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THE ACTUARIAL AGING OF ITALIAN VETERANS OF WORLD WAR I BORN 1889-1901 AND A COMPARISON TO THE COHORTS BORN DURING THE YEARS IMMEDIATELY FOLLOWING

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The actuarial aging of Italian veterans of World War I born 1889-1906 and a comparison to the cohorts born during the years immediately following

This paper develops a comparative analysis of results provided by the "human actuarial senescence" and by those of the demographic approach in relation to mortality in old age. In particular we will examine a case which has previously been recorded, but not with regards to Italy. It deals in particular with the cohorts born between 1889 and 1900; the men involved therefore, took part directly in the first world war whilst the women experienced the harsh conditions of life during those years.

JEL Classification: N 4, keywords: First World War, mortality, actuarial senescence, birth-cohort life tables, demography.

1. Introduction

When Gompertz (1825) formulated his "universal law of human mortality", which later became famous, he could not have imagined that it would have become an important tool to measure the aging process, not only of the human population, but also for experimental applications in species of animals and, more generally, in the biodemographic approach to mortality modelling (Kirkwood, 2015).

In time other models have been put forward although the Gompertz model has never lost its luster because it has continued to play an important role in the development of theoretical hypotheses in relation to the patterns of mortality at old ages. Among these approaches, the actuarial method for life table analysis is studied here. This method has connections to the evolutionary theory of senescence (Rose, 1991; Tuljapurkar, 1997; Ricklefs and Scheuerlein, 2002) and examines the increase in mortality hazard at older ages as a consequence of increased physiological vulnerability. This process is referred to as "actuarial senescence" or "actuarial aging" and the actuarial method supplies the so-called aging measures which are obtained, among others, by the parameters of the model used to describe the course of actuarial senescence.

The results can be different depending on the approach used to study the phenomenon: a period or a cohort approach, because in the first case the level of mortality is influenced by the conditions "of the moment", whilst the second case brings to light the long-lasting effects of living conditions experienced from pre-adult age.

It is exactly on these latter aspects that this work focuses by examining a particular period in Italian history. It deals with a critical examination of both the approach and the explanation of the results provided by the actuarial method for cohort life table analysis regarding the mortality of young Italian adults who experienced the impact of World War I (WWI), both for the soldiers who fought on the war front, and for the women who remained at home or at work and suffered hardships. This paper will compare the evolution of the mortality of the older survivors from 85 years of age onwards with the process of actuarial senescence experienced by the cohorts that were born later. The results also provide insight for analysis of differential mortality according to sex measured by the gap between average lengths of life. The corresponding female birth-cohorts too, like the rest of the civilian population, suffered the extreme conditions and severe restrictions imposed by the war economy in order to support those who were at the war-front.

The birth-cohorts that are studied here are those born during the period from 1889 to 1919. This choice is obviously conditioned by the statistical data available (Istat, 2002) that does not give a complete picture of the mortality rates of all the male cohorts engaged in the conflict. Nevertheless, of the cohorts examined those that were born between 1889 and 1899 are the ones who suffered the experience of war to a far greater extent. The brief historical digression that follows may help to focus on this aspect (also see the relevant bibliography: Schaumann, 1993).

2. An historical outline

In Europe on the eve of World War I two alliances met face-to-face, the Triple Entente (United Kingdom, France and Russia) and the Triple Alliance (Austria-Hungary, Germany and Italy); the latter was of a defensive nature and guaranteed military support to whichever ally came under attack by another state. As is widely known the conflict began with Austria-Hungary declaring war on Serbia (July, 28th 1914), which was followed just a few days later by Germany's declaration of war on France and Russia; after that, one declaration of war succeeded another at a frantic pace thereby ushering in the enormous catastrophe that continued until November 11th1918.

Since the expected conditions foreseen by the alliance to intervene in the war were lacking, Italy declared itself neutral, but however, it came under strong pressure to support the war effort from two sides. As we know, in the end Italy deployed it's troops in favour of France and the United Kingdom, arriving at this decision not only due to the concessions that these two countries guaranteed in the case of victory, but also because of a more widespread popular sentiment in favor of these two countries on the one hand, but also due to general sentiments of protest towards the political choice of an alliance with Austria and Germany on the other hand. It must be said that the birth and building of Italy as a unitary state had originated from three wars (1848, 1860 and 1870), known as the Wars of Independence, fought precisely against Austria for the liberation of most of the Italian territories that were then part of the Austro-Hungarian Empire.

Once Italy decided on war in support of France and the United Kingdom, the Italian general headquarters didn't proceed immediately with a general mobilization but rather chose a preliminary individual call to arms so as to avoid suspicion from the Austrian government. So when the war started on May 24th1914 the initial operations were conducted by those who were already enrolled or had been released a short time before, which especially included cohorts born in the years prior to 1895 because in those times all men were conscripted to the army for two years upon reaching twenty years of age. However, besides having recalled the cohorts born in and up to the year 1875, by the autumn of 1916 the army proceeded to bring forward the enrollment of the cohort born in 1897, and then this happened again to those born later, up until the year 1900. As it will be seen, in this paper I aim to describe cohort patterns in all-cause mortality among the oldest of the elderly who fought during the war at young-adult ages.

The war initially gave some victories to the Italian army, but early in the fall of 1917 the first phase of the October Revolution gave rise to the conclusion of military operations on the Russian front and this allowed Austria-Hungary and Germany to concentrate their forces on the Western Front. As a consequence the Italian army suffered a dramatic defeat (October 1917) at Caporetto and was forced to withdraw to the Piave River. The subsequent Italian victory at this position, where the Austrian advance was stopped, lead a little later, to the collapse of the Austro-Hungarian Empire and the end of the war in November 1918. According to Italian government sources of the time the deaths were 651.000 and the wounded admitted to hospitals were 1.050.000. Approximately 70% of those deaths belong to the cohorts born between 1889 and 1900 (Jdanov, Glei, Jasilionis, 2010) and as such they are amongst those whose mortality is herewith studied according to the actuarial senescence method. It should also be noted that 28,6% of deaths (186.000) were due to disease from the harsh environmental conditions on the front; until around the end of 1917 the war was fought in the Alps, in some stretches of the front at high altitudes, with the risk of frostbite and in the general risk that diseases such as tuberculosis and rheumatic fever could become chronic. After the defeat of Caporetto, the war epicenter moved largely to the plains, along the Piave River, where there were malarial areas, and in the end it was in this environment that also the Spanish flu epidemic spread.

