943	$\mu(x) = A + Bx + Cx^2 + \frac{I}{N - x}$
		$\mu(x) = A + Bc^x + \frac{c}{N - x}$

[26] Taken from Tabeau E (2001)

POLYNOMIALS

Unnamed
(in Keyfitz, 1982)

$$y(x) = e^{(a_0 + a_1 x + a_2 x^2 + \ldots + a_k x^k)}$$

$$y(x) = q(x),\ \frac{q(x)}{p(x)},\ \mu(x),\ or\ e(x)$$

RECENT NON-POLYNOMIALS

Brillinger 1960

$$\mu(x) = \sum_i \left(H_i (x - B_i)^{C_i - 1} + \frac{A_i}{(b_i - x)^{C_i + 1}} + E_i d_i^x \right)$$

Beard 1961

$$\mu(x) = \frac{Be^{ux}}{1 + De^{ux}}$$

Petrioli 1981

$$s(x) = \frac{1}{x^a (\omega - x)^{-b} e^{\frac{c}{2}x^2 + dx} \left(\frac{1}{k} + 1 \right)}$$

Martinelle 1987

$$\mu(x) = \frac{A + Be^{kx}}{1 + De^{kx}} + ce^{kx}$$

British actuaries 1980s
(in Keyfitz, 1982)

$$\frac{q(x)}{p(x)} = A - Hx + bc^x$$

RECENT NON-POLYNOMIAL

Siller 1979

$$\mu(x) = a_1 e^{-b_1 t} + a_2 + a_3 e^{b_3 t}$$

Heligman-Pollard 1980

$$\frac{q(x)}{p(x)} = A^{(x+B)^C} + De^{-E(\ln x - \ln F)^2} + GH^x$$

$$q(x) = A^{(x+B)^C} + De^{-E(\ln x - \ln F)^2} + \frac{GH^x}{1 + GH^x}$$

$$q(x) = A^{(x+B)^C} + De^{-E(\ln x - \ln F)^2} + \frac{GH^x}{1 + KGH^x}$$

$$q(x) = A^{(x+B)^C} + De^{-E(\ln x - \ln F)^2} + \frac{GH^{x^K}}{1 + GH^{x^K}}$$

Brooks *et al.* 1980

$$\mu(x) = \mu_I(x) + \mu_A(x) + \mu_S(x)$$

$$\mu_I(x) = \begin{cases} Q_0 & \text{for } x = 0 \\ Q_1^{x^\gamma} & \text{for } x > 0 \end{cases}$$

$$\mu_A(x) = Q_A e^{-\frac{(\ln x - \ln x_A)^2}{\delta^2}} \quad \text{for } x \geq 0$$

$$\mu_S(x) = \frac{Q_S e^{\frac{x}{x_S}}}{1 + Q_S e^{\frac{x}{x_S}}} \quad \text{for } x \geq 0$$

Rogers and Planck 1983

$$q(x) = A_0 + A_1 e^{-\alpha_1 x} + A_2 e^{-\alpha_2(x-\mu_2) - e^{-\lambda_2(x-\mu_2)}} + A_3 e^{\alpha_3 x}$$

Kostaki 1992

$$\frac{q(x)}{p(x)} = \begin{cases} A^{(x+B)^C} + De^{-E_1^2(\log x/F)^2} + GH^x, x \leq F \\ A^{(x+B)^C} + De^{-E_2^2(\log x/F)^2} + GH^x, x > F \end{cases}$$

Rogers and Little 1993

$$y(x) = a_0 + m_1(x) + m_2(x) + m_3(x) + m_4(x)$$

Where:

$m_1(x) = a_1 \exp(-\alpha_1 x)$

$m_2(x) = a_2 \exp(-\alpha_2(x - \mu_2) - \exp(-\lambda_2(x - \mu_2)))$

$m_3(x) = a_3 \exp(-\alpha_3(x - \mu_3) - \exp(-\lambda_3(x - \mu_3)))$

$m_4(x) = a_4 \exp(\alpha_4 x)$

$y(x) = q(x), \ \frac{q(x)}{p(x)}, \ \mu(x)$

RECENT NON-POLYNOMIAL, PARTIAL AGE FUNCTIONS

Hartmann 1981

ages 0 to 15 years:

$y(x) = A_1 + B_1 \ln x$

ages 15 to 35 years:

$y(x) = A_2 + B_2 x$

ages 35 to 60 years:

$y(x) = A_3 + B_3 c^x$,

	where $Y(x)$ - *logit of* $l(x)$
Mode and Busby 1982	ages 0 to 10 years: $\mu_0(x) = \alpha_0 \beta_0 e^{-\beta_0 x}$ ages 10 to 30 years: $\mu_1(x) = \alpha_1 - \beta_1 (x - \gamma_1)^2$ ages 30 and over: $\mu_2(x) \quad \alpha_2 + \beta_2 \gamma_2 e^{\gamma_2 x}$

Note: In this table the standard demographic notation is used. The survival function s is such that for continuous x, s(x) is the probability of survival from birth until age x. The life table symbol l(x) is the empirical equivalent of the function s(x). The probability that a person x will survive to age x+1 is p(x)= s(x+1)/s(x) and the probability that a person aged x dies before reaching age x+1 is q(x)=1-p(x). The force of mortality, also known as mortality intensity μ (x) = -(ds(x)/dx)/s(x). The symbol m(x) is used for its empirical equivalent, the mortality rate. The symbol e(x) denotes life expectancy at age x, ω is the highest attainable age, and the remaining symbols are model parameters.

Annex II : Main parameterization functions for old age mortality[27]

exponential	Gompertz	$m_x = be^{ax}$
shifted exponential	Makeham	$m_x = c + be^{ax}$
exponential-quadratic	Coale-Kisker	$m_x = e^{a+bx+cx^2}$
logistic 2-param	Himes-Preston-Condran	$m_x = \frac{be^{ax}}{1+be^{ax}}$
logistic 3-param	Beard	$m_x = \frac{be^{ax}}{1+ce^{ax}}$
logistic 4-param	Perks	$m_x = \frac{d+be^{ax}}{1+ce^{ax}}$
logistic 2-param	Heligman-Pollard	$q_x = \frac{be^{ax}}{1+be^{ax}}$
power	Weibull	$m_x = bx^a$
shifted power		$m_x = c + bx^a$
polynomial 3-param		$m_x = a + bx + cx^2$
polynomial 4-param		$m_x = a + bx + cx^2 + dx^3$

Note: All the functions are formulated in terms of the force of mortality μ_x. For the sake of simplicity of the calculations, the force of mortality μ_x has been replaced by the central death rate m_x. One function (Helligman-Pollard) is specified for the probability of dying q_x.

[27] Taken from Boleslawski & Tabeau (2001)