

Chapter 18

How Useful Are the Causes of Death When Extrapolating Mortality Trends. An Update



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Old age and adult mortality have over the last decades enjoyed a remarkable decline throughout the western world, posing the researcher with new challenges and opening up fresh horizons in life expectancy trends. The recent drop in mortality may be largely traced to the unexpected decline in cardiovascular diseases and certain cancers. Thus it could be hoped that in the future these trends would continue and extend to include other causes where, for the moment, little change has occurred. Such a hypothesis is all the more realistic in view of the fact that recent changes are linked, not just to advances in more efficacious medical treatment, but also to a growing awareness on the part of the general public regarding questions of health and the crucial role played by life style and behaviour. These include improved dietary habits, for example, a better attitude to risk factors, particularly to smoking, alcohol abuse, dangerous driving, etc. This awareness, which prevails among more recent, well-informed and better educated cohorts, not only produces immediate results, but maybe even more so in the future, should this spare coming generations the accumulation of risks which were and continue to be the burden particularly of older cohorts.

These considerations have increasingly encouraged researchers to refute the timid claims regarding future mortality generally made by Institutes of Statistics when

This paper is an update of Caselli and Vallin (1999a) (in French) and Caselli and Vallin (1999b) (in English).

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T. Bengtsson, N. Keilman (eds.), *Old and New Perspectives on Mortality*

Forecasting, Demographic Research Monographs,

https://doi.org/10.1007/978-3-030-05075-7_18

producing population estimates (Vallin 1989, 1992; Vallin and Meslé 1989; Meslé 1993; Caselli 1993; van Poppel and de Beer 1996) and to seek to take better account of more recent progress when estimating future mortality trends. This has led to including causes of death as a component of mortality (Benjamin and Overton 1985; Caselli and Egidi 1992; Wilmoth 1996) and to seek methods to account for the cohort effect, and indeed to combine the two at times (Caselli 1996).

More complex data or more sophisticated methods are not themselves a guarantee for better results. Numerous experiences of this nature have ended up more as a disappointment than anything else. Our goal here is to focus on the advantages and disadvantages of taking causes of death into consideration when making mortality estimates and to explore the results of the different possible methods. It is beyond the scope of this paper to take a stand regarding the present debate on life expectancy outcomes or even to contribute to this. Rather, our task is to establish whether, by refining the methods, the results of a simple extrapolation of past trends could be improved, without making future hypotheses and irrespective of those directly stemming from an analysis of past trends.

The first obstacle one meets when projecting mortality trends cause by cause depends on the fact that even if there is one cause for which mortality increases, this will inevitably, sooner or later, depending on the relative importance of this cause, lead to a general increase in mortality for all causes, the overall perspective thus being more pessimistic than that yielded by extrapolating total mortality, as we will show below. In other words, it is almost not worthwhile considering mortality outlooks by cause if we are unable to “predict” the inflexion points or the changes in the direction of the evolution curve. Therefore the question which must be posed is if by some means, when using the model of past trends, we can predict such changes in the trends.

To do so we will focus on the England & Wales male population and on mortality risks between 60 and 85 years. Opting for this population will help focus on mortality trends among the elderly, these being more sensitive to changes described above, and elude the thorny question of life expectancy thresholds, which to our mind calls for an entirely different approach.

When dealing with causes of death, for the sake of clarity, obviously only a limited number of groups of specific causes may be referred to, albeit with adequately diverse recent trends to be able to highlight the difficulties involved and evaluate the possible solutions. Five sufficiently descriptive causes were selected:

- cardiovascular diseases,
- bronchial and lung cancers,
- digestive cancers,
- other tumours,
- other diseases and violent deaths.

This classification is particularly suited to England & Wales as it includes a cause, bronchial and lung cancer, for which male mortality underwent a sharp rise followed by a decline, from the 1970s.

A reference period also had to be selected to elaborate a model of past trends. It was decided to focus alternatively on a long series, 1950–2000, which includes the period where mortality from bronchial and lung cancers was steep, as well as a shorter series (1981–2000), showing more recent trends.