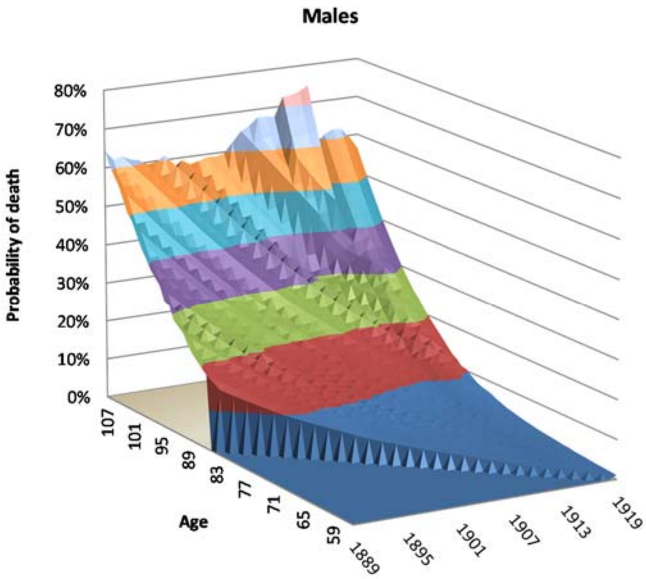
3. The construction of the dataset

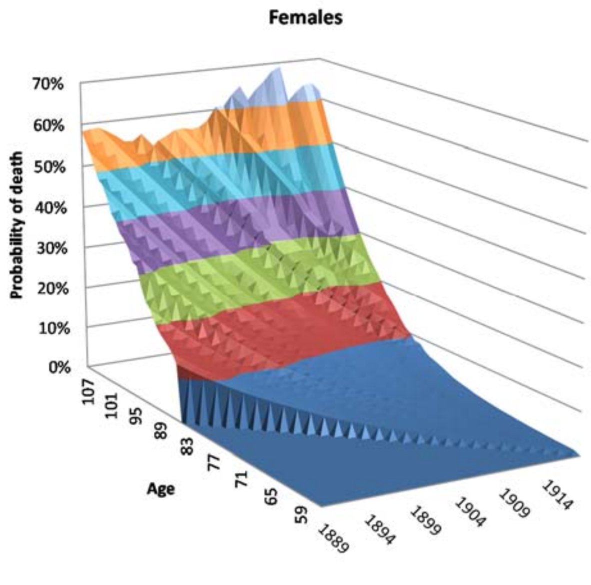
In recent years, the mortality analysis of extinct cohorts of WWI veterans born after 1889 may be based on death probabilities that in Italy, since 1974 are provided by Istat[1] in the time series of period life tables. As is known, there is also the HMD source, but its data regarding the period of interest for us presents a lower quality for the period 1872-1905, as HMD (2015) clearly specifies. Therefore we will not use the HMD source directly, because that work will lead us to consider also the differential mortality by sex at old ages, which is a subject that has already been studied (Maccheroni, 2014) using the Istat period life tables. The comparisons of the results obtained from those period life tables and from these birth cohort life tables must derive from the same source.
In Italy the study of mortality among the elderly population has for years found a more robust data base than in the past because demographic change has been characterized by an intense process of population aging that began with a rapid decline in fertility and an accelerated reduction in mortality; as a result there has been a steady increase of the elderly (Preston *et al.*, 1989). According to a recent study (Bonarini, 2009), in Italy the census of 1971 revealed that there was already a considerable number of centenarian women and this number increased progressively with the generations born after 1881; on the other hand the number of centenarian men has grown at a much slower pace.
Despite this however we can't assume that it's possible to get enough data from the population to construct the probability of death and period life tables for those over 100 years of age, and this especially applies to the 1970s and 1980s. There are, however, graduation methods (Thatcher, Kannisto, Vaupel, 1998) which enable one to extrapolate the probability of death for the age group for which the available data is problematical. Even Istat, which has so far built only period life tables, has followed this approach to determine the probabilities of death at elderly ages; at present this Institute provides the homogeneous time series 1974-2013 where period life tables end at age 119 (istat.demo.it) and obviously this ultimate age is older than the oldest age in the population. In the birth cohorts tables that we rewrote starting from the previous above-mentioned ones, the ultimate age is considered the one for which the number of survivors in the table is close to 1, which occurs towards 107 years for men and 108[2] years for women (Kannisto, 1994).
As already specified, the actuarial aging study was carried out on the cohorts born from 1889 until 1906 which means they are to be considered already extinct in 2013. Hence all the relative probabilities of death are obtained by relaborating the Istat 1974-2013 time series. The aim of the work was also, however, to understand the effects of the war and of the Spanish flu on the cohorts born in those years and to compare the results with those born during the years preceding those events. It is for this reason, that the life tables of the cohorts born between 1907 and 1919 were closed with an extrapolation made according to the Gompertz model [1] because those cohorts were not yet completely extinct in 2013. In fact, as a first step, the Gompertz model has been fitted to mortality data from age 85 onwards (Vaupel *et al.*, 1998) to study the actuarial aging for the cohorts born between 1889 and 1906 and some very satisfying results have been obtained (see R^2 statistic in table 1, following paragraph). It's precisely from these results that one proceeds to adapt and to extrapolate the [1] to the following cohorts which leads to conclude the birth cohort life table with a minimal addition for the 1907 cohort and with consistently greater integrations for the others, up to and including the 1919 cohort.

1 Italian national statistical office.

2 According to records the longest living survivor of the First World War died at the age of 108 years and belonged to the 1899 cohort.

Fig.1 – Probabilities of death (q_x) in Italian cohorts born in 1889-1919 from ages 55 on (cohort 1919) to 85 on (cohort 1889)

