## The future of longevity in Switzerland : background and perspectives

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## Executive summary

1. In many developed countries, including Switzerland, the ongoing increase in life expectancy is driven by the mortality decline among older persons. This has important consequences for both the provision of health care and the management of pension funds.
2. In this context, the Swiss Federal Office of Statistics mandated a small group of experts to provide a critical review on the future evolution of mortality in developed countries. The report starts with an analysis of the past trends in life expectancy (see $\S 21$ and ff, page 6) and in healthy life expectancy (see $\S 56$ and ff, page 16). Longevity is defined here as the duration (or the length) of life as observed in population or in individuals. The oldest and still most used indicators of longevity are life expectancy at birth $\left(\mathrm{LE}_{0}\right)$ at a population level, and maximum life span (MLS) at the individual level. A discussion on the future evolution of mortality and health is then presented (see $\S 93$ and ff, page 23). A set of recommendations is finally proposed (see §173 and ff, page 33).

## Past and current trends in longevity and health

3. In Switzerland, life expectancy at birth ( $\mathrm{LE}_{0}$ ) continues a steady increase that began at the end of the 19th century, while LE 65 showed an increase since the 1950s. As in other countries, there is no clear sign that the rise in $\mathrm{LE}_{0}$ might come to an end, although the yearly increase is slower since the 1980s.
4. The modal age at death, which can be seen as the "usual" mean longevity at a given time, increased steadily since the 1920s. Among the adults, the variability of age at death decreased during the last fifty years, compatible with a compression of mortality.
5. There is however no fate dictating that $\mathrm{LE}_{0}$ should continue on the same line. A plateau of $\mathrm{LE}_{0}$, or even a decline, has been observed in several European countries, probably related to crises affecting the socio-economical environment, the life styles (e.g., alcohol and tobacco use), or the supply of health services.
6. Healthy life expectancy, as measured by the number of years spent without disability (DFLE), increased during the last fifty years, due to a decrease of agespecific disability prevalence. However, international trends in DFLE are heterogeneous, with some countries showing stability or even decrease in the proportion of years spent without disability after age 65 . Whether these results indicate real variation or differences in measurement of disability is still undetermined.

## Future evolution of longevity and healthy life expectancy

7. Most projections of LE use historical trends in mortality rates or life expectancy as an input. Such an approach is based on the remarkable stability of the changes over the past 200 years. However, most projections based on past trends consistently underestimated the increase in LE.
8. Epidemiological approaches use information on the determinants of mortality by cause of death (e.g. smoking, obesity...) ; they provide results that are not very different from those of extrapolative methods.
9. In summary, most methods predict that LE ${ }_{0}$ will increase up to 2050 in men as in women in industrialized countries such as Switzerland, with estimates ranging from five to nine years adding to the current figures.
10. Previsions of healthy life expectancy rely on projected total life expectancy, combined with projections of the prevalence of disability. Available estimates predict a longer DFLE in Switzerland. These previsions are often criticized because they fail to capture the complexity of the determinants of disability and the difficulty to interpret any trend (e.g., the development of new assistive technologies might have contributed to the decline in reported disability).
11. According to epidemiological projections, major upheaval in health would have a limited impact on health-adjusted life expectancy : in the absence of 20 major risk factors for diseases and injuries, the health-adjusted life expectancy would increase by about 5 years as compared to the current situation in 2000.

## Recommendations

12. There is currently no definite argument in favour of a specific method of prevision. Thus, we recommend to provide estimates based on several methods, e.g., (i) a method using a set of observed lowest mortality rates at a given time, (ii) a method using the epidemiological projections of the determinants of mortality (e.g., smoking, diet, etc.), and (iii) a method estimating the effect of the projected evolution of education and socio-economic status on the future mortality rates.
13. When using historical periods to establish future trends, the historical period should be at least as long as the projection period. Further, the mortality experience of other populations should be assessed, and cohort effects should be carefully considered.
14. A closer monitoring of longevity is needed. This means that mortality rates of the oldest-old should be followed up not only with cross-sectional estimates, but also with appropriate cohorts.
15. Another aspect is to rethink the system of cause of death because of its currently low information content.
16. Finally, in order to develop methods using health related information, epidemiological knowledge on the oldest population should be improved through health surveys, which should also include those living in institutions.
17.A committee on the future of longevity and healthy life should be set up to further address these issues.

## Introduction

18. In many developed countries ${ }^{1}$, including Switzerland ${ }^{2}$, the ongoing increase in life expectancy is driven by the mortality decline of the old and the very old persons. Further, because the main causes of death are related to degenerative diseases (cardiovascular and cancer are responsible of $60 \%$ of causes of death in people aged more than 60), the determinants of mortality take place in the adult life, or even before ${ }^{3}$.
19. In this context, the Swiss Federal Office of Statistics decided to mandate a small group of experts to summarize the current information on past trends in mortality and longevity, as well as on their future evolution in Switzerland and other industrialized countries. The scope of this report is to provide a critical review of the literature on the future evolution of mortality in developed countries and some suggestions regarding the most promising approaches of projections. This report will also address the health of the older persons, in the perspective of the healthy life expectancy.
20. The first two chapters start with a couple of definitions, then with an analysis of the past trends in life expectancy and health indicators. The future evolution of mortality and health are presented in the two next chapters. A set of recommendations is finally developed, on the strategies of and on what is needed in term of information for monitoring and projecting longevity.

## Past and current trends in mortality and longevity

## Definitions

21.Longevity is defined here as the duration (or the length) of life as observed in population or in individuals. The oldest and still most used indicators of longevity are life expectancy at birth ( $\mathrm{LE}_{0}$ ) at a population level, and maximum life span (MLS) at the individual level.
22. Several other indicators have been proposed to overcome the limitations of both $\mathrm{LE}_{0}$ and MLS. They allow examining more closely the patterns of old age mortality. These indicators are increasingly used, including in this report. They are briefly presented below.
23. Indicators of longevity:

- Life expectancy at birth (LEO), or at any age $x(L E x)$ : An estimate of the average number of additional years an individual could expect to live if the age-specific death rates for a given year prevailed for the rest of his life ${ }^{\text {a }}$. It is the mean duration of life in a population at a given calendar time. LEO is still a popular indicator of longevity ${ }^{4-9}$, but it is sensitive to early mortality, in particular to child and infant mortality. This is a drawback when late mortality is the driving force of longevity ${ }^{10-12}$.
- Median age at death (Med) : age that separates the younger half of the deceased from the older half. Med is usually given as the average value of the yearly median ages within a period of study.
- Modal age at death $(M)$ : the most common age at which adult people die (i.e. age with the maximum number of deaths) at a given period. M is sometimes difficult to estimate because of the flatness around the modal area ${ }^{13}$, and smoothing techniques are in general needed. M is a companion indicator of the "normal life duration", a concept introduced by Lexis in $1878{ }^{14}$. Briefly, the concept distinguishes three parts in the mortality of a population : (i) one part related to infant and child deaths, i.e., premature mortality, (ii) another part related to aging-related deaths (i.e., the late mortality or the "normal" deaths) and (iii) an intermediate part where premature and agingrelated deaths overlap. In this perspective, M was viewed by Lexis as the central value around which aging-related deaths are normally distributed (i.e., the mode is an estimate of the arithmetic mean of the normal life duration), although this is wrong, as mortality is skewed towards younger ages, with the mode higher than the mean. According to this hypothesis, the right-hand side of the distribution of age of deaths (deaths occurring after M) gives the parameters of a normal distribution centred on M (i.e., the lefthand side of the distribution is constructed by symmetry of the right-hand side). Kannisto later suggested that M and the standard deviation above it provide a good account of longevity under a given mortality regime ${ }^{15}$.
- Maximum life span (MLS) : the maximum number of years a member of a population has been observed to survive, at a given period. MLS has the advantage to be based on straightforward information, that may however be of poor quality, due to the inaccuracy of reported ages of very old persons. Also, as a single event, it depends on the size of the population under study, although this might not be the major determinant of change in MLS. In Sweden for example, MLS increased from 101 years to 108 years between the 1860s and the 1990s. Of this increase, $72.5 \%$ is attributable to a decline in mortality above age 70 , only about $12 \%$ to the increasing size of successive birth cohorts, and about $16 \%$ to mortality reductions below age 70.
- Life endurancy : the age reached by 1 per 10 '000 (or 1 per 100'000) individuals of a cohort.
- Number of deaths at age 100 and over: as the absolute number of deaths occurring at age 100 and over, this indicator also depends on the size of the population.
- Number of centenarians/supercentenarians : number of persons aged 100 and over, respectively 110 years and over, at a given period in a population. It also depends on the size of the population.
- Centenarian rate $\left(C R_{x}\right)$ : incidence of centenarians in a given cohort, estimated by the number of survivors aged 100 at a given date divided by the size of the corresponding cohort at age x (for example: $\mathrm{CR}_{80}$ is the number of centenarians observed in a cohort of people alive 20 years before, i.e. when they were aged 80 years).

24. Indicators of concentration, verticalization and rectangularization:

- Rectangularization of the survival curve : pattern of mortality characterized by an important proportion of persons surviving until old age and dying over a short period of time (i.e., with a compression of mortality within a short period).
- Standard deviation above the mode $\left(S D_{(M+1)}\right.$ : measures the dispersion of the deaths occurring above the modal age at death. When $\mathrm{SD}_{(\mathrm{M}+)}$ decreases, then the survival curve becomes more rectangular.


## Longevity and mortality

25. Most of the available studies are based on population census and vital statistics, i.e., on an information system available only since the mid-19th century in the developed countries.
26. A substantial amount of information is provided by demographic history ${ }^{16}$ on the pre-modern patterns of mortality, including in Switzerland. Although this literature will not be commented here, at least one observation deserves attention. In Europe, there is a slow and steady increase of the life expectancy at birth ( $\mathrm{LE}_{0}$ ) since the Middle-Age, i.e., well before the sanitary and hygienic reforms of the late $19^{\text {th }}$ century ${ }^{17}$, possibly in relation with factors such as diet, infectious environment, etc... This raises the possibility that future changes in mortality might be determined by unexpected changes of currently unknown factors.
27. Although $\mathrm{LE}_{0}$ is not the most appropriate indicator in countries where mortality is low among young and middle-aged people ${ }^{18}$, it is still the most widely used indicator of longevity, and hence, the most widely available. It also has the advantage of being easily interpreted. The paragraphs below provides an analysis of the recent evolution.
28. Figure 1 shows that $\mathrm{LE}_{0}$ has increased steadily in Switzerland since 1876, from about 40 and 42 years for men and women, respectively, to the current values of 79 and 84 years ${ }^{2}$. In other words, $\mathrm{LE}_{0}$ almost doubled in both genders, with a yearly increase amounting to about 4 or 5 months from 1876 until 1950, slightly faster among women than among men. It then dropped off to a slower rhythm, about 3 months per year, during the period 1950-2000.
29.The increase in $\mathrm{LE}_{0}$ was first attributable to the decline in infant and childhood mortality (which probably started in the first half of the 19th century).
30.In the 1950s, a new pattern of evolution emerged : (i) $\mathrm{LE}_{60}$ and $\mathrm{LE}_{80}$ began to increase in both gender, and (ii) a gender gap in $\mathrm{LE}_{0}$ appeared and widened, with women gaining more years of $\mathrm{LE}_{0}$ than men. This gap is partially attributable to causes of death (e.g., cardiovascular or cancer) which are sensible to lifestyle habits (e.g., smoking or alcohol use). The gap peaked in the 1970s. Since the 1980s, the gender gap diminished, partially due to the evolution of lung cancer mortality, much more favourable among men than among women ${ }^{19}$.


Figure 1. Life expectancy at birth ( $\mathrm{LE}_{0}$ ), at age $60\left(\mathrm{LE}_{60}\right)$, and at age $80\left(\mathrm{LE}_{80}\right)$, by gender. Switzerland, 1876-2000 (Source: Robine J-M, Paccaud F. $2005{ }^{\text {² }}$ )
31. Figure 2 shows the evolution of both the modal age at death $(\mathrm{M})$ and the maximum age at death (MLS). The rising trends of both indicators is less marked than the one of $\mathrm{LE}_{0}$. M increased from 70 to 84 years for men, from 70 to 88 years for women.
32. M started to increase well after $\mathrm{LE}_{0}$. Before the 1920 s, M did not change, fluctuating between 70 and 75 years. In women, M increased at a rhythm of two months per year, from 75 years in 1921-1925 to almost 90 years in 2001-2005. In men, M increased at a slower pace (less than half a month per year) from the 1920s until 1946-1950. It then ran in parallel with the women. Overall, the pattern of increase is similar across genders, although faster for women.
33. In both gender, there is a secular decline of the difference between M and $\mathrm{LE}_{0}$, from 30 years (about $70 \%$ of $\mathrm{LE}_{0}$ ) to 6 years (about 7\%), related to the decline in children mortality that previously had a major impact on $\mathrm{LE}_{0}$.
34. The same Figure 2 shows the increase in MLS, with an acceleration from the 1950s. MLS was about 102 years between 1880 and 1920, then reached 104 years between 1920 and 1960. It currently reaches 110 years, with higher values for women than for men. The pattern of increase is similar across genders.