The estimations made were obtained by extrapolating the logarithms of age specific mortality rates, which vary according to the number and types of variables considered to adapt the data sources.

Having, first of all, highlighted the absurdity of extrapolations based on a simple linear adjustment of a chronological series of age specific mortality rates (referred to here as the “*linear*” model), we will then try to obtain better results by gradually refining the modelling of the data series. Thus three increasingly complex models will be explored. First, while keeping to the approach where an independent adjustment is made for each chronological series of rates by age, an effort will be made to improve the outcome by selecting the best curve possible to adjust the data series (referred to here as the “*least squares*” method). Then, a model elaborated by Ronald Lee and Lawrence Carter, referred to here as “*Lee-Carter*”, will be used, where the logarithm of age specific mortality rates is a function of age as well as of period. Finally, thanks to a solution described elsewhere (Caselli 1993; Burgio and Frova 1995), a third component, that is the cohort effect, will be considered, using the “*APC*” model (age, period, and cohort).

However, to judge the comparable validity of these different approaches, extrapolations using older series must be compared with reality as it occurred. We will do this by using data from 1950–1980 to make projections for 1981–2000, which can then be compared with real mortality trends.

18.1 Extrapolation of Mortality by Cause Risks Absurdity

Figure 18.1 describes the results of a simple logarithmic extrapolation for mortality rates for all causes (the “*linear*” model), for each of the five age groups considered here (from 60–64 to 80–84 years), until the year 2050, based on data for 1950–2000, and shows a mortality projection which ignores individual trends for each cause of death. Average life expectancy between 60 and 85 years for an English male passes from 18.1 years in 2000 to 20.0 years in 2050, in other words a two-year gain.

Figure 18.2, on the other hand, illustrates the results of summing similar type extrapolations performed separately for each group of causes. A systematic increase in total mortality immediately occurs at older ages between 75 and 85 years, while the trend of reduction for ages between 60 and 75 years is less important than that obtained from the extrapolation for all causes, to such an extent that the average number of years one could expect to live between 60 and 85 years remains quite stable (around 18.0) over all the projection period (Table 18.5). Not only is this absent increase in survival at older ages hard to believe, it also appears somewhat absurd as sooner or later it yields mortality rates twice as high as the present for the highest ages. The problem, as we know, stems from the fact that causes of death are

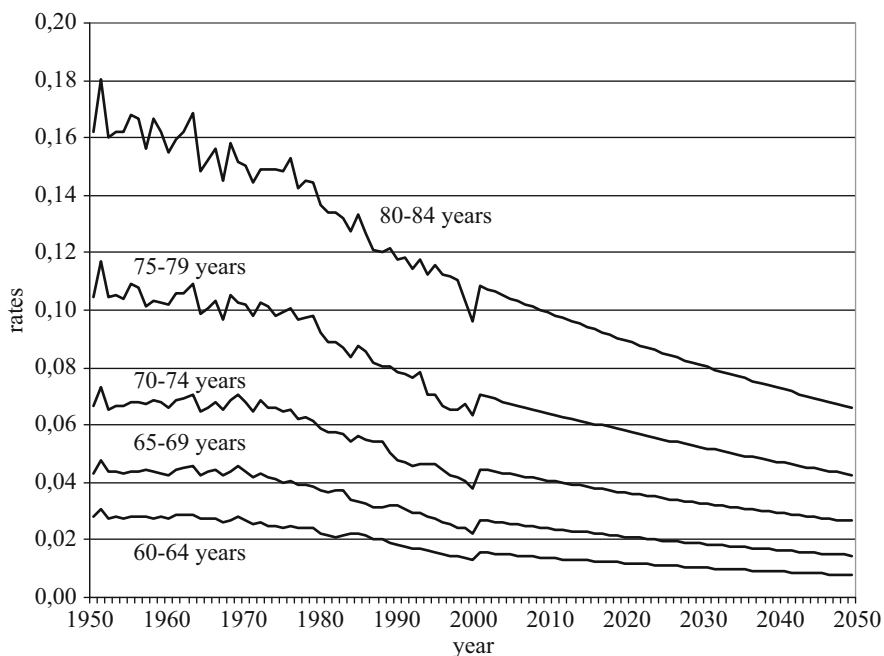


Fig. 18.1 Extrapolation of mortality rates for all causes by age group 2001–2050, based on a “linear” adjustment of data for 1950–2000 (England and Wales, males)