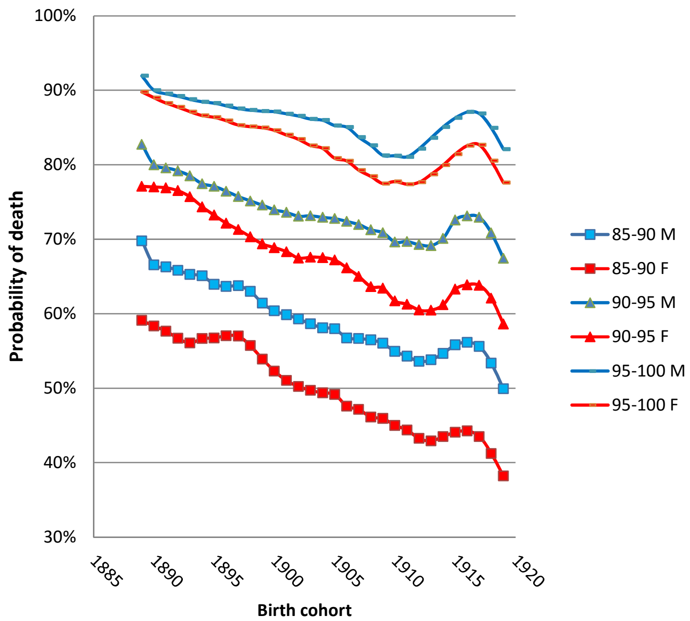




Our reconstruction of the extinction process of all the considered birth cohorts are shown in figure 1 in which the profile of the probabilities of death varying according to age and to cohort, highlights a general and progressive decline. There were also, however, certain years in which the health of the elderly suffered particularly. Reference is made here, in addition to what happened in 1983, especially to the consequences of the hot summer of 2003 that led in both cases to the net increase in mortality from diseases connected with the circulatory and respiratory systems (ISTAT, 2011). In figure 1 the effects of these changes are visible beginning with those aged 84 born in 1919 and continuing up to those aged 99 born in 1904.

Figure 2 gives us a preview of some of the results that will be examined in the following paragraphs. Here it is evident that those who were born during the war have experienced an old age mortality that is much higher than those who were born immediately before or after this period. In fact the rise in the mortality profile for both males and females clearly show this phenomenon, known as the cohort effect.
The effect of the end of WWI emerges for people who in those years were on the threshold of adulthood - those born between 1898 and 1902 - and who therefore experienced more rapid decline in mortality than the cohorts born before: at age 85 the mortality ($_5q_x$) decreases quickly, for both sexes, in particular for women, but only for those born after 1898. Fig. 2 also gives evidence of a "selection" effect in the case of 90 and 95 year old survivors, because in these cases the effect of severe living conditions throughout the war period cannot be seen for the cohort mortality born between 1889-1900.

Fig.2 – Cohort probabilities of death within a five year period ($_5q_x$) at selected ages, by sex and year of birth



4. The choice of the Gompertz model and the interpretation of its parameters

As mentioned, in Italy the present day aging of the population has taken place, not only due to the decline of fertility, but also as part of a profound change in the death rates at advanced ages (Vaupel, 2010) which trajectories have for many years been characterized by a continual decline in pace. Obviously it was not so in the past. Between 1960 and 1980, life expectancy at age 60 for men reamined stationary whilst life expectancy for 80 year olds even slightly decreased between 1970 and 1980. It is during this period that the birth cohorts who in youth or adulthood experienced the wartime conditions with it's uncertainty, suffering and deprivation, became either extinct or reached very advanced ages. In order to focus on their mortality, the Gompertz[3] model has been chosen from

3 Another model employed for studying actuarial aging is the Weibull one, but it was not here considered because it implies, amongst other notions, that causes of death of young adults and the elderly are indipendent. The Weibull model also seems to be a better describer of mortality for purer, single causes-of-death, while the Gompertz model would be a better describer of 'all-causes' of deaths (Juckett, Rosenberg, 1993; Gavrilov, Gavrilova, 2001).

among the available models as it has been widely employed to represent senescent mortality (Pollard and Valkovics, 1992; Olshansky and Carnes, 1997) and it takes into account the legacy of living conditions in youth and adult ages.
The model (Kirkwood, 2015) describes the increase of the mortality hazard, expressed by the instantaneous rate or the force of mortality μ_x, with increasing age *x*; it results in a mortality trajectory that would reflect the increasing vulnerability of individuals due to the decline of physiological functions with aging.
The formalization of the Gompertz's hypothesis about instantaneous rates leads to the following expression for the force of mortality

$$\mu_x = \mu_a \exp(\gamma x) \qquad [1]$$

where μ_a and γ are constant and the exponential term describes how this vulnerability increases with age. The [1] should apply therefore at least from early adult life; for the more recent cohorts it generally best fits data pertaining to the over 80's.
Evolutionary biology has for a long time been studying the questions of why senescence occurs and it considers Gompertz as one of the fathers of the discipline because with his model he went beyond the empirical observations about patterns of mortality in order to attribute biological significance to the life table. Between this discipline and the evolutionary theory of senescence, there are obviously large intersections; the latter proposes, among other things, a substantial partition of the total mortality in extrinsic and intrinsic (Carnes and Olshansky, 1997; Carnes *et al.*, 2006) and this partition does not claim to be exhaustive, but to support the experimental analysis. The former, being the consequence of external or violent causes, is age-independent and accounts for most pre-adult mortality; the latter is age-dependent. At the origin of extrinsic mortality there are, in fact, external causes such as environmental disasters, famine, war, severe climatic conditions, etc. The intrinsic mortality is on the other hand, the result of the decline of the physiological functions of the individual.
Returning to [1], for both disciplines here cited μ_a is also known as initial mortality rate (IMR) (Finch *et al.*, 1990), where *a* is the pre-adult age starting from which μ_x grows exponentially. The other parameter of [1], γ, is the rate of increase of the mortality rate with increasing age x. To graphically represent [1] the log-arithmetically transformation of μ_x is used and as such $\ln \mu_x$ appears as a straight line with x increasing; $\ln \mu_a$ is its intercepts and γ is its slope: this is the term with which γ is often indicated.
In this approach hereto described the Gompertz model thus incorporates the two components of mortality with these two parameters: the extrinsic one with μ_a, typical of the pre-adult or young adult ages and the intrinsic one with γ, that is, the expression at advanced ages, of vulnerability that is also the legacy of the past.
For the birth cohorts herewith examined, the statistics do not reveal mortality by age and cause of death, hence it is not here possible to try to make comparisons with the extrinsic or intrinsic levels provided from the model-based estimates. This approach presents however, from the application point of view, a margin of arbitrariness: on the one hand μ_a is defined as age-independent term, but, on the other hand, when [1] is adapted to the adult age, μ_a is associated with the extrinsic mortality. In this last case one would reconcile references to social age concepts such as adulthood or old age, which may vary from society to society and that in a certain society change over time, with the results of a mathematical model that describe the relationship between "chronological" age and mortality rates within an age group, whose threshold is still conventional; estimates of the parameters for the same cohort will therefore vary depending on the age groups to which the model has been adapted.
Broadly speaking IMR is considered to be a scale or level parameter or background mortality rate that affects all ages and then raises or lowers the mortality curve; this level of mortality can, depending on the case, be associated with hazard present in the history of the entire cohort and which in our case is actually observed.