Figure 2. Secular trends in modal age at death (M) and in maximum life span (MLS, in both genders. Switzerland, 1876-2002. (Source: Cheung SL, Robine J-M, Paccaud F, et al., personal communication)
35. Another indicator reflecting the decline in old age mortality is the number of deaths occurring at age 100 and over, that began to increase in the 1950s, as shown in Figure 3. Although there is a large difference in the absolute numbers of male and female centenarians, the increase rate is similar across genders, as shown in the insert of the same Figure 3.


Figure 3. Number of deaths at age 100 and over, by gender, Switzerland, 19402000 (Insert shows the same data, with the number of deaths on a $\log$ scale). (Source: Robine J-M, Paccaud F. $2005{ }^{2}$ )
36. Table 1 disentangles the determinants of the increase in centenarians, from the cohort 1870 to the cohort 1900. For females, the number of centenarians has increased by a factor of 12.5 . One part is due to the increase in the cohort size by a factor of 1.2. The remaining effect is attributable to the mortality drop, which is broken down into (i) an effect of early mortality (between birth and age 80), and (ii) an effect of late mortality (between 80 and 100). The multiplicative model for the increase in centenarians thus includes three factors of change (size of the birth cohort and two elements of mortality) to determine the increase in female centenarians $(1.2 * 2.3 * 4.5=12.5)$. According to this, the drop in mortality after 80 explains more than half of this increase, the drop in mortality from birth to 80 explains another third.
37. Similar proportions can be found among males, as shown in the same Table 1.

|  | Males |  |  |  | females |  |  |  |
| ---: | ---: | ---: | ---: | ---: | ---: | ---: | :---: | :---: |
|  | 1870 | 1900 | factor | 1870 | 1900 | factor |  |  |
| Cohort sizes |  |  |  |  |  |  |  |  |
| • At birth | 40,570 | 48,223 | 1.2 | 38,638 | 46,093 | 1.2 |  |  |
| • at 80 | 4,086 | 9,065 | 2.2 | 6,185 | 17,304 | 2.8 |  |  |
| • at 100 | 9 | 53 | 6.2 | 23 | 294 | 12.5 |  |  |

Table 1. Determinants of the increase in the number of centenarians: cohort size at selected ages and increase factors. Switzerland, 1870 and 1900 (Source:
Robine J-M, Paccaud F. $2005{ }^{\text {² }}$ )
38. As a result, the number of people aged 100 and more increased rapidly, more than doubling every ten years. Note that the centenarians doubling time shortened over time. This is shown in Figure 4.


Figure 4. Number of people aged 100 and more. Switzerland, census data, 18602000. (Source: Robine J-M, Paccaud F. $2005{ }^{2}$ )
39. Table 2 shows the centenarian rates at birth $\left(\mathrm{CR}_{0}\right)$ and at $60\left(\mathrm{CR}_{60}\right)$ (see definition on $\S 23$ above). $\mathrm{CR}_{0}$ and $\mathrm{CR}_{60}$ increased in both genders. Overall, $\mathrm{CR}_{0}$ varied from 1.5 centenarians per 10,000 births in 1860 (.8 for males, 2.2 for female) to 38.6 in 1900 (11.6 for males, 66.8 for females). The increase was more marked among females.
40. As expected, values are higher for $\mathrm{CR}_{60}$, but follow a similar pattern: $\mathrm{CR}_{60}$ increases from 2 to 22 for males, from 5 to 100 for females. A slowing down in the increase of CRs is apparent since the 1980s.
41.The advantage of females over males is striking: female $\mathrm{CR}_{0}$ is always larger than male $\mathrm{CR}_{60}$, and this advantage increases over the period. The same Table 2 shows that the ratio $\mathrm{CR}_{60} / \mathrm{CR}_{0}$ is always larger among males than among females, suggesting that the selective effect of early mortality is stronger for males than for females. A further point to note is that, in both genders, the ratio $\mathrm{CR}_{60} / \mathrm{CR}_{0}$ decreases over the period : this is compatible with a stronger decrease in mortality after the age of 60 than before this age.

|  | $C R_{0}$ |  | $C R_{60}$ |  | Ratio $C R_{60} / C R_{0}$ |  |
| :--- | :---: | :---: | :---: | :---: | :---: | :---: |
|  | males | females | males | females | males | Females |
| 1960 | 0.8 | 2.2 | 2.2 | 4.8 | 2.8 | 2.2 |
| 1970 | 2.2 | 6.5 | 5.7 | 13.7 | 2.6 | 2.1 |
| 1980 | 5.6 | 16.3 | 13.5 | 32.1 | 2.4 | 2.0 |
| 1990 | 6.2 | 38.2 | 13.0 | 62.6 | 2.1 | 1.6 |
| 2000 | 11.6 | 66.8 | 21.6 | 99.3 | 1.9 | 1.5 |

Table 2. Centenarian rates for cohorts at birth ( $C R R_{0}$ ) and at age $60\left(\mathrm{CR}_{60}\right)$. Switzerland, centenarians at census 1960, 1970, 1980, 1990 and 2000. (Source: Robine J-M, Paccaud F. $2005{ }^{\text {² }}$ )
42.There are several ongoing studies on the mortality trends in older people in Switzerland. In general, the observations are compatible with a compression of mortality ${ }^{20}$. This was already visible in an earlier work, suggesting a narrowing of the distribution beyond the mean age at death ${ }^{21}$. This can be seen on Figure 5, showing the evolution of the pattern of distribution of age at death for four periods in women.


Figure 5. Distribution of age at death: empirical (dots) and fitted densities (lines). Women, Switzerland, four periods (from left to right: 1876-80, 1906-10, 1956-60 and 2001-2) (Source: Cheung SL, Robine J-M, Paccaud F, et al., personal communication)
43. Demographic information show a continuing decrease of mortality even in low mortality populations, suggesting that there is still further room for improvement in mortality and life expectancy. In other words, the current pattern is a compression of mortality occurring around an increasing mean age of death.

## Other countries

44. Figure 6 shows LE $\mathrm{D}_{0}$ as reported for 2005 in selected countries, including Switzerland for comparison purposes. Two observations are worth mentioning. First, in all countries, there is a substantial gap between women and men. Although biological differences make the gender gap likely to persist, this variability can be interpreted as a possible room for improvement for males. Second, even among the developed countries displayed in this Figure, there is a gap of about 6-7 years in LE $\mathrm{L}_{0}$. Again, these gaps are important because they quantify possible gains for those with the low values of $\mathrm{LE}_{0}$.


Figure 6. Life expectancy at birth ( $L_{0}$ ) in selected countries, by gender. OCDE, 2005 (www.oecd.org).
45. Comparing longevity between countries is difficult prior to the $20^{\text {th }}$ century, due to the scarcity of credible data. In general, most European countries show a pattern similar to the one observed in Switzerland, often with a gap of a couple of years : $\mathrm{LE}_{0}$ is increasing since the middle or the end of the 19th century, and continued to increase throughout the $20^{\text {th }}$ century (see Figure 9, page 36 and Figure 10, page 36).
46. With $\mathrm{LE}_{0}$ reaching 75 and 85 years in men and women, respectively, Japan is enjoying the highest $\mathrm{LE}_{0}$ worldwide in 2002.
47.In the US, trends in $\mathrm{LE}_{0}$ from 1900 to 2000 also show a steady increase, however less marked since the 1980s ${ }^{22}$. As compared to the highest figures recorded, US LE 0 was 10 years shorter in the early $20^{\text {th }}$ century, and then converged to the highest life expectancies, with a current deficit of one year in $2000{ }^{7}$. LE $_{0}$ increased from 68.2 years in 1950 to 77.8 years in 2004. $\mathrm{LE}_{65}$ and $\mathrm{LE}_{85}$ increased from 13.8 years and 4.7 years, respectively, in 1950 to 18.7 years and 6.8 years, respectively, in $2004{ }^{23}$.
48. A closer look to European data suggests that the evolution of European $L_{0}$ followed a pattern common to all countries until the 1940s and the 1960s (Source: European Health Expectancy Monitoring Unit, www.ehemu.org). After that, the evolution of $L E_{0}$ followed three specific patterns (see Figure 11, p. 37, and Figure 12, p.38) :

- a group of "high convergence" countries (which includes Switzerland), in which the LE 0 did increase steadily (i.e., without slowing down during the 1960s), resulting in the currently highest LE L in $_{0}$ Europe (83-84 years for women, 78-79 years for men);
- a group of "low convergence" countries (which includes England and Wales, Belgium) which is characterized by a slowdown in the $\mathrm{LE}_{0}$ increase during the 1960 s. $L_{0}$ then converged in the 1970 s to a value which is about two years below the $\mathrm{LE}_{0}$ reached by the "high convergence" countries.
- a group of "divergent" countries, which include most of the Eastern Europe countries, but also Denmark, Norway and the Netherlands, show a plateau or even a decrease in $\mathrm{LE}_{0}$.

49. These three patterns of evolution result in significant differences in $\mathrm{LE}_{0}$ between European countries, reaching a maximum of twelve years for men and seven years for women in 2002-2003 (when comparing countries of the Eastern Europe and countries like France or Switzerland). Most recent data suggest that the divergence is enduring. In the Netherlands, for example, an unexpected and sudden reversal in old-age mortality decline occurred around 1980, leading to a decrease in life expectancy in men, and to a plateau in women.
50. The reasons underlying the make-up of these three groups are unclear. They are likely to be related to crises in the social and health environments, perhaps common among the countries belonging to the same group of pattern of evolution ${ }^{24 ; 25}$. As noted by Bongaarts ${ }^{26}$, this divergence in Western Europe is largely due to differences in smoking mortality. Regarding the Netherlands, trends in smoking mortality are a partial explanation for the stagnation in mortality decline, and other factors are difficult to identify, as their relation to specific causes of deaths is unclear ${ }^{24}$. Moreover, inaccuracies and secular changes in the coding of causes of deaths make these analyses complex. Several detailed analyses of the situation have been produced in the Eastern countries ${ }^{27-29}$, including by an international group based in Warsaw ${ }^{\mathrm{b}}$. Analysis of causes of death are not very useful, because of the poor quality of the data for deaths occurring after 80 years ${ }^{30}$.
51. The trends in $\mathrm{LE}_{65}$ are shown in Figure 13, p. 39 and Figure 14, p. 39. In most European countries, the decrease in old age mortality became obvious from the 1950s onwards. Globally, LE 65 showed a one-year increase between 1996 and 2002. In 1996, the largest differences between European countries reached five years in both genders.

[^0]52.The modal age at death (M) was fluctuating between 65 and 75 years throughout the $18^{\text {th }}$ and $19^{\text {th }}$ century, and began to increase steadily from the end of the $19^{\text {th }}$ century. Since the 1960s, M tends towards a value of 90 years for women in most European countries.
53. The higher M in female is observable from the beginning of the $20^{\text {th }}$ century and reached a maximum in the 1970s. Since then, M also increases in men, although not enough to reduce the gender difference.
54. Sweden provides data on maximum life span (MLS) since 1861. There is a clear upward trend (more pronounced after 1969), increasing by 1.1 year per decade ${ }^{11 ; 31}$. Similar trends are observed in other European countries and in Japan 11;32-34. Part of this trend is attributable to the decrease of mortality in the oldest-old, adding to the larger number of survivors to old age and to larger birth cohorts ${ }^{11}$.
55. In Japan, from 1950 to 2004, M increased steadily from 73.7 years to 84.7 years in men, from 78.0 to 90.2 years in women ${ }^{35}$, i.e., 2.5 months per year. There is no sign of a slowing down for the increase of M , but the standard deviation above the mode $\left(\mathrm{SD}_{(\mathrm{M}+1)}\right)$, a measure of the distribution of death, decreased steadily until the 1990s in men and the mid-1980s in women. The $\mathrm{SD}_{(\mathrm{M}+)}$ then reached a plateau. The number of death occurring at and above the mode continued to increase, but at a slower pace than in the mid-seventies. These observations are compatible with a compression of mortality, that still continues but at a much slower pace, as $\mathrm{SD}(\mathrm{M})+$ remains constant.

## Past and current trends in healthy life expectancy

56. To improve the understanding of the report, the following definitions of the indicators of healthy life are given:

- Healthy life expectancy (HLE) : average number of years an individual is expected to live in a specific health state. There are many possible dimensions of health, therefore many different health expectancies. In practice, HLE is often used as a generic name for the indicators combining mortality and health status.
- Health-adjusted life expectancy (HALE) : Average number of years that a person can expect to live in "full health" by taking into account years lived in less than full health due to disease and/or injury c.
- Disability-free life expectancy (DFLE) : average number of years an individual is expected to live without disability, i.e. without limitation in daily living activities such as bathing, dressing, walking, etc. DFLE combines agespecific mortality with age-specific prevalence of disability (more rarely, with age-specific incidence of disability). Like LE, DFLE may be computed at each age x .
- Ratio DFLEx/LEx : ratio of disability-free life expectancy over total life expectancy, reflecting the proportion of remaining years (after age x ) that are spent without disability.