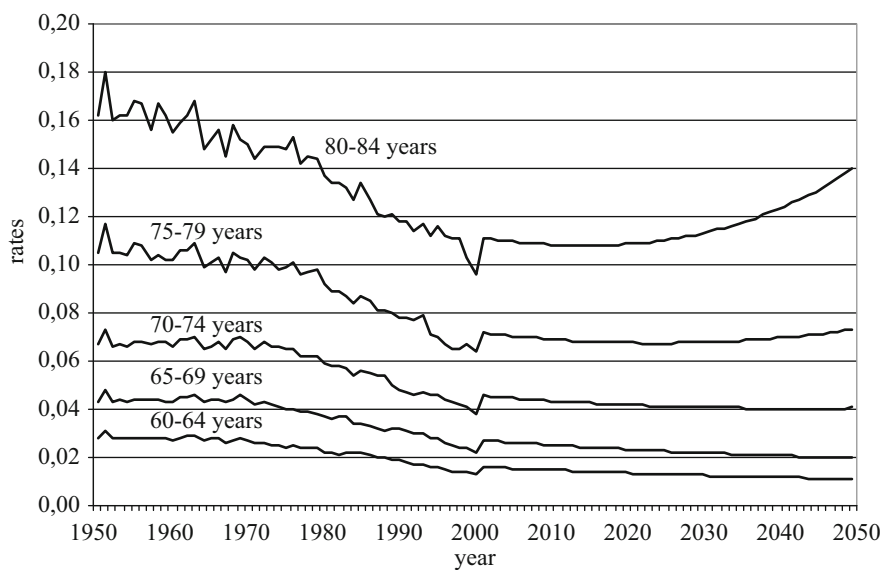


Fig. 18.2 Mortality trends by age group 2001–2050 obtained by summing specific rates by cause extrapolated using a “linear” adjustment of 1950–2000 data (England and Wales, males)

included where mortality trends were rising during a large part of the period of reference. This is the case with bronchial and lung cancers, as well as “other tumours”, where unfavourable trends are contrasted with favourable trends in cardiovascular diseases and digestive cancers (Fig. 18.3).

According to this outline, the impact of bronchial and lung cancers on total mortality comparative rates at 60–84 years would rise from 9.9% in 2000 to 30.9% in 2050, while that of cardiovascular diseases would fall from 45.7 to 23.4% (Table 18.1)!

No doubt this example is too extreme. Obviously, for England & Wales no one would dream of extrapolating bronchial and lung cancer mortality trends for