To adapt [1] to the cohort life tables, the probabilities of death q_x (fig. 1) have been converted into instantaneous rates of mortality μ_x and can be conveniently approximated as

$$\mu_x \approx -\ln(1-q_x)$$

and parameter estimates μ_a and γ have been obtained by using the minimum sum of squared errors criterion (SSE minimum) based on the log-arithmetically transformation of μ_x; as aforementioned in the previous paragraph, the results of the adaptation have been very satisfying as can be observed by the index R^2 (tab. 1).

With regards to the time series of estimates obtained by the IMR and apart from the mortality level provided by μ_a, the results obtained may have a preliminary interpretation according to the evolutionary approach, especially in the case of men. In fact, the IMR has, comparatively, the highest values for those born until 1897 (tab. 1), thus registering a high mortality that is linked to high "frailty" caused by acquired weaknesses for the hardships borne during the wartime. As a matter of fact, from a comparison with the cohorts born after 1900, for which the estimates are much lower μ_a and in continuous decline (tab. 1), it can be deduced that with the passing of time the long-lasting deleterious effects of war on cohorts whose range of birth dates from 1889 to 1899 have not disappeared.

Turning our attention to the female sex, the μ_a model-based estimates have, obviously, lower levels than those of men, but the strong correlation among sexes (r = 0.898) confirms the harshness of the wartime living conditions for the civilians and in particular for the younger ones; just as in the case of their male peers, similarly for the women the highest values of μ_a are in fact those of the cohorts born during the 1889-1897 period, but this matter will be reconsidered later on.

Even in the historical series of slope there is a turning point for the cohorts born after 1897, which turning point may therefore be linked to the war. For these cohorts, the upward trend of γ may reflect a stronger intensity of natural selection with age since they from birth to young-adulthood endured a weaker selection than previous cohorts and therefore have a greater heterogeneity in later life with respect to frailty.

Also concerning γ, the provided model-based estimates for males are all lower than those of females (tab. 1) and these rates are positively correlated (Pearson's r = 0.9475); this higher rate of growth in mortality may be linked to the fact that if on the one hand women are characterized by a lower background mortality (IMR), on the other hand male and female mortality rates have to converge with increasing age, so inevitably their extinction process take place with a higher γ.

5. The aging measures and the historic trajectory of their change in cohorts born between 1889 and 1906

The parameters of the Gompertz model [1] are used for the construction of two indexes both indicated as "rates of aging" (Ricklefs and Scheuerlein, 2002) or aging mesures; one of these is the so-called $\boldsymbol{\omega}$ index

$$\omega = \sqrt{\mu_a \gamma} \qquad [2]$$

which is a geometric mean between two rates and hence it itself is a rate.
The results of [2], but as will be seen, also those of the following [3], are in line with our expectations, although with some difference between sexes during the first evolutionary time period across the birth cohorts (fig. 4). In the case of males ω it reaches its maximum with the generation born in 1890, and then slowly declines until reaching the 1897 birth cohort, those who at the beginning of the war (1915) were aged 18; the increase in mortality rate at older ages is then comparatively higher among those who were mobilized right from the beginning of the hostilities. The ω temporal decline then stops with the cohorts born in 1897 and 1898 who were sent to the war front in the most dramatic period of the conflict; after that the successive generations have experienced a gradual reduction in actuarial aging.
As regards the females, $\boldsymbol{\omega}$ grows after an initial decrease, reaches its maximum precisely with the cohort born in 1897 and then starts again to decrease (fig. 3-C); these women, as was so with the others, had to substitute in the workplace the men who had been mobilized. As already said, due to the war the cohorts born between 1874 – 1900 were gradually called to arms, even though only the youngest men were involved in military operations. About five million men thus left the productive work place and women were called to fill up the resultant vacant jobs, mostly in farming and in factories. They too, as with all other civilians, were subjected to food rationing, experienced a decrease in their purchasing power due to increased taxation in order to finance the war and they were without health care because almost all the medical staff was concentrated at the war front. Therefore it was those who at that time were on the threshold of adulthood who suffered to a greater extent the consequences of wartime living conditions in their old age, as already noted in the preceding paragraph.
The other index is a $\boldsymbol{\gamma}$ transformation and it is known as mortality rate doubling time (MRDT), where

$$\mathbf{MRDT} = \ln 2/\gamma \qquad [3]$$

This index indicates after how many years the risk of death tends to double; in human populations MRDT usually should be implied between 7 and 9 years (Gurven and Fenelon, 2009), which respectively identify a situation of low and high mortality. As can be seen from tab.1 and from fig.5 our results fall substantially in this range for the male cohorts born between 1889-1898 which have values slightly above 9; these are the generations that at the beginning of the war were between 17 and 26 years of age and as such the results are completely coherent with those of ω . In the case of the women MRDT values are all within those of the previous range and their trajectory presents a profile very similar to $\boldsymbol{\omega}$ (figures 3-C and 3-D). Nevertheless for a comparative analysis between these two measures of aging it is necessary to also consider the estimates of the two parameters of the model [1].
Just as the indexes [2] and [3] are strongly correlated with parameters of [1], so they are correlated amongst themselves (tab. 2). In particular tab. 2 reveals that the Pearson correlation between $\boldsymbol{\mu_a}$ and ω and between $\boldsymbol{\mu_a}$ and MRDT is almost 1 both for males and females (tab. 2) and practically −1 between $\boldsymbol{\gamma}$ and $\boldsymbol{\omega}$ and somewhat more between $\boldsymbol{\gamma}$ and MRDT.