[^1]57. $\mathrm{LE}_{0}$ is a relevant health indicator as far as morbidity is dominated by rapidly lethal diseases. The increasing prevalence of degenerative and chronic diseases means that an increasing proportion of the population will suffer from diseases with a low risk of dying from them. Further, an important proportion of the population will suffer from disability from degenerative diseases ${ }^{36}$.
58. The relationship between longer life expectancy and health has been examined by Fries ${ }^{37}$, who considered three scenarios of evolution ${ }^{38}$ (see Figure 7). According to the paradigm of compression of morbidity, the average age at first chronic disease or disability is postponed (III in Figure 7): if this postponement is larger than the increase in life expectancy, people will gain years of healthy life or, in other words, the period of morbidity from chronic diseases will be compressed at the end of life.
59. Another scenario (I in Figure 7) postulated a longer life with a constant age at the beginning of morbidity, i.e., an expansion of morbidity. Finally, the scenario II on Figure 7 assumes that the age at death increases in the same amount as the increase of the age of the incidence of degenerative disease : this is simple shift towards a longer life with a stable duration of unhealthy life.


Figure 7. Possible scenarios for future morbidity and longevity. (Source : Fries JF, $2005{ }^{38}$ )
60. In this context, new indicators have been developed since the mid- $20^{\text {th }}$ century, in order to take into account not only the years of life enjoyed by the population, but also the quality of life ${ }^{39}$. The concept of health expectancies was first proposed by Sanders in $1964{ }^{40}$, and then formalized by Sullivan who proposed a method to compute health expectancies ${ }^{41}$. This method is based on the age-specific prevalence of healthy and unhealthy states (data gathered in cross-sectional health surveys) combined with age-specific mortality (from standard period life table).
61. An alternative, less convenient, approach is the multi-stage method, that uses incidence of health states instead of prevalence data and thus requires longitudinal observation to provide the transition rates between health states.
62. Many researchers have commented on the differences between the methods. Both seem equivalent when transition rates are smooth and regular over time ${ }^{42}$. The Sullivan method can be generally recommended for its simplicity, its relative accuracy and the ease of interpretation.
63. As disability often represents the cumulative consequence of chronic diseases ${ }^{43}$, disability-free life expectancy (DFLE, see also $\S 56$ above) is a commonly used indicator of healthy life. This concept further allows to split life expectancy into years lived with and without disability. The assessment of aging populations commonly uses DFLE 65 in absolute number and as a proportion of LE 65 . DFLE has been criticized as not incorporating information related to the severity of disability.
64. In contrast to mortality, information on disability has been collected over a shorter period, mainly from the 1980s, and in fewer countries. Comparing disabilityrelated figures is therefore difficult, also because of differences in the definitions used (based on the presence of any incapacity in activities of daily living, for example in Katz' scale ${ }^{44}$, or in instrumental activities such as walking outside home, shopping, preparing meals).
65. Another problem is the difference in sampling : typically, the prevalence of disability is underestimated when measured only in community-dwelling persons, i.e., omitting institutionalised residents who are at higher risk of being disabled. Note that the Swiss Health Survey (an interview survey on a Swiss-wide sample of 20'000 community-dwelling persons since 1992) will include a sample of institutionalized persons from the 2007-8 survey on. More generally, the classical selection bias affecting all health surveys, with a disproportionate number of disabled persons among non-participants, is especially important in aged population, where the prevalence of disability is high.
66. Figure 8 displays DFLE $_{0}$ in year 2002 in various countries. In all countries, DFLE $_{0}$ is higher among women than among men, although the difference is less striking than for $\mathrm{LE}_{0}$.


Figure 8. LEo by gender, with disability (DLEo) and without disability (DFLEo). Selected countries, 2001 (Source: Mathers $2003{ }^{45}$ )
67. In Switzerland, between 1981 and 1997, the increase in DFLE $0_{0}$ paralleled the increase in $\mathrm{LE}_{0}{ }^{46}$. This increase was observed in both genders (see Table 3). The simultaneous increase in both DFLE $_{0}$ and $\mathrm{LE}_{0}$ suggests that the population is enjoying an "healthy ageing", i.e., ageing means here adding both years to life and life to years.
68. $\mathrm{LE}_{65}$ and DFLE $_{65}$ also showed an increase, although the gender difference is more marked : the proportion of years spent without disability (ratio DFLE $65 / \mathrm{LE}_{65}$ ) varies according to the gender. Between 1981 and 2002, a compression of morbidity is observed in men as in women : the ratio DFLE $_{65} /$ LE $_{65}$ increased from $79 \%$ to $83 \%$ for men, from $66 \%$ to $75 \%$ for women, in a context of strong increase of $\mathrm{LE}_{65}$ in both gender, from 14.6 to 17.5 years and from 18.5 to 21.1 years, for men and women respectively.
69. Both the 2002 figures and the evolution of the compression of morbidity between 1981 and 2002 vary between the genders. Between 1981 and 1997, women gained years of life without disability and experienced a decline in the number of disabled years : the proportion of years with disability significantly thus decreased in women, an evolution compatible with a compression of morbidity.
70. During the same period, men gained 15 months without disability and 6 months with disability 46 : compression of morbidity was thus relative in men, as they gained some time with disability, although in a lower proportion than the time without disability. Moreover this compression occurred later than the one observed for women.
71. Although women have a longer $\mathrm{DFLE}_{0}$ than men and gained more disability-free years, the absolute number of years spent with disability is higher in women. In 2002, $\mathrm{LE}_{0}$ was 77.8 in men and 83.1 in women, and DFLE $_{0}$ was 73.7 years in men and 76.8 years in women : thus, on average, men would spend about 4 years in a disabled state, versus 6 years for women ${ }^{47}$.

|  | Men |  |  |  | Women |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $L E_{65}$ | DFLE65 | DLE65 | DFLE65/LE65 | $L E_{65}$ | DFLE65 | DLE65 | DFLE 65/ $^{\text {L }}{ }_{\text {E65 }}$ |
| 1981/82 | 14.6 | 11.5 | 3.1 | 79\% | 18.5 | 12.2 | 6.3 | 66\% |
| Change 1981-1997 | + 2.1 | + 1.5 | + 0.6 |  | + 2.1 | +4.1 | -2.0 |  |
| 1997/99 | 16.7 | 13.0 | 3.7 | 78\% | 20.6 | 16.3 | 4.3 | 79\% |
| Change 1981-2002 | + 3.5 | + 3.1 | - 0.2 |  | + 2.6 | + 3.7 | - 1.1 |  |
| 2002 | 17.5 | 14.6 | 2.9 | 83\% | 21.1 | 15.9 | 5.2 | 75\% |

Table 3. Evolution of LE $_{65}$ and DFLE 65 , by gender. (Source: Höpflinger ${ }^{46}$, OFS 47 and author's calculations)

## Other countries

72. Caution should be applied when comparing healthy life expectancies among different countries, because of the quality of the data often varies, as well as the methods used to assess healthy life expectancy.
73.The OECD examined the trends in disability at age 65 years and over resulting from national health surveys in 12 countries. There was a clear decline in disability in 5 countries (Denmark, Finland, Italy, The Netherlands, the USA), two showed stability (Australia, Canada) and three (Belgium, Japan, Sweden) showed an increase. More importantly, the OECD study shows that the prevalence of disability in activities of daily living ranges from a low of 7\% in The Netherlands to a high of $18 \%$ in the United Kingdom, suggesting differences in the assessment of disability as a partial explanation for these differences ${ }^{48}$.
73. The European Community Household Panel provides data and indicators to monitor the trends of DFLE between 1995 and 2003. In men, the gain in $\mathrm{LE}_{0}$ was larger than the one in $\mathrm{DFLE}_{0}$. The pattern is similar in women, though the changes are smaller. This suggests an expansion of morbidity rather than the compression as observed in Switzerland.
74. However, when $\mathrm{LE}_{65}$ and DFLE $_{65}$ are analysed, the gains are similar for both indicators (see Figure 15, p. 40), like in Switzerland. The gender-specific secular trends of the ratio $\mathrm{DFLE}_{65} / \mathrm{LE}_{65}$ are used in Table 4 to characterize each EU country, i.e., if the evolution suggests (i) a compression of morbidity (defined as a relative change $>=5 \%$ ), (ii) an expansion of morbidity (relative change $<=-5 \%$ ), or (iii) a stability ( $-5 \%<$ relative change $<5 \%$ ).
75. Table 4 shows a diversity of evolution among these countries. Further, there is no strong correspondence across genders, i.e., the same country shows different evolution according to the gender. These differences cannot be explained, and might be attributable to methodological and cultural differences, but also to changes in institutionalization rates (as the study excludes institutionalised persons).

| DFLE65/LE65 (in \%) trends ... | Men | Women |
| :---: | :---: | :---: |
| ... decrease by 5\% or more (expansion of morbidity, scenario I in Figure 7, p. 17) | Denmark <br> Netherlands <br> Portugal <br> Sweden <br> UK | Germany <br> Greece <br> Ireland <br> Netherlands <br> Portugal |
| ... is constant (stability, scenario II in Figure 7, p. 17) | France <br> Greece Ireland Spain | Austria Denmark Finland France Spain UK |
| ... increase by 5\% or more (compression of morbidity, scenario III in Figure 7, p. 17) | Austria Belgium Finland Germany Italy | Belgium Italy <br> Sweden |

Table 4. Distribution of countries among categories of secular trends in the ratio DFLE $_{65} /$ LE $_{65}$ (in \%), by gender. EU countries, 1995-2001 (Source: Jagger C and EHEMU team, European Health Expectancy Monitoring Unit, www.ehemu.org)
77. Another study, based on six longitudinal studies on ageing ( 5 from Europe and one from Israel), reported a significant variation of the DFLE $65 / \mathrm{LE}_{65}$ ratios across countries ${ }^{49}$. Italy had the lowest ratio, amounting to $75 \%$ and $83 \%$ in women and men, respectively, and Sweden the highest ( $83 \%$ and $93 \%$ ). Note that these differences essentially result from different patterns in DFLE 0 and $\mathrm{LE}_{0}$. For example, Italian men enjoy higher life expectancy among the six countries, but wit the lowest DFLE.
78. A Dutch study ${ }^{50}$ showed that the increasing number of years with disability were in fact related to mild disabilities. DLE 65 with mild disability increased from 9.9 to 13.9 years in men (from 10.6 to 14.8 years in women), while DLE $_{65}$ with moderate or severe disability showed a slight reduction. This can be interpreted as evidence for the theory of dynamic equilibrium, in which the increased prevalence of chronic disabling diseases is counterbalanced by a decrease in the severity of the disability induced by these diseases.
79.The effect of gender on disability is partly explained by differences in social and health related factors ${ }^{51}$. However, the association between female gender and mobility problems is independent, probably mediated by musculoskeletal conditions and depressive symptoms. An alternative explanation is that women are more likely to report disease and disability than men.
80.Japan has limited data on the prevalence of disability, due to the fact studies did not include residents of institutions. In 2002, HALE at birth was 72 years for men and 78 years for women ${ }^{\text {d }}$. According to Schoeni ${ }^{52}$, the prevalence of disability declined in Japan between 1993 and 2002. There would have been more than 1.1 million more disabled elderly in 2002 in an older population totalling 23 millions if disability had been constant over that period. Nevertheless, these results should be taken with caution as the decline in disability was not accompanied by a decline in functional limitations, suggesting that the results might be partially explained by changes in the built-in environment or developments in assistive technology.
81. In the US, studies found that the prevalence of disability increased during the 1970s, paralleling the increase in life expectancy ${ }^{53}$.A decline was then documented during the 1980 s, with an acceleration in the 1990s, for both mild and moderate/severe disability $54 ; 55$ : the increase in DFLE for both genders paralleled the increase in $\mathrm{LE}_{0}$. However, as a proportion of $\mathrm{LE}_{0}$, DFLE increased only in persons with higher educational status, while those with lower education were still experiencing an expansion of morbidity 56 .
82. The most recent trends in the US have been analysed by Cai $\&$ Lubitz for the population aged 65 and over ${ }^{57}$. During the 1992-2003 period, DFLE 65 increased from 9.5 to 10.3 years, and DLE 65 slightly decreased, mainly as a result from a decrease in severe disability and an increase in moderate disability. These trends result in HALE of 67.0 years in men and 71.0 years in women e . Finally, a crucial question is to what extent the rise in obesity will lead to a reversal in the trends in disability.
83. As a conclusion, trends in disability are heterogeneous. Moreover, several studies showed that an increase in the reported or diagnosed morbidity is not incompatible with a decrease in disability.