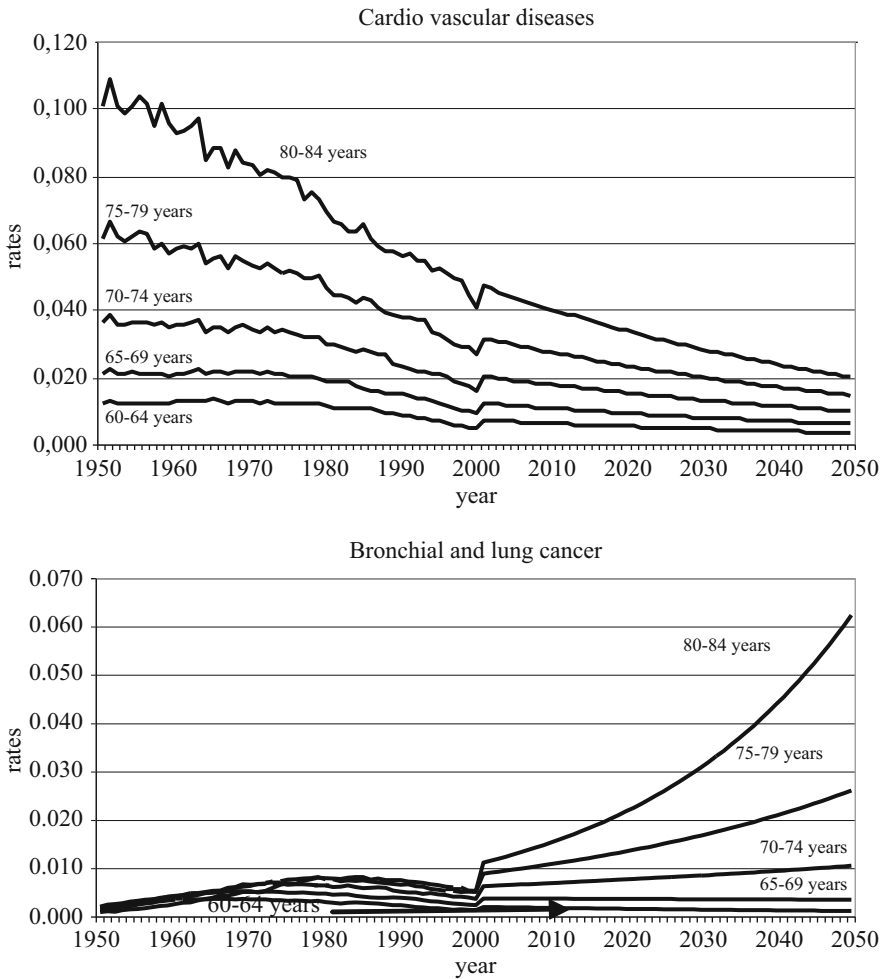


Fig. 18.3 Extrapolation of mortality rates by age group 2001–2050, for 4 groups of causes, the trends of which are in contrast, using a “linear” adjustment of 1950–2000 data (England and Wales, males)

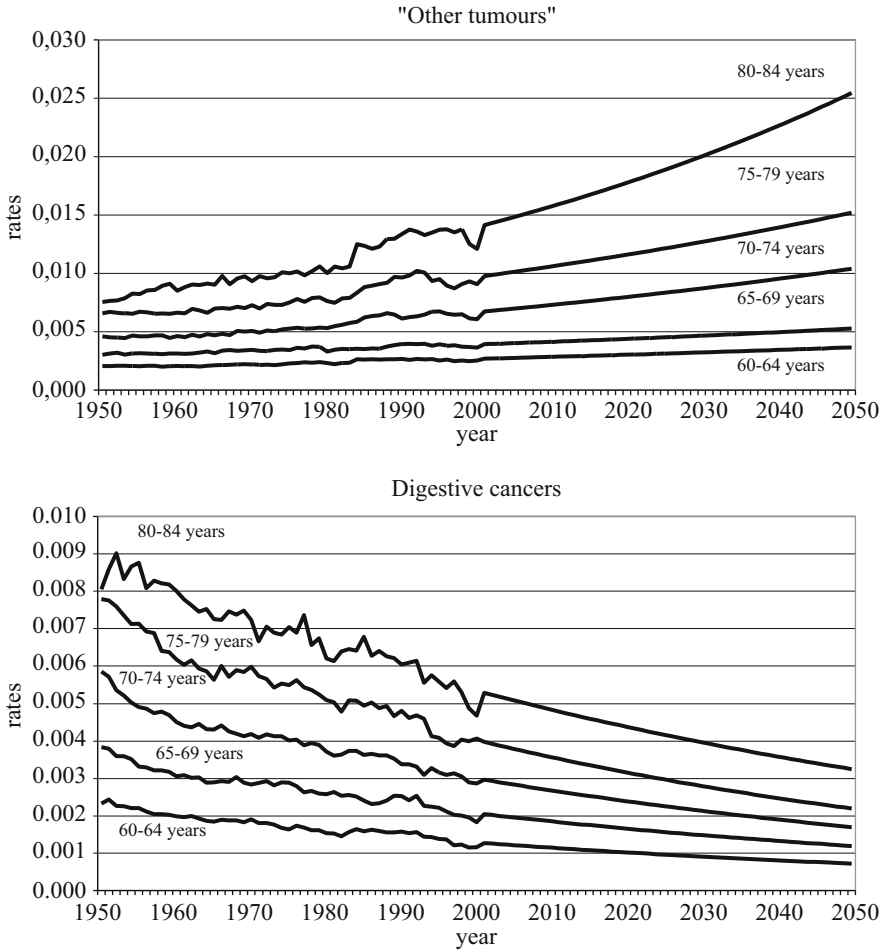


Fig. 18.3 (continued)