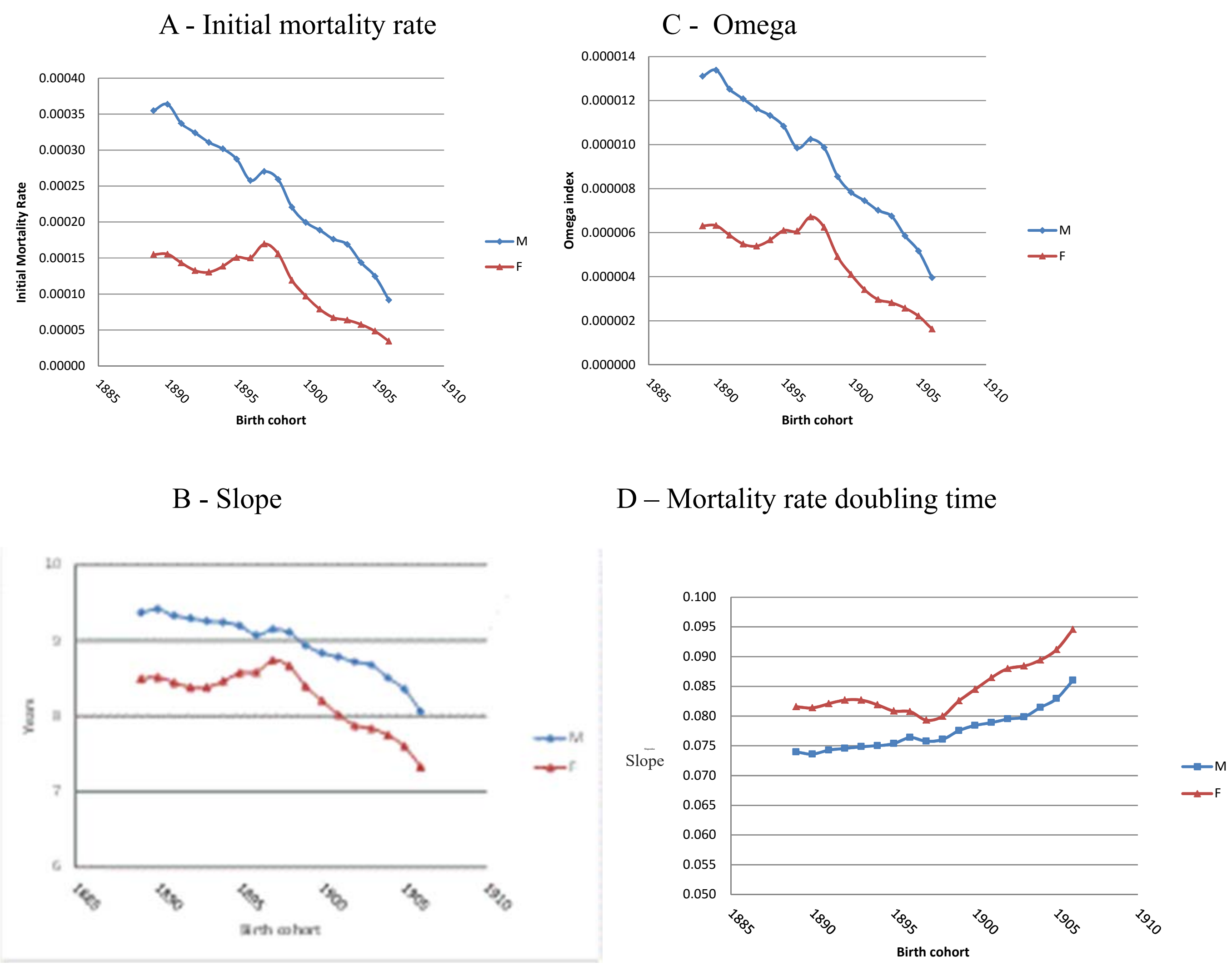
Tab.1 – Parameter estimates of Gompertz model*, R^2 statistic and aging indicators among Italian cohorts born 1889-1906

Cohort	IMR	SLOPE	R^2	MRDT	Omega
			Males		
1889	0.000355	0.073978	0.99796	9.369658	0.0051226
1890	0.000364	0.073613	0.99859	9.416131	0.0051733
1891	0.000337	0.074277	0.99843	9.331959	0.0050027
1892	0.000324	0.074560	0.99828	9.296469	0.0049148
1893	0.000311	0.074863	0.99822	9.258916	0.0048224
1894	0.000302	0.075008	0.99745	9.241024	0.0047572
1895	0.000287	0.075375	0.99698	9.195925	0.0046543
1896	0.000258	0.076396	0.99704	9.073027	0.0044373
1897	0.000270	0.075774	0.99676	9.147602	0.0045255
1898	0.000259	0.076078	0.99641	9.110983	0.0044411
1899	0.000220	0.077560	0.99741	8.936940	0.0041343
1900	0.000200	0.078438	0.99773	8.836888	0.0039571
1901	0.000189	0.078908	0.99751	8.784297	0.0038596
1902	0.000176	0.079510	0.99771	8.717712	0.0037432
1903	0.000169	0.079862	0.99731	8.679289	0.0036729
1904	0.000144	0.081458	0.99543	8.509287	0.0034214
1905	0.000124	0.082944	0.99063	8.356802	0.0032131
1906	0.000092	0.086027	0.98496	8.057321	0.0028091
			Females		
1889	0.000155	0.081577	0.98641	8.496795	0.003553
1890	0.000155	0.081387	0.98804	8.516656	0.003556
1891	0.000143	0.082071	0.98884	8.445668	0.003429
1892	0.000133	0.082695	0.98931	8.382011	0.003311
1893	0.000130	0.082694	0.99000	8.382097	0.003283
1894	0.000138	0.081891	0.99058	8.464258	0.003368
1895	0.000151	0.080857	0.99174	8.572550	0.003492
1896	0.000150	0.080763	0.99288	8.582455	0.003483
1897	0.000170	0.079316	0.99290	8.739075	0.003667
1898	0.000156	0.079982	0.99365	8.666297	0.003531
1899	0.000119	0.082557	0.99397	8.395950	0.003133
1900	0.000097	0.084479	0.99454	8.204922	0.002864
1901	0.000079	0.086450	0.99492	8.017906	0.002613
1902	0.000067	0.087985	0.99494	7.877999	0.002431
1903	0.000064	0.088429	0.99549	7.838484	0.002375
1904	0.000058	0.089420	0.99558	7.751576	0.002269
1905	0.000048	0.091167	0.99439	7.603013	0.002101
1906	0.000034	0.094558	0.99271	7.330418	0.001802

* The *F* test was significant in all cases ($P < 0.01$).

The strong relationship between ω and MRDT (tab. 2) masks however two different patterns of actuarial aging decline over time. In fact, if both measures show that the substantial actuarial aging decrease begins with the cohort born in 1898, the MRDT decline between 1897 and 1906 is nevertheless only 11.9% for males and 16.1% for females, whilst from ω we obtain decreases of 37.9% and 50.8%, which decrease is over three times higher; we are therefore dealing with two very different measures of the changes in late-age mortality.