[^2]
## Summing up: lessons from the past

84. In Switzerland, life expectancy continues a steady increase that began sometime before the end of the 19 th century for $\mathrm{LE}_{0}$, and in the middle of the 20th century for $\mathrm{LE}_{65}$. The latter increased at a pace of 1-1.5 month per year in the 1990s. As in other countries, there is no clear sign that the rise in $\mathrm{LE}_{0}$ might come to an end, although the yearly increase is less pronounced since the 1980s. The modal age at death, which can be interpreted as the "normal" mean longevity at a given time, fluctuated until the 1920s, and then increased steadily, at a yearly rate of 2-3 months. Adult variability of the age at death decreased during the last fifty years, compatible with a compression of mortality.
85.There was a widening gender gap in $\mathrm{LE}_{0}$ throughout the 1900s, which then narrowed in most countries since the late 1980s. Another widening gap in longevity, also in Switzerland, is related to differences in socio-economic status ${ }^{58}$.
85. It is unclear what the driving forces of the continuing increase in life expectancy are.
87.In Switzerland, the 1950s marked both the end of the epidemiological transition (i.e., with the disappearance of infectious diseases as a public health problem) and the beginning of a "post-transition area", characterized by the mortality decline in old and very old people. Causal mechanisms explaining the turning point of the 1950s are related either to contemporary improvements (i.e., changes directly benefiting the older people like antibiotic treatment for pneumonia, pension scheme, etc.), or to past improvements in the corresponding birth cohorts.
86. The improvement in the socio-economic environment has been massive in Switzerland after World War II, with direct benefits for the older persons at that time. A Swedish study on the period 1861-1999 ${ }^{59}$ suggests that mortality among people aged 90-94 is directly correlated with manufacturing wages (used as an indicator of general wealth). It is not obvious to identify massive improvements in the history of birth cohorts born in 1890 and after (i.e., those aged 60 and over in the fifties): they were confronted with two wars (less dramatic in Switzerland than elsewhere, but still a challenge) and a marked economic crisis in the 1930s.
87. One can also imagine that the explanation of the continued rise in life expectancy may lie in the self-propelling nature of some of these health improvements. For example, if younger adults are healthier, they get healthier children, who in turn will get even healthier children. This idea of self-propelling improvement might also be related to improvement in education. Further, the decline or delay in widowhood related to the decline in older persons mortality might also be associated to a longer life expectancy in those escaping to widowhood.
88. On the other hand, there is no fate dictating that $\mathrm{LE}_{0}$ should continue on the same line. A plateau of $\mathrm{LE}_{0}$, or even a decline, has been observed in some European countries, probably related to crises affecting the socio-economical environment, life styles (in particular, alcohol and tobacco use) or the supply of medical services.
89. The favourable trend in LE is associated with a decrease of age-specific disability prevalence during the last twenty years in several industrialized countries, including Switzerland. Consequently, DFLE $_{65}$ increased during the last twenty years, as well as the proportion of years spent without disability, suggesting a compression of morbidity. Women consistently spend a longer proportion of life with disability, perhaps because of the differences in educational, social and health related factors. Globally, the male-female gap is lower for DFLE than for total life expectancy ${ }^{60}$.
90. However, similar to trends in $\mathrm{LE}_{0}$, trends in DFLE are heterogeneous, with some countries showing stability or even decrease in the proportion of years spent without disability after age 65.

## Future evolution of longevity and healthy life expectancy

93. Predicting the future of mortality and morbidity means that one has some idea on their past and current determinants. Moreover, a model of the interaction between mortality, morbidity and determinants is needed. For the sake of clarity, the typology below presents two main approaches of predictions:

- "demographic" approaches :
- projections of the past trends of mortality or life expectancy (cf. §95, p. 23)
- projections based on limit distributions (cf. §124, p.27),
- "epidemiological" approaches (cf. §136, p.28), i.e., models based on the future evolution of diseases and their known risk or protective factors; these approaches include the evolution of healthy life expectancy;

94. Other approaches using for example a combination of the above mentioned methods are finally presented.

## "Demographic" approaches : Projections of the past trends of mortality or life expectancy

95. Selected projections of $\mathrm{LE}_{0}$ from demographic approaches are presented in Table 5, p. 32. According to Swiss Federal Office for Statistics, the forecasted LE ${ }_{0}$ in 2050 are 85.0 years for men( ranging from 82.5 to 87.5 years according to the optimistic and pessimistic scenarios) and 89.5 ( 87.5 to 91.5 years) for women ${ }^{47}$. From 2005, the annual increase is thus situated between 4 and 10 weeks for men, and between 4 and 9 weeks for women.
96. In these approaches, the past trends are either directly extrapolated, or used in mortality models ; several scenarios are usually considered (e.g., with various secular changes in mortality ratios, or with various reference periods, etc.).
97. Projections for the short run typically rely on a linear extrapolation of historical trends in log of mortality rates or life expectancy. Most studies in this group use a limit for life span, although others ignore such a limit (for the latter, see §123and ff.).
98. Extrapolation without constraints usually produces implausible results in the long run. For example, linear extrapolation of past $\mathrm{LE}_{0}$ trends for Denmark and Japan will lead to huge future differences between these two countries because the different past trends ${ }^{61}$. The same is true for extrapolation of male and female $\mathrm{LE}_{0}$. For example in most countries male and female $\mathrm{LE}_{0}$ are converging. Simple extrapolation of these trends leads to crossover with male $\mathrm{LE}_{0}$ exceeding female $L_{0}$.
99. Thus, choosing an upper limit to life span is a reasonable approach. Among the arguments for a limited duration of life ${ }^{62}$, the most important is probably the decline of physiological parameters associated with ageing in humans, with a loss of $80 \%$ of the functional capacity by age 80 . A further demographic argument is the slowdown of the $\mathrm{LE}_{0}$ increase, as observed in several countries, which can be taken as an argument in favour of an upper limit. However, this slowdown is paralleled by an accelerated decline in mortality at older ages. Moreover, the fastest decline of mortality has been observed in countries with the lowest levels of old-age mortality, i.e., the opposite of what is expected if mortality were pushing against an upper limit ${ }^{63}$.
100. Although there are biological arguments in favour of an upper age limit, there is debate about the value of this limit. The many proposals for the maximum LE ${ }_{0}$ found in the literature vary between 65 years (as proposed by Louis Dublin in 1928) to 85 years 64 (with a maximum life span of 122 years).
101. Using a biodemographic approach, Olshansky refers to the schedule of age-specific death rates as an "intrinsic mortality signature", that might change only when the forces of selection acting to maintain the genetic composition of a population are disrupted (either through environmental challenges, interventions, or diseases). This model estimated the maximum life expectancy being 85 years in $1990{ }^{65}$, with gender-specific limits of 82 years for men and 88 years for women. To get these estimates, death rates would have to decline at every age and for every cause by about $55 \%$ from levels existing in 1895. The authors claim the magnitude of this changes would be equivalent to the elimination of both cancer and heart disease.
102. Apart from biological determinants of mortality, man-made determinants should be considered. Studies report it is not unlikely that the development of innovative technologies benefiting the health of older persons will continue on the same line, contributing to the lowering of death rates $66 ; 67$.
103. Another important issue is to set the observation period used for the linear model. Janssen and Kunst ${ }^{68}$ explored systematically the impact of choosing two reference periods ( $25-$ or a 50 -year) on the projected mortality of people aged 80 and more up to 2050 in seven European countries (Switzerland not included). The objective was not to produce a forecast, nor to recommend a specific technique, but to show the different ways in which mortality forecasts could integrate knowledge about past mortality trends (reflecting the effect of duration of the historical period, sex, smoking and country). As recommended by Wilmoth ${ }^{22}$, the projections used indirect extrapolations of age-specific mortality rates instead of life expectancies because the latter has proved to be less insightful owing to lack of information on underlying changes in age-patterns of mortality.
104. Choosing a reference period of 25 years (1975-1999) instead of 50 (1950-1999) resulted in gains in $\mathrm{LE}_{80}$, higher in men, but lower in women ${ }^{68}$; this obviously reflects the gender-specific changes in the secular trends. On average, for the seven countries together, the gain in life expectancy at birth at age 80 in men in the period 1999-2050 was 1.72 years and 2.13 years using the periods 1950-1999 and 1975-1999 as projection base, respectively. In women, corresponding figures were 3.78 and 3.39 years ${ }^{68}$.
105. Using non-smoking related mortality trends only ${ }^{68}$, gains in life expectancy were usually higher than those calculated using all-cause mortality. The projections based on non-smoking mortality should be considered with caution, as levels of smoking-related mortality were only estimated : detailed data were lacking. In men, the impact of the period used to project life expectancy also illustrated trends in smoking mortality, as using the period beginning in 1950 led to a higher projected gain 68.
106. Projections by country show substantial differences in the magnitude of the gain in $\mathrm{LE}_{80}$, explained by the variations in the pace of decline in old-age mortality over time. For example, for Norway, the stagnation of old-age mortality decline in the 1950s-1060s results in a lower gain in LE $_{80}$ when using the period 1950-1999 instead of 1975-1999 as projection base.
107. The Lee-Carter method and its variants are the most frequently used methods to forecast mortality ${ }^{69 ; 70}$. It is based on the linear decomposition of the logarithm of observed force of mortality at age $x$ during year $t\left(\mu_{x}(t)\right.$ into (i) a dominant component changing with calendar time, and (ii) an age component that remained fixed over calendar time.
108. As for linear projections of log of mortality rates, the Lee-Carter method may produce implausible age patterns in the long run if any rate trends differ, because these differences will be magnified in the projections.
109. By its very nature, the Lee-Carter method is extrapolative and projecting mortality in this way is likely to result in a continuous increase in life expectancy. Moreover, the forecasts (even when based on similar extrapolative procedures) are likely to imply increasing divergence in life expectancy in the long run. This contradicts the observation made by Wilson who documented a global convergence in mortality ${ }^{71}$. Li and Lee considered how a particular extrapolative method can be modified to forecast mortality for countries by taking into account their membership in a group, rather than individually ${ }^{61}$.
110. This method was used to model observed and forecasted sex-specific life expectancies and longevity in the United States from 1900 to $2065{ }^{72}$. The method is currently used by the US Bureau of the Census.
111. Besides the application of the method to several countries by Lee ${ }^{70}$, the Lee-Carter method have been used in the G7 countries projections, and in long-range projections for the UN Population Division $73 ; 61 ; 6$.
112. In the model proposed by the U.N. Population Division, based on the Lee-Carter method as modified by Li, the increments of $\mathrm{LE}_{0}$ over time decrease as higher levels of $\mathrm{LE}_{0}$ were reached ${ }^{74}$. This allowed to limit $\mathrm{LE}_{0}$ at 92.5 years in the medium-range projections.
113. For the long-term projections, the choice was made not to cap future $L_{0}$ to 100 years, providing allowance was made for the necessarily modified shape of mortality schedule. Because the Lee-Carter method might produce divergence of $\mathrm{LE}_{0}$ (instead of convergence) in the long-range projections, there was a proposal by Lee to modify the method to ensure non-divergence. In general, in the normal mortality scenario, mortality is projected on the basis of the models of change of LE ${ }_{0}$, using the period 1980-2000 as projection base, and choosing a medium pace of mortality decline. The long-range projections used a cohort-component method for the 100 years of projection then a simpler method for the rest of the projection period.
114. The Lee-Carter method has been compared to four variants (Lee-Miller, Booth, Hyndman-Ullah, De Jong-Tickle). The variants performed better in forecasting death rates, but there were no significant differences in accuracy for the shortterm forecasts for life expectancy 75.
115. Retrospective projections suggest that the Lee-Carter method tended to underestimate the gains in the US, but did well in France, Sweden, Japan, and Canada ${ }^{76}$. On the other hand, one of the Lee-Carter assumption (the decline of mortality at each age remains constant over time) contradicts current observations, i.e., the decline of mortality slowed down at younger ages and rose at older ages.
116. To overcome this limitation of the Lee-Carter method, a "shifting" logistic model has been proposed by Bongaarts ${ }^{77}$, in order to capture the age-specific pattern of secular change in age-specific mortality. In this model, the shape of the pattern of mortality rates by age is held invariant while the pattern itself shifts to higher ages over time as life expectancy rises. The projections made according to the Bongaarts' model compared well with the Lee-Carter one, being more robust when long range projections are concerned.
117. Pedroza ${ }^{78}$ developed a Bayesian approach which incorporates several sources of variation in the model when forming forecasts, and allow to handle missing data both in the time series and across age groups. It also takes into account the uncertainty associated with it.
118. Babel et al. ${ }^{79}$ recently developed a mortality model incorporating the "volatility" of mortality, containing two separate dimensions : (i) a common time effect over all ages and (ii) a common age effect of mortality evolvement. The time effect denotes the common level of mortality growth/decline, while the age effect is a stochastically independent age-specific term. Using a common time period from 1959 to 2002, life expectancies for 12 regions (including the Alps region, represented by Austria and Switzerland) are estimated in 2050. Beside traditional period life expectancies, more realistic but less commonly used cohort life expectancies are also calculated.
119. Using data from 1950 to 1999 in 7 European countries (excluding Switzerland), Janssen and Kunst ${ }^{68}$ predict an increase of $\mathrm{LE}_{80}$ averaging 2.3 years among men and 4.0 years in women by 2050 . Assuming unchanged mortality rates at younger ages, a 2.3 years increase in $\mathrm{LE}_{80}$ would lead to an increase in $\mathrm{LE}_{0}$ of 1.0 year in men and 2.5 years in women. As a comparison, adding these estimates of increase to $\mathrm{LE}_{0}$ observed in 2000 in Switzerland results in projected $\mathrm{LE}_{0}$ for 2050 of 78.2 and 85.5 years for men and women respectively, somewhat lower than the pessimistic scenario by SFOS.
120. Bongaarts examined trends in life expectancy when decomposed into juvenile, background, senescent and smoking-related mortality ${ }^{26}$. After removing the effects of juvenile, background and smoking mortality, the average rate of increase in senescent life expectancy over the past 50 years was 0.15 years per year, and was almost linear.
121. For projecting $L_{0}$, Bongaarts assumes that this trend is likely to continue for a few more decades, as there is no reason to believe that technological and medical advances will be less effective in reducing senescent mortality in the future than in the past. On the contrary, his model makes allowance for the future disappearance of the effect of declining juvenile and background mortality. Projections of life expectancy to 2050 for the two sexes based on this decomposition model (projecting separately trends in juvenile, background, senescent and smoking mortality) show that $\mathrm{LE}_{0}$ is likely to increase by an average of about 7.5 years up to 2050 depending from the country (see Table 5, p. 32). Although less optimistic than those of Oeppen and Vaupel, because they assume little impact of further improvement in juvenile mortality, Bongaarts' projections are more optimistic than those of the United Nations.
122. Several authors provide confidence intervals around the estimates, generally increasing with the duration of the period of projection. For example, Lee and Carter forecasted a $L_{0}$ increase of 10.5 years between 1990 and 2065, reaching 86.0 years with a confidence interval between 80.9 and 90.2 years. For 2050 and for Switzerland, Babel forecasted a $90 \%$ confidence interval for $L_{0}$ of 4.9 years (for the period-related data) and 6.6 years (for the cohort-related data) in men; corresponding values for women are 5.6 and 9.2 years ${ }^{79}$. Providing sound confidence intervals was one of the aim of Pedroza in his Bayesian approach of the Lee-Carter model ${ }^{78}$.
123. Those avoiding the use of a life expectancy argue that this assumption repeatedly proved to be too conservative. Using the last 160 years showing a strikingly linear increase of $\mathrm{LE}_{0}$ in women, the model used by Oeppen and Vaupel projects life expectancies throughout the $21^{\text {st }}$ century by continuing this trend ${ }^{80}$.