Table 18.1 Percent of each group of causes as part of the standardized mortality rates for all causes at 60–84 years, in 2000 and 2050, following an extrapolation using a “linear” adjustment of 1950–2000 rates, and then 1981–2000 rates (England & Wales, males)

Group of causes of death	2000	2050	
		Base 1950–2000	Base 1981–2000
Cardiovascular diseases	45.7	23.4	16.2
Bronchial and lung cancers	9.9	30.9	3.6
Digestive cancers	7.7	4.0	5.6
Other tumours	16.9	24.0	37.6
Other diseases and violent deaths	19.8	17.7	37.1
TOTAL	100.0	100.0	100.0

2001–2050 by a linear adjustment of the entire period 1950–2000, when in fact a reversal trend occurred in the early 1970's.

Thus fresh calculations were made, restricting the adjustment of past trends to the period 1981–2000. The results are visibly improved for bronchial and lung cancer, as this time mortality for this cause decidedly follows a downward trend for all age groups (Fig. 18.4). However, the problem is still not solved as mortality from “other tumours” increases for all ages. Therefore, in the final calculation the sum of the extrapolations by cause generate a reduction in overall mortality (Fig. 18.5). There is no doubt, given this scenario, that the total number of years lived between 60 and 84 years increases, rising from 18.1 years in 2000 to 20.2 years in 2050. However, this rise is less rapid than when total mortality is extrapolated (reaching 22.0 years), but continues to fall if the extrapolation continues beyond 2050 and does not top for the oldest old high levels, as in the previous instance.

18.2 Would More Sophisticated Methods Be Any Better?

Could we do any better with more sophisticated methods? The first attempt to be made, while keeping to the approach which adjusts only one dimension of mortality (chronology), is to choose, should it exist, an adjustment curve which is more appropriate than a simple straight line.

18.2.1 *A Better Adjustment of Chronological Series of Rates by Age*

Here a choice was made between four classic functions (straight line, parabola, hyperbole, logistic) which offered the least sum of the square distances to the observed values being selected. Thus, for bronchial and lung cancers, for example, the parabolic method was opted for, as this would effectively appear to prolong more satisfactorily observed trends in mortality by age (Fig. 18.6).

Again, the fact that mortality tends towards zero is obviously disputable. Unfortunately, for “other tumours”, the “least-squares” are obtained by the straight line method and we come up again against the same problem which arose previously, albeit not quite as quickly, where a major cause such as bronchial and lung cancers has been totally eliminated. Thus, we have merely delayed the march of time towards the unlimited increase in mortality for older ages (as in Table 18.2), but in 2050, the mortality profile by cause is much more deformed than in the previous instance, with cardiovascular diseases are no longer at the top of the list, falling from 46% in 2000 to 5.5% in 2050, while the impact of “other tumours” is remarkably increased, from 17% to 55%, keeping the lead, to such an extent that the role of bronchial and lung cancer is eliminated.