Fig. 3- Trends in initial mortality rate (A), slope (B) and aging measures, omega (C) and MRDT (D), among men and women for Italian birth cohorts 1889–1906



The corresponding IMR decrease is also very substantial: 66.1% and 79.7% respectively for males and females and this result also lends itself to highlighting the relationship between μ_a and ω. For construction ω is the geometrical mean between γ and μ_a [2]; if we denote by ω^* the arithmetical mean between γ and μ_a, it being known the properties of the means (Hardy, Littlewood, Polya, 1964), and since $\mu_a < \gamma$ (tab. 1), the following relation is worth

$$\mu_a < \omega < \omega^* < \gamma$$

consequently it is μ_a that has the greater weight on ω and the strong relationship between μ_a and ω

indirectly confirms this fact (tab. 2). It should also be considered that in reality, that is, practically from the beginning of 1900 and in the time frame of 10 generations, mortality changes as great as those highlighted by ω could not have taken place in the late ages of these birth cohorts; this result gives rise to doubts as to whether ω can be used as the rate of relative aging in the age group here considered.

Tab. 2 – Pearson's correlation coefficients between Gompertz estimates and aging measures

	IMR (μ_a)	Slope (γ)	MRDT	Omega
		Males		
IMR (μ_a)	1	-0.960	0.973	0.994
Slope (γ)		1	-0.985	-0.999
MRDT			1	0.992
Omega				1
		Females		
IMR (μ_a)	1	-0.973	0.980	0.995
Slope (γ)		1	-0.986	-0.999
MRDT			1	0.989
Omega				1

Finally, with regard to the MRDT trajectory, it should also be noted that while the reduction in mortality for the generations born after 1897 may be in line with the general evolution of the phenomenon, this cannot be said of that which happens to the previous generations. This emerges from a comparison of the average lifetime, especially for women.

6. Mean remaining lifetime after age 85 year

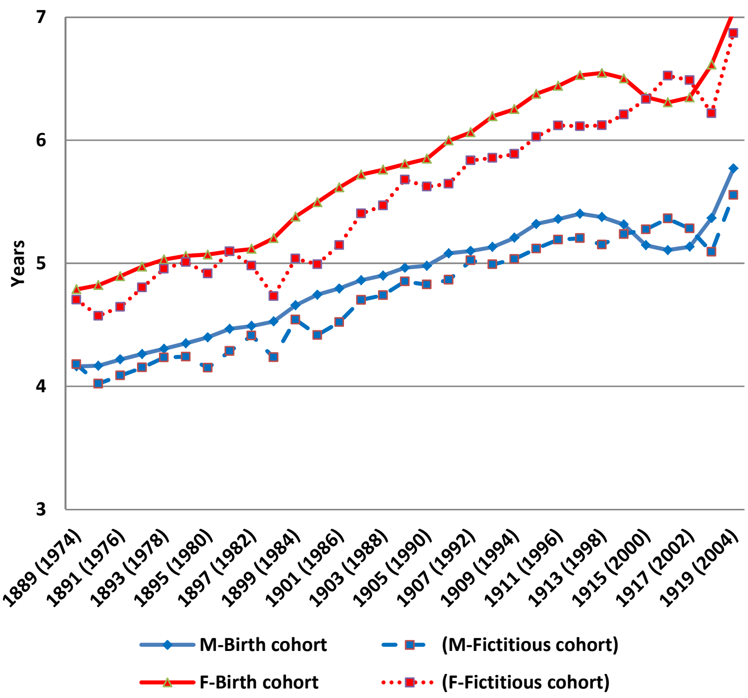
A review of the previous findings concludes by crossing them with those perhaps of the best known indicator of the level of mortality which is the birth cohorts average length of life after age 85 years; it is here indicated by e_{85c} to distinguish it from e_{85p} which is the corresponding period life expectation. We begin with a comparison of the aging measures which concern only the cohorts born between 1889-1906; however as regards just the average length of life, the analysis covers a longer time span.

6.1 Mean lifetime in cohorts born between 1889-1906

As already stated, these birth cohorts are completely extinct and the relative life tables have been reconstructed entirely on the basis of the Istat database. From figures 3-C and 3-D the case of women, whose temporal sequences of aging measures are characterized by two phases, become especially evident: the first presents an initial slight decrease of ω and MRDT followed by a sharp turnaround in tendency between the birth cohort of 1894 and 1897 when the actuarial aging reaches its maximum; the second phase rather is characterized by a continued sharp decrease of the indexes. In the case of males there is only one break in the trend of decrease which is always circumscribed around the generation born in 1897. With regards to women, therefore ω and MRDT show an increase in mortality for birth cohorts from 1894 to 1897, whilst for men this is limited to the generation of 1897. There are no signs of these mortality reversals however, in the trend of the mean life span, but only a gradual slowing down (fig. 4) after an initial growth and this precisely for cohorts born from 1894 to 1897; the average increase in remaining lifetime from one generation to another with respect to those born between 1889-1906 is approximately fifteen days for both sexes.

The comparison between the evolution of e_{85c} and e_{85p} (fig. 4) shows that the cohort effect was lower in respect to that of those born afterwards: their e_{85c} was slightly higher than the corresponding e_{85p} by approximately a month and a half both for males and females; for those born after 1897 however, the increases were much higher and especially for women. The latter in fact benefited more during the last few years of their existence from the progress made in living conditions since the early 80s both with respect to the past and with respect to the men, as shown by the gap with the corresponding period life expectancy (fig. 4). Consequently the gap between mean residual lifetimes has increased and tends to be higher than that which emerges from the comparison of life expectancies.

Fig. 4 – Average number of years actually lived after 85 years from birth cohorts and corresponding remaining life expectancy of fictitious cohorts at the same ages. Italy, males and females



One of the most recent studies (Rogers *et al.*, 2010) shows that the lower incidence of cardiovascular diseases in women, and consequent lower mortality, is due to the positive action of female sexual hormones on blood fat levels; however, this protection ceases after menopause and hence the social and environmental factors, in particular the family, educational and working environments, determine the mortality differences at all ages and these are also indicated as main causes (Maccheroni, 2014). The study of women's life paths who had reached these ages led to identify a further explanation of higher longevity in women: their psychology, which leads them to build social and solidarity networks in a much more efficient way than men do. Thanks to their different way of relating to the external world, women show a higher capacity to adapt to life's changes, to life's various stages and to changing human relationships (Cesa Bianchi, 2000).