## "Demographic" approaches : Projections based on limit distribution

124. As noted above (see §93, p.23), demographic approaches include some assumptions on the extreme levels of mortality which will be reasonably reached. These approaches include combination of the lowest mortality rates observed by sex-age groups, or gaps between countries (i.e., considering the time needed by a specific country to catch up the most advanced countries), or estimate the lowest achievable cause-specific death rates. Some results of these approaches are displayed in Table 6, p. 32.
125. Achievable life expectancy can be estimated by combining the lowest mortality rates observed worldwide. Uemura collected the lowest age-specific mortality rates observed worldwide from 1950 to 1980, and calculated a potentially achievable LE ${ }_{0}$ of 76.2 years in men and 82.1 years in women ${ }^{81}$ (see Table 6, p. 32). As a comparison, observed LE $\mathrm{L}_{0}$ in Switzerland in 1980 was 72.4 for men and 79.1 for women ${ }^{47}$, and surpassed in 1999 the values calculated by Uemura.
126. More recently, Vallin and Meslé used a similar approach by combining the lowest age- and cause-specific mortality rates worldwide from 1950 to 2000. In 2000, the resulting LE $_{0}$ would reach 84.4 years in men and 88.9 years in women. A comparison between the highest observed $\mathrm{LE}_{0}$ and the potential $\mathrm{LE}_{0}$ resulting from the model showed that observed $\mathrm{LE}_{0}$ reached potential $\mathrm{LE}_{0}$ about 25-30 calendar years later (see Table 6, p. 32) ${ }^{82}$. This is similar to Uemura's findings. This suggests that the potential $\mathrm{LE}_{0}$ of 84.4 years (men) and 88.9 years (women) might be reached in the next two or three decades in countries with the highest current $\mathrm{LE}_{0}$. These values fall into the range of values estimated for Switzerland in year 2050: 82.5-87.5 for men and 87.5-91,5 for women ${ }^{47}$.
127. Eradication of one or more causes of deaths and the resulting change in mortality rates have been used to predict achievable gains in life expectancy. One of the problem here is the interdependency between causes of deaths ${ }^{83}$.
128. In the 1970's, Tsai examined the potential gains in $\mathrm{LE}_{0}$ when the three leading causes of death are totally or partially eliminated. The impressive gains theoretically achieved by total elimination do not hold up under the more realistic assumption of partial elimination or reduction. The number of years gained by a newborn child, with a $30 \%$ reduction in cardiovascular disease, would be 1.98 years, for cancer, 0.71 years, and for motor vehicle accidents, 0.21 , years. Simulating the same reduction for the working ages ( 15 to 70 years) results in a gain of $0.43,0.26$, and 0.14 years, respectively for the three leading causes of death. The author concluded that even with a scientific breakthrough in combating these diseases, future gains in life expectancies for the working ages will not be spectacular ${ }^{84}$.
129. Nusselder estimated the gain in life expectancy resulting from the elimination in selected chronic diseases ${ }^{85}$. For example, eliminating heart diseases added 3.1 years for men and 2.7 years for women to $\mathrm{LE}_{65}$, while eliminating cancers added 2.7 years and 1.9 years, eliminating diabetes added 0.1 to 0.3 years, for men and women, respectively (see Table 6, p. 32). The elimination of other chronic diseases, such as chronic obstructive pulmonary diseases or Parkinson disease, had a negligible effect on life expectancy. Authors did not try to estimate the cumulated gain resulting from the cumulative elimination of several groups of diseases.
130. Oeppen and Vaupel recommended using the observed gaps between countries and regions : the life expectancy in the countries with the highest life expectancy can set as the achievable limit for all ${ }^{80}$. This approach is close to the one computing life expectancy based on the lowest mortality rates. However, this approach is less significant in Switzerland which has the second highest life expectancy worldwide.
131. The models based on the "rectangularization" of the survival curve belongs to these "analytical" approaches. The theory states that at a given period, there is strong compression of mortality around a given age (and a stiffer resistance to further increase is expected), without stating what will be the value of this limit.
132. Based on the inverse relationship between the modal age at death ( M , see also §18) and the standard deviation of the age at death above the mode ( $\mathrm{SD}_{(\mathrm{M}+)}$, see also §18), Kannisto developed his hypothesis of an "invisible wall" to the extension of human longevity ${ }^{15}$ : as the modal age at death increases, the right-hand slope of the distribution of age at death becomes more and more vertical.
133. Up to now, there are few observations backing up this hypothesis. One study reported clear signs of verticalization in Switzerland between 1969 and 1994, using the trend in distribution of age at death after 50 year ${ }^{21}$. Recent studies noted that rectangularization might slowly come to an end, with a shift of the mortality towards older ages ${ }^{86}$.
134. There are few published data about the future evolution of $\operatorname{SD}(\mathrm{M}+)$. A model of the curvilinear relationship between M and $\mathrm{SD}(\mathrm{M}+$ ) can be used to estimate the values of $\operatorname{SD}(\mathrm{M}+)$ if M goes on increasing up to 95 years. Using data from France, Italy and Japan, $\mathrm{SD}(\mathrm{M}+)$ was estimated to decrease up to the values of 5.5 to 6.5 years when M reaches the value of 95 years, while for Sweden, $\mathrm{SD}(\mathrm{M}+$ ) might take the value of about 4.5 years. This curvilinear relationship however suggests that $\mathrm{SD}\left(\mathrm{M}+\right.$ ) will eventually reach some kind of floor values ${ }^{87}$.
135. It is indeed unlikely that the survival curve will become totally rectangular, due to the heterogeneity of the human population, so that the question remains up to what degree of rectangularization the survival curve will evolve.

## "Epidemiological" approaches : Projections based on health-related information

136. These models are using information related to the future evolution of lethal, degenerative diseases, which represents the major burden in ageing populations : in developed countries, heart disease, cancer and cerebrovascular disease are responsible of $60 \%$ of all deaths of people aged sixty-five or older ${ }^{23}$.
137. Obviously, the credibility of the projections depends on the current knowledge about the evolution of known determinants of these diseases, i.e., the risk or protective factors related to the physical and social environments, to life styles, to health care and even to the genetics. The future of climate change, inequalities in health, diet, technological innovation or migration are classical examples. Factors considered in this report will be cardiovascular risk factors and diseases, smoking, cancers, and genetics.
138. These approaches are based on the expected future trends in prevalence of diseases and their determinants. Olshansky claimed that the reason why official forecasts consistently underestimated mortality was their reliance on static extrapolation of past trends, neglecting medical, behavioural or social factors that influence mortality ${ }^{88}$. Therefore, he proposed a "multiple cause-delay model" to capture the impact of more favourable risk factors on mortality.
139. Because of its frequency, cardiovascular disease (CVD) might be the single most important determinant of the future of longevity. An analysis of mortality of people aged 75-84 years in Europe between 1970 and 1996 showed that the decline in total mortality was almost entirely due to the decline in CVD mortality, especially stroke ${ }^{89}$. Mean blood pressure has fallen in Switzerland as in most developed countries ${ }^{90 ; 91}$, and this probably is the most important determinant of the decline in stroke mortality.
140. CVD mortality declined in most developed countries ${ }^{92}$. In Switzerland, the decline of incidence between 1985 and 1993 was $3.6 \%$ for men ${ }^{93}$. This secular decline is more marked as age increases, and its impact is therefore more marked in an ageing population ${ }^{93-95}$. Further, available evidence show that there is still a room for improvement ${ }^{96}$ for diet ${ }^{97 ; 98}$ (including the reduction of salt consumption $99 ; 100$ ), sedentarity, tobacco consumption, etc. There is also room to improve the care of hypertensive and dyslipidemic patients ${ }^{93 ; 101}$.However, increases in total CVD mortality were recently observed in some central European countries and Greece. In the United Kingdom, CVD mortality among young men recently increased for the first time since two decades, perhaps as a consequence of unfavourable trends in risk factors, especially obesity and diabetes ${ }^{102}$.
141. CVD mortality also matters to explain the gender difference of overall mortality. Currently, gender differences in CVD mortality rise steeply with age in parallel for both genders, but the female rates are those observed $5-10$ yr earlier in men ${ }^{103}$. In the US, the levels of elevated blood pressure (with or without medication) have declined for older men but have increased for older women ${ }^{23}$.
142. Trends in cancer incidence are heterogeneous. Between the 1960s and 2002, the contribution of a reduced cancer mortality to the increasing $L E E_{0}$ was less than 1 year, as compared to a contribution of almost 3 years from cardiovascular diseases 104. However, because of the decline in CVD mortality, the cancer mortality increased its contribution to the overall mortality decline after 1990.
143. The evolution of gender differences in mortality is consistent with smoking patterns. Recent changes will probably reduce sex differences in mortality in the coming decades ${ }^{105}$. An analysis of the trends in male and female life expectancy and of the proportion of deaths attributable to smoking allows determining the effect of smoking on $\mathrm{LE}_{0}{ }^{26}$. In year 2000, data from 16 countries suggested that smoking reduced $L_{0}$ by 1.0 year in women and 2.4 years in men; corresponding figures for Switzerland were 0.5 and 1.9 years.
144. Janssen and Kunst ${ }^{68}$ explored systematically the impact of choosing nonsmoking related mortality instead of all-cause mortality, this approach yielded to higher gains of life expectancy in women. Bongaarts also estimated the impact of smoking on trends in $\mathrm{LE}_{0}$ during the last 50 years. Even after eliminating the effect of smoking, $\mathrm{LE}_{0}$ remains lower in men than in women.
145. Because of its consequences on CVD, diabetes and cancer, obesity might play a crucial role in the evolution of longevity in the next decades, as its prevalence increases in several industrialized countries. According to Olshansky ${ }^{9}, \mathrm{LE}_{0}$ was reduced by 4-9 months in the US in 2000 because of the current frequency of obesity. Based on its future trends, obesity could reduce $\mathrm{LE}_{0}$ by two to five years in the coming decades (see Table 6, p. 32).
146. An effective control of hypertension could add 75 million DALYs, and eliminating diabetes would add 90 million life-year equivalents ${ }^{106}$.
147. Body mass index (BMI) also increased among older people. In the US, after remaining roughly stable in the 1970s, the proportion of non-institutionalized older women and men who are obese (BMI > $30 \mathrm{~kg} / \mathrm{m}^{2}$ ) increased among older men from 19 percent in 1988-1994 to 26 percent in 2001-2004, and among older women from 23 percent in 1988-1994 to 31 percent in 2001-2004. As a consequence, the prevalence of diabetes has increased among older people in several countries. In the US, this increase was from $18 \%$ in 1988-1994 to $25 \%$ in 2001-2004 ${ }^{23}$.
148. In Switzerland, obesity currently affects about one in ten adults, but overweight is found in almost one in two persons. However, the effect of overweight on longevity seems negligible ${ }^{107}$.
149. However, the relationship between obesity and CVD is more complex than initially thought. In the US, obesity did increase between the 1970s and the early 2000s, but the overall cardiovascular risk profile of the population has improved, mainly because of the reduction in smoking and better control of blood pressure 108. Another study showed that the association between BMI and blood pressure has decreased over time ${ }^{109}$.
150. Diet has an substantial effect on survival ${ }^{110}$. A healthy Mediterranean-type diet has been shown to reduce mortality risk by $30 \%$ in older women ${ }^{111}$.
151. Genetic influences on human life span and longevity are under study. Within a given birth cohort, there are considerable variations in life span. Twin studies suggested that about one quarter of the overall variation in lifespan might be accounted for by genetic differences $112 ; 113$.
152. The rapid increase in $L E^{0}$ throughout the past century is unlikely to be due to genetic factors, although some authors believe that there is a possible role for a selection mechanism favouring long survival ${ }^{114}$.
153. The impact of health services on longevity are debated and thus, the impact of potential developments in health care. Some experts claim that major discoveries are likely in the next few decades and that they will impact on life expectancy $115 ; 116$, while others are sceptical ${ }^{117}$. In any case, there is still room for improvement only by a better coverage of people with existing effective intervention, e.g., by increasing the coverage by vaccination ${ }^{23}$.
154. With joint replacements (knee and hip), cardiac revascularization procedures (angioplasty and bypass surgery) has become a substantial part in improving functioning and quality of life in recent years. The rate for bypass surgery increased during the 1980s and 1990s. Coronary angioplasty was introduced in the late 1970s : between 1995 and 2004, its rate was doubling, with the largest increase occurring among people aged 85 and more ${ }^{23}$.
155. Diehr et al. estimated the impact of five interventions on mortality on people aged 65 and more ${ }^{118}$. Longevity could be increased by 3 months as the result of a oneshot intervention that would make healthy all sick people at baseline. An improvement of similar magnitude could be obtained from an intervention decreasing the probability of getting sick each year by $12 \%$, or from an intervention increasing the probability of a sick person recovering by $16 \%$, from an intervention decreasing the probability that a sick person dies by $15 \%$, or from an intervention decreasing the probability that a healthy person dies by $14 \%{ }^{118}$.