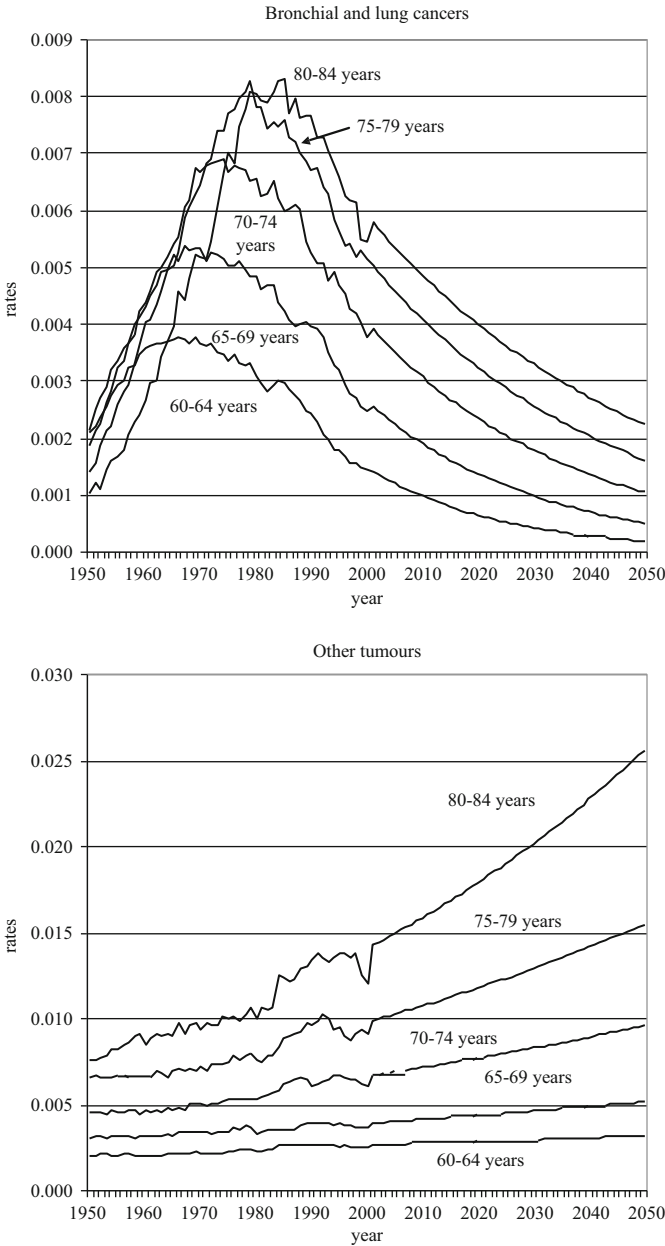


Fig. 18.4 Extrapolation of mortality rates by age group 2001–2050, for bronchial and lung cancers and for “other tumours”, using a “linear” adjustment of 1981–2000 data (England & Wales, males)

If the data observed are only adjusted for the most recent period (1981–2000), bronchial and lung cancers remain largely unchanged, but this tends to modify the changes foreseen for “other tumours” and thus delay the moment in which these raise

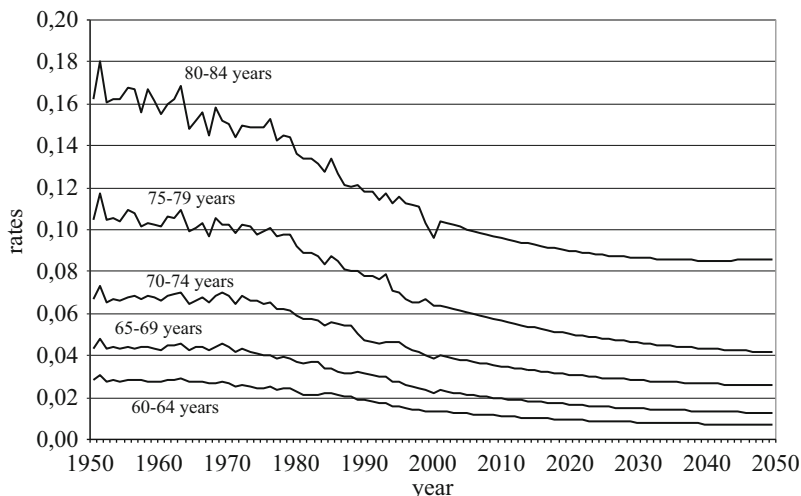


Fig. 18.5 Mortality trends by age group 2001–2050, obtained by summing rates by cause extrapolated using a “linear” adjustment of 1981–2000 data (England & Wales, males)