6.2 The mean life time in cohorts born between 1907-1919

In order to carry out an analysis of mortality for the cohorts whose range of birth dates 1907-1919, it was necessary to close the related life tables by extrapolating a logarithmic transformation of [1]

at late ages. This is because the high values of the R^2 statistic (tab. 1) suggest that also in their case the regression model fits well, and they validate its use for predictive or forecasting purposes.
The results display another consequence of the war that both both male and female groups have in common: e_{85c} of individuals born between 1915-1918 shows a severe decrease which reaches its minimum with the birth cohort of 1916; thereafter only with respect to those who were born after 1917 does the decrease lessen rapidly (fig. 4). It might also be observed that the men belonging to these cohorts took part in the Second World War in pre-adulthood, so that even in their case the consequences of that event could affect their mortality at older ages; however the perfect synchronism that characterizes the e_{85c} trajectories for both males and females (fig. 4) seems to greatly minimize such consequences.
The bases of these findings are to be connected to the rise in mortality in the extreme age groups that had already been disclosed by the temporal sequences of the probabilities of death given in fig. 2. It's only for the individuals born in 1919 that the average length of life realigns itself on the temporal trend prior to the beginning of the war, and this occurs even though about half of those born during the widespread Spanish flu epidemic which, in Italy in its acute phase, raged between autumn 1918 and early summer 1919[4]; according to the chronicles, in it's early phase the epidemic struck above all the soldiers on the front before spreading nationwide (Melegaro and Alfani, 2010).
These latest findings indicate how a sudden and adverse change in the living and environmental conditions experienced at the time of birth of these generations can have negative repercussions on their mean remaining lifetime (Barker, 1994) even at older ages. Moreover the increase in infant mortality during the years of the conflict, which ceases precisely in 1919, as illustrated in the next tab. 3, provides a further element of proof, albeit indirectly, of the deterioration of living conditions.
In this particular case there was a prolonged deterioration of living conditions. On the contrary, the war of 1911 between Italy and Turkey, which − although involving a considerable number of men and much equipment − was limited in duration, so the increase in infant mortality in that year (tab. 3) was not accompanied by a decrease in the mean remaining lifetime in the ages here examined for the ones born in that period.

Tab. 3 - Deaths before age 1 year per 1.000 live births; Italy, 1910-1920

	1910	1911	1912	1913	1914	1915	1916	1917	1918	1919	1920
Males	146.4	162.7	135.2	145.8	137.2	153.7	174.5	159.2	194.4	134.4	132.1
Females	133.3	150.4	120.4	130.2	123.1	139.7	157.7	146.8	180.1	123.6	120.9

Source: Istat, *L'Italia in 150 anni. Sommario di statistiche storiche 1861-2010*, 2011, Roma.

It should however be noted that, in the past, there have been other cases in which the adverse environmental conditions however, have not led to what has now been highlighted in relation to the war in 1915-'18. A comparison of almost the same ages can be done with the results of a study of the cohorts born in Finland during the great famine of 1866-'68; here the findings have pointed out that their mean remaining lifetime at 80 years of age was not less than that of the birth cohorts born either five years before or five years after the famine (Kannisto, Christensen, Vaupel, 1997). But in our case the war situation and related family and social interactions affected pregnant mothers and those with very young children who found themselves alone whilst their husbands were on the front, in a different way from those who lived during the famine in Finland and so there could also have been this different cohort "effect" on later life mortality (fig. 2).
In the case that we have examined the statistics alone do not allow for speculation on why the pregnancies during the war and the conditions under which the births took place in Italy had such drastic effects on the long-term health and hence on the mean remaining lifetime of these cohorts

[4] Before the Spanish flu epidemic there had been a serious epidemic of cholera in 1884-'85.

(Elo, Preston, 1992).

7. Conclusions

The Gompertzian aging model adapts itself well to the mortality of the birth cohorts that have herewith been examined thus making it possible to perform a comparative analysis of both the role assigned to the parameters and of the aging measures ([2] and [3]) proposed by the actuarial aging or senescence.
With regards to the findings provided by the aging measures across the birth cohorts, some critical issues emerge that reflect negatively on the heuristic component of this type of research, at least in connection with its application to human mortality. The evolution of the model-based estimates of parameter μ_a clearly highlight that for the cohorts born between 1889 and 1900 on the threshold of 85 years of age the negative legacy of environmental conditions determined by war had still not been cancelled out, and such negative effects had affected, albeit in a different selective way, both males and females. Hence it appears that the well known William's hypothesis (1957), according to which "low adult death rates should be associated with low rates of senescence, and high adult death rates should be associated with high rates of senescence"[5] seems to be supported also in this case (tab. 1); in fact, the records show a steady decline of rates of senescence for those who, born after 1900, had not experienced the war high mortality environment at the threshold of the pre-adult age.
The trajectories of the two measures of actuarial aging, ω and MRDT, relating to cohorts born after 1899 highlight however two different reductions in mortality that ω amplifies significantly. These cohorts began to reach age 85 years in 1984, when the fight against cardiovascular diseases, which began in Italy about ten years earlier, continued successfully to reduce mortality in adult and mature age groups; but it was relatively less successful with the elderly (Istat-ISS, 1999). The dramatic reduction of the ageing rate provided by ω is certainly not consistent with the decline of the mortality levels at the ages that are herein considered.
An indirect confirmation of this tendency to amplify the changes in late-age mortality would come from a comparison between e_{85c} and aging measures: in fact, where ω and MRDT show a rise in mortality as in the case of the cohort born in 1897, e_{85c} simply points out a slowdown in growth.
The question is whether today extrinsic mortality still has a role in driving the evolution of aging in low-mortality societies. The social environment has strongly affected lifespan: the improvements in medical care, the prevention of major death causing diseases and the changes in life-style have minimized both the infant and the pre-adult age mortality rate, which is almost exclusively extrinsic and above all caused by road accidents. At advanced ages one far-reaching consequence has been the deceleration of the process of deterioration with age and the related mortality postponing (Vaupel et al., 2010) that has involved a distinction among early, middle and late old age too.
There is however, another great revolution taking place in our societies, the objective of which would not seem to be that of increasing longevity but rather to rid it of disabilities and functional dependence. We refer to the effects that the alliance of medical research, new therapies introduced by biotechnology and advances in molecular nanotechnology and artificial intelligence (www.a4minfo.net) have had on health in general. The effect will certainly not be that of stopping the aging process, but it could however lead to a reassessment of old age with respect to how it is currently considered since these interventions are at the origin of the biological processes of the "natural" deteriorating of the physiological functions. Thus there could be an impact on the mortality pattern at older ages and the assumptions underlying the application of the models used for the study of the actuarial aging could be undermined.

[5] *Evolution*, Volume 11, Issue 4, (Dec. 1957), p. 404.