## Conclusion

156. Apart from demographic and epidemiological approaches, other experts use approaches that combine demographic approaches with analytical, epidemiological and biological approaches. However, these approaches have been more frequently used as theoretical bases, and there is less literature containing projections based on these theories.
157. The process of ageing itself can be a central aspect of the theory underlying the future evolution of longevity as a large part of the ageing might be related to biological phenomenon ${ }^{119}$.
158. The search for a single cause of aging has recently been replaced by the view of aging as an extremely complex, multifactorial process ${ }^{120}$. The different theories of aging are now considered as complementary.
159. A straightforward and simple approach ${ }^{64}$ attributes age-related changes to genetic defects, perinatal and post-natal development, environment, disease, plus an inborn process, aging itself. Accumulation of deleterious changes produced by aging throughout the cells and tissues progressively impairs function and can eventually cause death. The mortality at a given age is a measure of the cumulated change at that age (the physiologic age), and the rate of mortality change is the rate of aging.
160. Risk of death is now largely determined by the inherent aging process after age 28. Only $1.1 \%$ of female cohorts in Sweden die before this age; the remainder die off at an exponentially increasing rate with advancing age.
161. According to this theory, risk of death in developed countries are now reaching limiting values : $\mathrm{LE}_{0}$ is approaching plateau values that are 6-9 years less than the potential maximum of about 85 years ${ }^{64}$.
162. On the contrary, according to a theoretical model combining information related to both trends in cell growth and in survival ${ }^{121}$, life span could increase beyond 120 years. In fact depending from what types of cells is considered, this maximum value varies between 98 and 126 years. Relevant mechanisms may be partly related to endocrine control ${ }^{122}$.
163. Among other research on ageing, it has been shown that long-term caloric restriction may extend the life of the laboratory rat by about $25 \%$. Although most humans would not adhere to such a diet as the one imposed on laboratory animals, research in humans has shown that a $20-25 \%$ reduced caloric intake is able to improve risk factors for cardiovascular diseases, and biomarkers for longevity (deep body temperature and plasma insulin) and thus, should increase life expectancy.
164. Using information from the survival studies of overweight and obese people, it is estimated that long-term reduced caloric intake to prevent excessive weight gain could add 3-13 years to life expectancy. However, the effects of caloric restriction on human life extension are probably much smaller than those achieved by medical and public health interventions : these have extended life by about 30 years in developed countries in the 20th century, by greatly reducing deaths from infections, accidents, and cardiovascular disease ${ }^{123}$.
165. Although several studies on animal models have shown that aging rates and life expectancy can be modified by "anti-aging" remedies, there is currently no evidence that these substances can slow aging or increase longevity ${ }^{124}$.
166. Based on projections of future economic and social development, and using historical relationships of economic and social development with cause-specific mortality rates, the WHO published projections of $\mathrm{LE}_{0}$ to 2030 in several regions of the world ${ }^{125}$. According to this analysis, $\mathrm{LE}_{0}$ is thought to increase in all regions of the World, provided future mortality in low income country will have a relationship to economic and social development similar to those that have occurred in higherincome countries. The increase in $\mathrm{LE}_{0}$ in high income countries should reach 4-5 years in men and women, with a range of about 1.5 years between the pessimistic and the optimistic scenarios.
167. A complete scenario of future trends in longevity should also include projections of modal age at death, as well as insights about the future evolution of the survival curve in terms of rectangularization. Unfortunately, we are not aware of published work using these indicators.

| Projection method | Swiss Federal Office of Babel \& al. 200779 Statistics, $2006{ }^{47}$ |  |  |  | Tuljapurkar <br> S, $2000{ }^{6}$ <br> Stochastic forecast | Li \& Lee $200561$ <br> Convergence of mortality, (see § 115) | U.N. <br> Population <br> Division, <br> 2007126Lee-Carter <br> (see § 107) | $\begin{gathered} \text { Bongaarts J, } \\ 2006^{26} \end{gathered}$ |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | $\begin{aligned} & \text { Period/cohort } \\ & \text { approaches } \\ & \text { (see §118, p. 26) } \end{aligned}$ |  |  |  |  | decom of $m$ (se | sition tality 91) |
|  | M | W | M | W | $M+W$ | $M+W$ | M + W | M | W |
| Switzerland | $\begin{gathered} 85.0 \\ (82.5- \\ 87.5) \end{gathered}$ | $\begin{array}{r} 89.5 \\ (87.5- \\ 91.5) \end{array}$ |  |  | - | 86.5 | 82.9 | 84.5 | 90.2 |
| Switzerland and Austria |  |  | 82.1/89.1 | 87.3/94.0 |  |  |  |  |  |
| Japan | - | - | 87.0/97.0 | 94.5/103.6 | 90.9 | 88.1 | 88.1 | 85.2 | 92.1 |
| France | - | - | 82.0/89.5 | 89.4/96.8 | 87.0 | 85.8 | 84.0 | 82.8 | 90.3 |
| Germany | - | - | 81.4/88.1 | 87.5/95.0 | 83.1 | 84.8 | 83.5 | - | - |
| United States | - | - | 80.4/87.3 | 84.9/91.4 | 82.9 | 84.9 | 81.6 | 81.6 | 87.0 |

Table 5. Values of projected $\mathrm{LE}_{0}$ for 2050 in selected countries.

|  | $\begin{gathered} \text { Nusselder et al. } \\ 1996^{85} \text { (see } \\ \S 129, \text { p.27) } \end{gathered}$ | Uemura 198981 (see § 125, p.27) | Vallin \& Meslé (unpublished) ${ }^{82}$ (see § 126, p. 27) | $\begin{gathered} \text { Olshansky } \\ 2005^{117} \\ \text { (see § 145, } \\ \text { p. 29) } \end{gathered}$ | Mathers et al. $2006{ }^{125}$ (see § 162, p. 31) |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Projection method | Elimination of selected disease | Lowest age- <br> specific <br> mortality rates <br> worldwide | Lowest age- and cause-specific mortality rates worldwide | Impact of obesity | Cause- <br> specific <br> mortality <br> rates |
|  | Potential change in life expectancy at the time of analysis |  |  |  | 2030 |
|  | $M / W$ | $M / W$ | $M / W$ | $M+W$ | $M / W$ |
| Increase in LEO | From CVD: $+3.1 /+2.7 \mathrm{yrs}$ <br> From cancer: $+2.7 /+1.9 \mathrm{yrs}$ | +2.5/+3.0 yrs | +5.4/+5.0 yrs | USA: <br> - 3-4 <br> months | Highincome countries: 85.0/79.7 |

Table 6. Projected $\mathrm{LE}_{0}$ according to selected approaches

## Determinants of healthy life expectancy

168. Healthy life expectancy (HLE) depends on the burden of chronic diseases that may result in disability. Past trends in the evolution of HLE showed different evolutions between countries, within countries and between genders. Expectedly, there is currently a lack of consensus about future trends in HLE.
169. Projections of HLE rely on projected total life expectancy, combined with projections of the prevalence of disability. Trends of the latter have been favourable over the past twenty years. Based on information on younger cohorts, the prevalence of disability is projected to further fall until about 2015, then to stabilize and even rise slightly ${ }^{127}$.
170. Moreover, the rising rates of obesity and diabetes might well reverse disability trends ${ }^{23}$. The consequence of obesity on disability-free life expectancy is a loss of about 2.8 years of years of healthy life, even if $\mathrm{LE}_{0}$ is not substantially reduced ${ }^{128}$. A trend analysis in obesity between 1985 and 2002 in middle-aged Americans, with an extrapolation to 2020, found that disability rates will increase by 1 percent more than if there were no further weight gain ${ }^{129}$.
171. Some recommend against extrapolating recent trends in disability forward into the future, because of the complex determinants of disability. In particular, the development of new assistive technologies might have contributed to the fall in reported disability ${ }^{130}$.
172. Using a similar approach as Nusselder for $\mathrm{LE}_{0}$ (see $\S 129$ above on page 27), Ezzati computed health-adjusted life expectancy in the absence of 20 major risk factors for diseases and injuries. The burden of disease attributable to the joint effects of 20 selected leading risk factors were assessed and population attributable fractions for these risk factors were estimated using information about risk factor prevalence and about the associated risk of disease. The population attributable fraction represents the proportional reduction in disease or mortality that would occur if exposure to a risk factor were reduced to an alternative level. In Western Europe, removing these risk factors would increase health-adjusted life expectancy by about 5 years as compared to the current situation in $2000{ }^{131}$.

## Summing up and recommendations

173. In summary, all methods of projection predict that $\mathrm{LE}_{0}$ will increase up to 2050 in men as in women in industrialized countries such as Switzerland, within a range of values of five to nine years ${ }^{79}$.
174. Most projections of life expectancy use historical trends in mortality rates or life expectancy as an input, viewed as the most reliable method of predicting the future trends ${ }^{22}$. The main argument in favour of this approach is the remarkable stability of the changes over the past 200 years. It has been noted that the increase in life expectancy has occurred parallel to improvements in social context and medical care, but also included events with substantial demographic impact, such as the 1918 Spanish Flu, or wars. Further, it is claimed that any alternative methods of projection require a better understanding of underlying mechanisms ${ }^{5}$.
175. On the other hand, previous projections based on past trends very often underestimated the increase in life expectancy, partly because of the belief that the main causes of longevity improvement in the $20^{\text {th }}$ century were on-off gains that could not be repeated ${ }^{132}$. Moreover, these extrapolative approaches neglect available information on past and future determinants of mortality and morbidity. In Switzerland for example, it seems that the 1950's have been a pivotal period with the start of the decline of the old age mortality.
176. It should be noted that projected values obtained through analytical and/or epidemiological hypotheses are not very different from those provided by extrapolative methods. But they are in a way more credible because based on some sort of logical information structure.
177. Despite available biological evidence, theories regarding an upper limit to life expectancy do not have much support.
178. Changing rates of specific chronic diseases (diabetes for example) and their determinants (obesity for example) will, at least in the short term, have a bigger impact on the years of life spent with disability rather than on life expectancy, although the observed increase in diabetes among younger adults might ultimately lead to higher death rates.
179. Recent trends in the proportion of healthy life expectancy are divergent across European countries. Whether these results indicate real variation or differences in measurement of disability is still undetermined. Robine and Michel claim that the compression and expansion of morbidity coexist, each of them being more prominent at a time ${ }^{133}$.
180. According to the general conclusions, two recommendations are proposed below, the first about making predictions, the second about the information system.

## Making predictions

181. There is currently no definite argument in favour of one unique, specific method: we thus recommend to publish regularly (e.g., on a 5 years basis) projections based on several methods rather than on a single one.
182. In practical terms, this means that SFOS should continue to provide projections based on the usual demographic methods, plus to provide at least three other different projections, namely (i) a method using a set of observed lowest mortality rates at a given time (see $\S 125 \mathrm{ff}$, p. 27), (ii) a method using the epidemiological projections of the main determinants (e.g., smoking, diet, etc.) of CVD mortality (see $\S 139 \mathrm{ff}, \mathrm{p} .28$ ), and (iii) a method estimating the effect of the projected evolution of education ${ }^{58}$ and socio-economic status on the future mortality rates.
183. Current demographic methods used by the SFOS should be complemented by several new development. One is to refine the use of historical trends and to use a historical period at least as long as the projection period 68. A further development is to take into account the mortality experience of other populations, as suggested by Li and Lee (see §109, p 25). Further, the cohort effects should be considered, i.e., the mortality at young age should be analysed to determine whether recent trends in old-age mortality are likely to persist into the future.
184. As for the limit to life span, there is no available evidence of such a limit, and there are few signs that we are approaching this limit : in fact, ongoing studies in Switzerland suggest that we have a rectangularization of the survival curve, plus an increasing mean duration of life.
185. The three methods suggested earlier (see $\S 181$ above) need a closer collaboration between demographers and epidemiologists. The best way to achieve this collaboration is to set up a formal group working regularly on the theoretical and practical issues related to the use of the three methods.
186. Within the framework of the development of health-related indicators for the use of the three methods, more will be available to provide regularly estimates of healthy life expectancy. Its importance will grow as the burden of our ageing societies on the health and social services will be highly influenced by the number of years older persons spend in a disabled state. In other words, there is a need to monitor the health status of the oldest old, in order to estimate future age-specific needs for health and social services. Projections related to mortality and healthy life expectancy will be provided at the same time.

## Develop appropriate information systems

187. A closer monitoring of longevity is needed. In practical terms, this means that mortality rates of the oldest-old (i.e., after 90) should be followed up not only with cross-sectional estimates, but also with cohort. The implementation of the Swiss National Cohort, a joint initiative of the Swiss Federal Office of Statistics and the Swiss Institutes for social and preventive medicine ${ }^{58}$ should help in this perspective.
188. In order to develop methods using health related information, epidemiology in the oldest population should be improved ${ }^{134}$. There are at least two practical proposals. The first is to develop the health surveys addressed to elderly people. The Swiss health survey should systematically include a sample of institutionalized population, for this is a substantial part in this age range.
189. A second aspect is to rethink the system of cause of death. As it is, it is useless for the majority of deaths occurring after 80 years because the information content is low. Several initiatives, experiments and pilot studies are needed to prepare a change in the registration system. This could be a topic for a national, or even international, R\&D programme.

## Implement innovative research

190. Finally, a program of research aiming at disentangling the social and health care effects on mortality is needed. This is particularly important as the social expenses (e.g. housing) are in concurrence with the medical expenses.
191. As far as migration is concerned, Switzerland has traditionally a strong immigration policy, as reflected by the $20 \%$ of foreign persons, but the mortality pattern of this population is still unclear, as well as its impact on longevity in Switzerland.
192. Finally, in the context of a continuously increasing longevity engendering the emergence of a frail population, at risk for becoming disabled, the study of frailty also deserves much interest.

## Annexes



Figure 9. Evolution of $\mathrm{LE}_{0}$ since 1825. Women, selected European countries (Source: European Health Expectancy Monitoring Unit, www.ehemu.org).


Figure 10. Evolution of LE $_{0}$ since 1825. Men, selected European countries (Source: European Health Expectancy Monitoring Unit, www.ehemu.org)


Figure 11. Three pattern of evolution of LE $_{0}$ since 1945 (i) "high convergence group", i.e., LE ${ }_{0}$ at the highest level in Europe, (ii) "low convergence group", i.e., $\mathrm{LE}_{0}$ at about 2 years lower than the highest level in Europe, (iii) "divergent group", $\mathrm{LE}_{0}$ diverging from overall trend. Women, various European countries (Source: European Health Expectancy Monitoring Unit, www.ehemu.org)


Figure 12. Three pattern of evolution of LE $_{0}$ since 1945 (i) "high convergence group", i.e., LE ${ }_{0}$ at the highest level in Europe, (ii) "low convergence group", i.e., $\mathrm{LE}_{0}$ at about 2 years lower than the highest level in Europe, (iii) "divergent group", $L_{E_{0}}$ diverging from overall trend. Men, various European countries (Source: European Health Expectancy Monitoring Unit, www.ehemu.org)


Figure 13. Evolution of LE $_{65}$ since 1825. Women, selected European countries (Source: European Health Expectancy Monitoring Unit, www.ehemu.org)


Figure 14. Evolution of $\mathrm{LE}_{65}$ since 1825. Men, selected European countries (Source: European Health Expectancy Monitoring Unit, www.ehemu.org)


Figure 15. Trend in life expectancy and DFLE ${ }_{65}$ in several European countries, 1995-2003, by gender. (Source: European Health Expectancy Monitoring Unit, www.ehemu.org)

## Literature

(1) Lutz W, Sanderson W, Scherbov S. The coming acceleration of global population ageing. Nature 2008; 451(7179):716-719.
(2) Robine J-M, Paccaud F. Nonagenarians and centenarians in Switzerland, 1860-2001: a demographic analysis. J Epidemiol Community Health 2005; 59(1):31-37.
(3) Gluckman P, Hanson M. The fetal matrix. Cambridge Univeristy Press; 2004.
(4) Vaupel JW, Gowan AE. Passage to Methuselah: some demographic consequences of continued progress against mortality. Am J Public Health 1986; 76(4):430-433.
(5) Wilmoth JR. The future of human longevity: a demographer's perspective. Science 1998; 280:395397.
(6) Tuljapurkar S, Li N, Boe C. A universal pattern of mortality decline in the G7 countries. Nature 2000; 405(6788):789-792.
(7) Oeppen J, Vaupel JW. Broken limits to life expectancy. Science 2002; 296(5570):1029-1031.
(8) Fries JF. Measuring and monitoring success in compressing morbidity. Ann Intern Med 2003; 139(5 Pt 2):455-459.
(9) Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, Brody J et al. A potential decline in life expectancy in the United States in the 21st Century. N Engl J Med 2005; 352(11):1138-1145.
(10) Wilmoth JR. Human longevity in historical perspective. In: Timiras PS, editor. Physiological Basis of Aging and Geriatrics. 3d ed. Boca Raton, FL: CRC Press; 2002. 11-24.
(11) Wilmoth JR, Deegan LJ, Lundstrom H, Horiuchi S. Increase of maximum life-span in Sweden, 1861-1999. Science 2000; 289(5488):2366-2368.
(12) Cheung SLK, Robine J-M, Tu E, Caselli G. Three dimensions of the survival curve: horizontalization, verticalization, and longevity extension. Demography 2005; 42(2):243-258.
(13) Kannisto V. Mode and dispersion of the length of life. Population 2001; 13(1):159-171.
(14) Lexis W. Sur la durée normale de la vie humaine et sur la théorie de la stabilité des rapports statistiques. Annales de Démographie Internationale 1878; 2(5):447-460.
(15) Kannisto V. Mode and dispersion of the length of life. Population 2001; 13(1):159-171.
(16) Livi-Bacci M. La population dans l'histoire de l'Europe. Paris: Seuil; 1999.
(17) Vandenbroucke JP. Survival and expectation of life from the 1400's to the present. A study of the Knighthood Order of the Golden Fleece. Am J Epidemiol 1985; 122(6):1007-1016.
(18) Mathers CD, Salomon JA, Murray CJ. Infant mortality is not an adequate summary measure of population health. J Epidemiol Community Health 2003; 57(5):319.
(19) Mesle F. [Gender gap in life expectancy: the reasons for a reduction of female advantage]. Rev Epidemiol Sante Publique 2004; 52(4):333-352.
(20) Bopp M, Spoerri A, Zwahlen M, Gutzwiller F, Paccaud F, Braun-Fahrlander C et al. Cohort Profile: The Swiss National Cohort--a longitudinal study of 6.8 million people. Int J Epidemiol 2008.
(21) Paccaud F, Sidoti Pinto C, Marazzi A, Mili J. Age at death and rectangularisation of the survival curve: trends in Switzerland, 1969-1994. J Epidemiol Community Health 1998; 52(7):412-415.
(22) Wilmoth JR. Demography of longevity: past, present, and future trends. Experimental Gerontology 2000; 35(9-10):1111-1129.
(23) Kramarow E, Lubitz J, Lentzner H, Gorina Y. Trends In The Health Of Older Americans, 19702005. Health Aff 2007; 26(5):1417-1425.
(24) Janssen F, Nusselder WJ, Looman CW, Mackenbach JP, Kunst AE. Stagnation in mortality decline among elders in the Netherlands. Gerontologist 2003; 43(5):722-734.
(25) Nusselder WJ, Mackenbach JP. Lack of improvement of life expectancy at advanced ages in The Netherlands. Int J Epidemiol 2000; 29(1):140-148.
(26) Bongaarts J. How long will we live? Population and Development Review 2006; 32(4):605-628.
(27) McKee M, Zatonski W. How the cardiovascular burden of illness is changing in eastern Europe. Evid Based Cardiovasc Med 1998; 2(2):39-41.
(28) Zatonski WA, Willett W. Changes in dietary fat and declining coronary heart disease in Poland: population based study. BMJ 2005 Jul 23;187-188.
(29) Rehm J, Sulkowska U, Manczuk M, Boffetta P, Powles J, Popova S et al. Alcohol accounts for a high proportion of premature mortality in central and eastern Europe. International Journal of Epidemiology 2007;dyl294.
(30) D'Amico M, Agozzino E, Biagino A, Simonetti A, Marinelli P. Ill-defined and multiple causes on death certificates--a study of misclassification in mortality statistics. Eur J Epidemiol 1999; 15(2):141-148.
(31) Wilmoth JR, Lundstrom H. Extreme longevity in five countries: presentation of trends with special attention to issues of data quality. Eur J Popul 1996; 12(1):63-93.
(32) Robine J-M, Saito Y, Jagger C. The emergence of extremely old people: the case of Japan. Exp Gerontol 2003; 38(7):735-739.
(33) An unprecedented increase in the number of centenarians. IUSSP seminar on longevity and health, Rockefeller University, New York, 20-22 October, 2003; 2003.
(34) Robine J-M, Saito Y. Survival beyond age 100: acceleration of the evolution in Japan. Population and Development Review 2003; 29(208):228.
(35) Cheung SL, Robine JM. Increase in common longevity and the compression of mortality: the case of Japan. Popul Stud (Camb ) 2007; 61(1):85-97.
(36) Summary measures of population health: Concepts. ethics, measurement and applications. Geneva: World Health Organization; 2002.
(37) Fries JF. Aging, natural death, and the compression of morbidity. N Engl J Med 1980; 303(3):130135.
(38) Fries JF. Frailty, heart disease, and stroke: The compression of morbidity paradigm. American Journal of Preventive Medicine 2005; 29(5, Supplement 1):164-168.
(39) Mathers CD. Towards valid and comparable measurement of population health. Bull World Health Organ 2003; 81(11):787-788.
(40) Sanders BS. Measuring Community Health Levels. Am J Public Health Nations Health 1964; 54(7):1063-1070.
(41) Sullivan DF. A single index of mortality and morbidity. HSMHA Health Rep 1971; 86(4):347-354.
(42) Mathers CD, Robine JM. How good is Sullivan's method for monitoring changes in population health expectancies? Journal of Epidemiology and Community Health 1997; 51(1):80-86.
(43) Ustun TB, Chatterji S, Bickenbach J, Kostanjsek N, Schneider M. The International Classification of Functioning, Disability and Health: a new tool for understanding disability and health. Disabil Rehabil 2003; 25(11-12):565-571.
(44) Katz S. Assessing self-maintenance: activities of daily living, mobility, and instrumental activities of daily living. J Am Geriatr Soc 1983; 31(12):721-727.
(45) Mathers CD, Murray CJ, Salomon JA, Sadana R, Tandon A, Lopez AD et al. Healthy life expectancy: comparison of OECD countries in 2001. Aust N Z J Public Health 2003; 27(1):5-11.
(46) Höpflinger F, Hugentobler V. Les besoins en soins des personnes âgées en Suisse. Berne: Cahiers de 1'Observatoire Suisse de la Santé; 2003.
(47) Office Fédéral de la Statistique. Statistique de la santé. www bfs admin ch [ 2007
(48) Lafortune G. Are disability rates among elderly people declining in OECD countries? A progress report on the current data collection and a preliminary assessment OECD costs of care for elderly populations. Paris: OECD; 2006.
(49) Minicuci N, Noale M, Pluijm SMF, Zunzunegui MV, Blumstein T, Deeg DHJ et al. Disability-free life expectancy: a cross-national comparison of six longitudinal studies on ageing. The CLESA project. Eur J Ageing 2004; 1:37-44.
(50) Perenboom RJ, Van Herten LM, Boshuizen HC, van den Bos GA. Trends in disability-free life expectancy. Disabil Rehabil 2004; 26(7):377-386.
(51) Wray LA, Blaum CS. Explaining the role of sex on disability: a population-based study. Gerontologist 2001; 41(4):499-510.
(52) Schoeni RF, Liang J, Bennett J, Sugisawa H, Fukaya T, Kobayashi E. Trends in old-age functioning and disability in Japan, 1993-2002. Popul Stud (Camb ) 2006; 60(1):39-53.
(53) Crimmins EM, Saito Y, Ingegneri D. Changes in life expectancy and disability-free life expectancy in the United States. Population and Development Review 1989; 15:235-267.
(54) Freedman VA, Martin LG, Schoeni RF. Recent trends in disability and functioning among older adults in the United States: a systematic review. JAMA 2002; 288(24):3137-3146.
(55) Manton KG, Gu X, Lamb VL. Change in chronic disability from 1982 to 2004/2005 as measured by long-term changes in function and health in the U.S. elderly population. Proc Natl Acad Sci U S A 2006; 103(48):18374-18379.
(56) Crimmins EM, Saito Y. Trends in healthy life expectancy in the United States, 1970-1990: gender, racial, and educational differences. Soc Sci Med 2001; 52(11):1629-1641.
(57) Cai L, Lubitz J. Was there compression of disability for older Americans from 1992 to 2003? Demography 2007; 44(3):479-495.
(58) Bopp M, Minder CE. Mortality by education in German speaking Switzerland, 1990-1997: results from the Swiss National Cohort. Int J Epidemiol 2003; 32(3):346-354.
(59) Catalano R. Economic antecedents of mortality among the very old. Epidemiology 2002; 13(2):133137.
(60) Mathers CD, Murray CJ, Lopez AD, Sadana R, Salomon JA. Global patterns of healthy life expectancy for older women. J Women Aging 2002; 14(1-2):99-117.
(61) Li N, Lee R. Coherent mortality forecasts for a group of populations: An extension of the Lee-Carter method. Demography 2005; 42(3):575-594.
(62) Carnes BA, Olshansky SJ, Grahn D. Biological evidence for limits to the duration of life. Biogerontology 2003; 4(1):31-45.
(63) Kannisto V, Lauritsen J, Thatcher R, Vaupel JW. Reductions in mortality at advanced ages: Several decades of evidence from 27 countries. Population and Development Review 1994; 20(4):793-810.
(64) Harman D. Aging: Overview. Ann NY Acad Sci 2001; 928(1):1-21.
(65) Olshansky SJ, Carnes BA, Cassel C. In search of Methuselah: estimating the upper limits to human longevity. Science 1990; 250(634):640.
(66) Cutler DM, Huckman RS. Technological development and medical productivity: the diffusion of angioplasty in New York state. Journal of Health Economics 2003; 22(2):187-217.
(67) Cutler DM, McClellan M. Is Technological Change In Medicine Worth It? Health Aff 2001; 20(5):1129.
(68) Janssen F, Kunst A. The choice among past trends as a basis for the prediction of future trends in old-age mortality. Popul Stud (Camb ) 2007; 61(3):315-326.
(69) Lee RD, Carter L. Modelling and forecasting the time series of US mortality. J Am Stat Assoc 1992; 87:659-671.
(70) Lee RD. The Lee-Carter method of forecasting mortality, with various extensions and applications. N Am Actuarial J 2000; 4:80-93.
(71) Wilson C. On the Scale of Global Demographic Convergence 1950-2000. Population and Development Review 2001; 27(1):155-172.
(72) Carter L, Lee R. Modeling and forecasting U.S. sex differentials in mortality. Int J Forecast 1992; 8(3):393-411.
(73) Booth H, Maindonald J, Smith L. Applying Lee-Carter under conditions of variable mortality decline. Popul Stud (Camb ) 2002; 56(3):325-336.
(74) World population to 2300. Department of Economic and Social Affairs, editor. 2004. New York, United Nations. Ref Type: Report
(75) Booth H. Demographic forecasting: 1980 to 2005 in review. International Journal of Forecasting 2006; 22:547-581.
(76) Lee RB, Miller T. Evaluating the performance of the Lee-Carter method for forecasting mortality. Demography 2001; 38(4):537-549.
(77) Bongaarts J. Long-range trends in adult mortality: models and projection methods. Demography 2005; 42(1):23-49.
(78) Pedroza C. A Bayesian forecasting model: predicting U.S. male mortality. Biostat 2006; 7(4):530550.
(79) Babel B, Bomsdorf E, Schmidt R. Future life expectancy in Australia, Europe, Japan, and North America. Journal of Population Research 2007; 24(1):119-131.
(80) Oeppen J, Vaupel JW. Broken limits to life expectancy. Science 2002; 296(5570):1029-1031.
(81) Uemura K. Excess mortality ratio with reference to the lowest age-sex-specific death rates among countries. World Health Stat Q 1989; 42(1):26-41.
(82) Vallin J, Mesle F. Les plus faibles mortalités. 2008.