REFERENCES

ALFANI G., MELEGARO A. (2010), *Pandemie d'Italia*, Egea, Milano.

BARKER D.J.P. (1994), *Mothers, babies, and diseases in later life*. London: BMJ Publishing Group.

BONARINI F. (2009), *Il numero dei centenari in Italia*. Working Paper Series. N.4. Department of Statistical Sciences University of Padua.

CAMERON N., DEMERATH E. W. (2002), Critical periods in human growth and their relationship to diseases of aging, *American Journal of Physical Anthropology* 35(Suppl.): 159 – 184.

CARNES B. A., OLSHANSKY S. J. (1997), "A biologically motivated partitioning of mortality." *Experimental Gerontology* 32(6): 615–631.

CARNES B. A. *et al.* (2006), Mortality partitions and their relevance to research on senescence. *Biogerontology* 7:183–198

CESA-BIANCHI, M. (2000), *Psicologia dell'invecchiamento*, Carocci, Roma.

DEL PANTA L. (1980), *Le epidemie nella storia demografica italiana (secoli XIV-XIX)*, Loescher editore, Torino.

FINCH C. E., PIKE M. C. (1996), Maximum Life Span Predictions From the Gompertz Mortality Model, *Journal of Gerontology,* Vol. 5IA, No. 3, B183-B194.

CARNES B. A., OLSHANSKY S.J. AND GRAHN D.A., (1996), Continuing the Search for a Law of Mortality, *Population and Development Review*, 2:231-64.

ELO I.T., PRESTON S.H. (1992), Effects of early-life conditions on adult mortality: a review, *Population Index*, Summer; 58(2):186-212.

FINCH C. E., PIKE M. C. AND WITTEN M. (1990), Slow mortality rate accelerations during aging in some animals approximate that of humans, *Science* 249:902–905.

GAVRILOV L.A., GAVRILOVA N.S. (2001), The reliability theory of aging and longevity. *Journal of Theoretical Biology*. 213:527–545.

GAVRILOVA N. S., GAVRILOV L. A. (2015), Biodemography of Old-Age Mortality in Humans and Rodents. *J Gerontol A Biol Sci Med Sci.*, 70(1):1-9.

GOMPERTZ B., (1825), On the nature of the function expressive of the law of human mortality and on the new mode of determining the value of life contingencies. *Phil Trans Roy Soc A*.115:513-80.

GURVEN M., FENELON A. (2009), Has actuarial aging "slowed" over the past 250 years? A comparison of small-scale subsistence populations and European cohorts. *Evolution* 63:1017–1035.

HARDY G.H. LITTLEWOOD J.E. AND POLYA G. (1964), *Inequalities*. Cambridge University Press, Cambridge.

KANNISTO V. (1994), *Development of Oldest-Old Mortality. 1950-1990: Evidence from 28 Developed Countries*. Odense University Press.

KANNISTO V., CHRISTENSEN K. AND VAUPEL J.W. (1997), No increased mortality in later life for cohorts born during famine. *Am J Epidemiol*. Jun 1;145(11):987-94.

KIRKWOOD T. B. L. (2015), Deciphering death: a commentary on Gompertz (1825) 'On the nature of the function expressive of the law of human mortality. and on a new mode of determining the value of life contingencies'. *Phil. Trans. R. Soc.* B 370: 20140379. http://dx.doi.org/10.1098/rstb.2014.0379

ISTAT (2011). *L'Italia in 150 anni*. *Sommario di statistiche storiche 1861-2010*. Roma.

ISTAT - *Tavole di Mortalità della popolazione per provincia e regione di residenza*. Anni 1974-2013, Banca Dati: Demografia in cifre http://demo.istat.it/ (data downloaded in January 2014)

ISTAT-ISS (1999, *La mortalità in Italia nel periodo 1970-1992: evoluzione e geografia*. Istat, Roma.

JDANOV D. A., GLEI D. A. AND JASILIONIS D. (2010), A modeling approach for estimating total mortality for Italy during the First and Second World Wars, *Genus*, **LXVI** (No. 1), 17-36.

JUCKETT D.A., ROSENBERG B. (1993), Comparison of the Gompertz and Weibull functions as descriptors for human mortality distributions and their intersections. *Mech Ageing Devel.* 69(1-2):1-31.

MACCHERONI C. (2014), Diverging tendencies by age in sex differentials in mortality in Italy, *South-East European Journal of Political Science*. Vol. II. N° 3, 42-58.
MORTARA G. (1925), *La Salute pubblica in Italia durante e dopo la guerra*. G. Laterza & figli, Bari; Yale University Press. New Haven.
OLSHANSKY S. J., CARNES B. A. (1997), Ever since Gompertz. *Demography.* 34(1). 1-15.
POLLARD H.J. (1991), Fun with Gompertz. *Genus*: 57:1-19.
POLLARD H.J., VALKOVICS E. J. (1992), The Gompertz distribution and its applications *Genus.* 48 (3-4). 15-27.
PRESTON S.H., HIMES C. AND EGGERS M. (1989), Demographic conditions responsible for population aging, *Demography*, 26(4):691-704.
RICKLEFS R. E. (1998), Evolutionary theories of aging: confirmation of a fundamental prediction. with implications for the genetic basis and evolution of life span. *The American Naturalist.*152:24–44.
RICKLEFS R.E. AND SCHEUERLEIN A. (2002), Biological Implications of the Weibull and Gompertz Models of Aging. *Journal of Gerontology:BIOLOGICAL SCIENCES* 2002. Vol. 57A. No. 2. B69–B76
ROGERS R. *et al.*. (2010), Social Behavioral, And Biological Factors, And Sex Differences in Mortality, *Demography*, Volume 47-Number 3, 555–578.
ROSE M. (1991), *Evolutionary Biology of Aging.* New York, Oxford University Press.
SCHAUMANN W. (1993), *Grande Guerra 1915-18*, 5 voll., Ghedina & Tassotti ed. Bassano.
THATCHER A.R., KANNISTO V. AND VAUPEL J.W. (1998), *The force of mortality at ages 80 to 120*. Odense University Press. Denmark.
TULJAPURKAR S.D. (1997), "Theoretical Perspectives on the Evolution of Senescence", in *Biodemography of Aging.* edited by K. Wachter and C. Finch. Washington. D.C.. National Academy Press.
VAUPEL J. *et al.* (2010), Biodemographic trajectories of longevity. *Nature* 426: 536–542.
WILLIAMS G. C. (1957), Pleitropy, natural selection and the evolution of senescence, *Evolution,* 11: 398–411.

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