Ref Type: Unpublished Work
(83) Wilmoth JR. Are mortality projections always more pessimistic when disaggregated by cause of death? Math Popul Stud 1995; 5(4):293-319, 377.
(84) Tsai SP, Lee ES, Hardy RJ. The effect of a reduction in leading causes of death: potential gains in life expectancy. Am J Public Health 1978; 68(10):966-971.
(85) Nusselder WJ, van d, V, van Sonsbeek JL, Lenior ME, van den Bos GA. The elimination of selected chronic diseases in a population: the compression and expansion of morbidity. Am J Public Health 1996; 86(2):187-194.
(86) Cheung SL, Robine JM, Paccaud F, Marazzi A. Dissecting the compression of mortality in Switzerland, 1876-2005. 2007.
Ref Type: Unpublished Work
(87) Cheung SL, Robine JM, Caselli G. The use of cohort and period data to explore changes in adult longevity in low mortality countries. 2008.
Ref Type: Unpublished Work
(88) Olshansky SJ. On forecasting mortality. Milbank Q 1988; 66(3):482-530.
(89) Kesteloot H, Sans S, Kromhout D. Evolution of all-causes and cardiovascular mortality in the agegroup 75-84 years in Europe during the period 1970-1996; a comparison with worldwide changesKESTELOOT2002. Eur Heart J 2002; 23(5):384-398.
(90) Tunstall-Pedoe H, Connaghan J, Woodward M, Tolonen H, Kuulasmaa K. Pattern of declining blood pressure across replicate population surveys of the WHO MONICA project, mid-1980s to mid1990s, and the role of medication. BMJ 2006; 332(7542):629-635.
(91) Galobardes B, Costanza MC, Bernstein MS, Delhumeau CH, Morabia A. Trends in risk factors for the major "lifestyle-related diseases" in Geneva, Switzerland, 1993-2000. Ann Epidemiol 2003; 13(7):537-540.
(92) Koek HL, de Bruin A, Gast A, Gevers E, Kardaun JWPF, Reitsma JB et al. Decline in incidence of hospitalisation for acute myocardial infarction in the Netherlands from 1995 to 2000. Heart 2006; 92(2):162-165.
(93) Tunstall-Pedoe H, Kuulasmaa K, Tolonen H, Davidson M, Mendis S, with 64 other contributors for The WHO MONICA Project. MONICA Monograph and Multimedia Sourcebook. Geneva: World Health Organization; 2003.
(94) Kuulasmaa K, Tunstall-Pedoe H, Dobson A, Fortmann S, Sans S, Tolonen H et al. Estimation of contribution of changes in classic risk factors to trends in coronary-event rates across the WHO MONICA Project populations. Lancet 2000; 355(9205):675-687.
(95) Tunstall-Pedoe H, Kuulasmaa K, Mahonen M, Tolonen H, Ruokokoski E, Amouyel P. Contribution of trends in survival and coronary-event rates to changes in coronary heart disease mortality: 10year results from 37 WHO MONICA project populations. Monitoring trends and determinants in cardiovascular disease. Lancet 1999; 353(9164):1547-1557.
(96) Wietlisbach V, Paccaud F, Rickenbach M, Gutzwiller F. Trends in cardiovascular risk factors (19841993) in a Swiss region: results of three population surveys. Prev Med 1997; 26(4):523-533.
(97) Eichholzer M, Camenzind-Frey E, Matzke A, et al. Cinquième rapport sur la nutrition en Suisse. Bern: Bundesamt für Gesundheit; 2005.
(98) Hooper L, Griffiths E, Abrahams B, Alexander W, Atkins S, Atkinson G et al. Dietetic guidelines: diet in secondary prevention of cardiovascular disease (first update, June 2003). Journal of Human Nutrition and Dietetics 2004; 17(4):337-349.
(99) Appel LJ. Nonpharmacologic therapies that reduce blood pressure: a fresh perspective. Clin Cardiol 1999; 22(7 Suppl):III1-III5.
(100) Appel LJ. Salt reduction in the United States. BMJ 2006; 333(7568):561-562.
(101) Vollenweider P, Hayoz D, Preisig M, Pécoud A, Warteworth D, Mooser V et al. L'état de santé des Lausannois : premiers résultats de l'étude CoLaus. Revue Médicale Suisse 2006;(86).
(102) O'Flaherty M, Ford E, Allender S, Scarborough P, Capewell S. Coronary heart disease trends in England and Wales from 1984 to 2004: concealed levelling of mortality rates among young adults. Heart 2008; 94(2):178-181.
(103) Liu PY, Death AK, Handelsman DJ. Androgens and Cardiovascular Disease. Endocr Rev 2003; 24(3):313-340.
(104) Klenk J, Rapp K, Buchele G, Keil U, Weiland SK. Increasing life expectancy in Germany: quantitative contributions from changes in age- and disease-specific mortality. Eur J Public Health 2007; 17(6):587-592.
(105) Preston SH, Wang H. Sex mortality differences in the United States: the role of cohort smoking patterns. Demography 2006; 43(4):631-646.
(106) Goldman DP, Shang B, Cutler DM, Joyce GF. The value of elderly disease prevention. Berkeley Electronic Press; 2006.
(107) Nyholm M, Merlo J, Rastam L, Lindblad U. Overweight and all-cause mortality in a Swedish rural population: Skaraborg Hypertension and Diabetes Project. Scand J Public Health 2005; 33(6):478486.
(108) Cutler DM, Glaeser EL, Rosen AB. Is the US population behaving healthier? National Bureau of Economic Research, Cambrridge, Massachusetts, editors. Working Paper 13013. 2007. Ref Type: Report
(109) Danon-Hersch N, et al., Chiolero A, Shamlaye C, Paccaud F, Bovet P. Decreasing association between body mass index and blood pressure over time. Epidemiology 2007; 18(4):493-500.
(110) Mitrou PN, Kipnis V, Thiebaut ACM, Reedy J, Subar AF, Wirfalt E et al. Mediterranean Dietary Pattern and Prediction of All-Cause Mortality in a US Population: Results From the NIH-AARP Diet and Health Study. Arch Intern Med 2007; 167(22):2461-2468.
(111) Waijers PM, Ocke MC, van Rossum CT, Peeters PH, Bamia C, Chloptsios Y et al. Dietary patterns and survival in older Dutch women. Am J Clin Nutr 2006; 83(5):1170-1176.
(112) Christensen K, Johnson TE, Vaupel JW. The quest for genetic determinants of human longevity: challenges and insights. Nat Rev Genet 2006; 7(6):436-448.
(113) vB Hjelmborg J, Iachine I, Skytthe A, Vaupel JW, McGue M, Koskenvuo M et al. Genetic influence on human lifespan and longevity. Hum Genet 2006; 119(3):312-321.
(114) Drenos F, Westendorp R, Kirkwood T. Trade-off Mediated Effects on the Genetics of Human Survival Caused by Increasingly Benign Living Conditions. Biogerontology 2006; 7(4):287-295.
(115) Stock GB, Callahan D. Would Doubling the Human Lifespan Be a Net Positive or Negative for Us, Either as Individuals or as a Society? Point-Counterpoint. Ann NY Acad Sci 2005; 1055(1):207-218.
(116) Goldman DP, Shang B, Bhattacharya J, Garber AM, Hurd M, Joyce GF et al. Consequences of health trends and medical innovation for the future elderly. Health Affairs (Millwood) 2005; 24( Suppl 2):W5R5-W5R17.
(117) Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, Brody J et al. A potential decline in life expectancy in the United States in the 21st Century. The New England Journal of Medicine 2005; 352(11):1138-1145.
(118) Diehr P, Derleth A, Cai L, Newman AB. The effect of different public health interventions on longevity, morbidity, and years of healthy life. BMC Public Health 2007; 7:52.
(119) Greider CW. Telomeres and senescence: The history, the experiment, the future. Current Biology 1998; 8(5):R178-R181.
(120) Kirkwood TBL. A systematic look at an old problem. Nature 2008; 451(7179):644-647.
(121) Ruiz-Torres A, Beier W. On maximum human life span: interdisciplinary approach about its limits. Adv Gerontol 2005; 16(14):20.
(122) Ruiz-Torres A, Soares de Melo Kirzner M. Ageing and Longevity Are Related to Growth Hormone/Insulin-Like Growth Factor-1 Secretion. Gerontology 2002; 48(6):401-407.
(123) Everitt AV, Le Couteur DG. Life extension by calorie restriction in humans. Ann N Y Acad Sci 2007; 1114:428-433.
(124) Tosato M, Zamboni V, Ferrini A, Cesari M. The aging process and potential interventions to extend life expectancy. Clin Interv Aging 2007; 2(3):401-412.
(125) Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. PLoS Med 2006; 3(11): e 442.
(126) Long-range population projections. United Nations, Population Division, editors. 21-8-2003. New York. Ref Type: Report
(127) Bhattacharya J, Cutler D.M., Goldman DP, Hurd MD, Joyce GF, Lakdawalla D et al. Disability forecasts and future Medicare costs. In: Garber AM, editor. Frontiers in Health Policy Research. 7 ed. MIT Press; 2004. 75-94.
(128) Lakdawalla DN, Goldman DP, Shang B. The health and cost consequences of obesity among the future elderly. Health Aff 2005;hlthaff.
(129) Sturm R, Ringel JS, Andreyeva T. Increasing obesity rates and disability trends. Health Aff (Millwood) 2004; 23(2):199-205.
(130) Wolf DA, Hunt K, Knickman J. Perspectives on the recent decline in disability at older ages. Milbank Q 2005; 83(3):365-395.
(131) Ezzati M, Hoorn SV, Rodgers A, Lopez AD, Mathers CD, Murray CJ. Estimates of global and regional potential health gains from reducing multiple major risk factors. Lancet 2003; 362(9380):271-280.
(132) Shaw C. Fifty years of United Kingdom national population projections: how accurate have they been? Popul Trends 2007;(128):8-23.
(133) Robine J-M, Michel J-P. Looking forward to a general theory on population aging. J Gerontol A Biol Sci Med Sci 2004; 59(6):M590-M597.
(134) Mathers CD, Murray CJ, Ezzati M, Gakidou E, Salomon JA, Stein C. Population health metrics: crucial inputs to the development of evidence for health policy. Popul Health Metr 2003; 1(1):6.


[^0]:    b http://hem.waw.pl

[^1]:    c www.who.int/whosis

[^2]:    d http://www who int/whosis/database [Accessed Feb 21st , 2008]
    ${ }^{\text {e http://www who int/whosis/database [Accessed Feb 21st , 2008] }}$