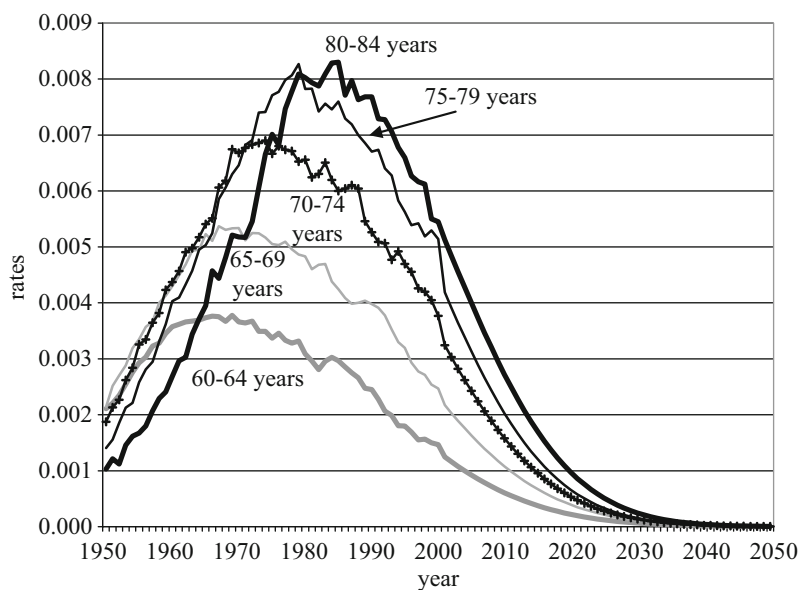


Fig. 18.6 Extrapolation of age group mortality rates for bronchial and lung cancers, using a “least-squares” adjustment of 1950–2000 data (England & Wales, males)

the sum of the total rates by cause. The mortality profile by cause for 2050 is thus considerably modified, with an increase to 16% for cardiovascular diseases and a decrease to 38% for “other tumours”.

Table 18.2 Percent of each group of cause as part of the standardized mortality rates for all causes at 60–84 years, in 2000 and in 2050, after a “least-squares” extrapolation of 1950–2000 rates and then 1981–2000 rates (England & Wales, males)

Group of causes of death	2000	2050	
		Base 1950–2000	Base 1981–2000
Cardiovascular diseases	45.7	5.5	15.9
Bronchial and lung cancers	9.9	0.0	3.5
Digestive cancers	7.7	6.9	5.6
Other tumours	16.9	55.3	37.6
Other diseases and violent deaths	19.8	32.3	37.4
TOTAL	100.0	100.0	100.0

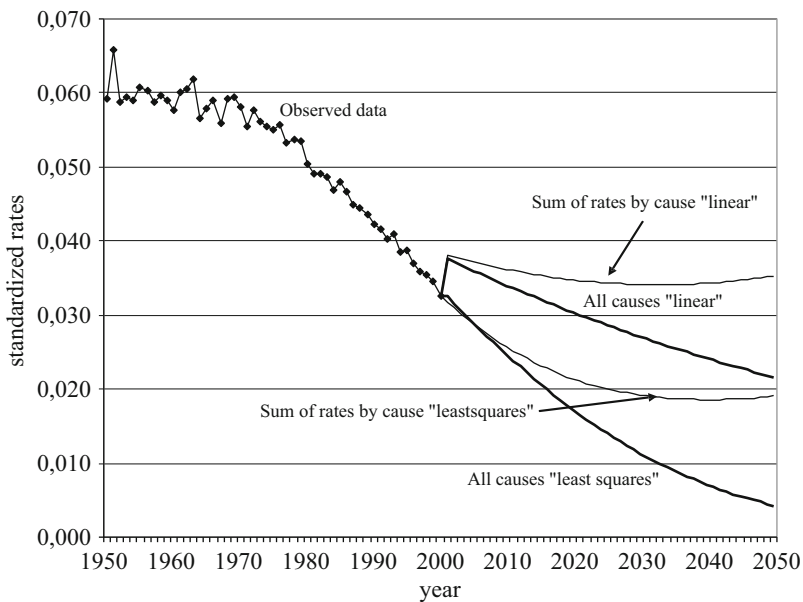


Fig. 18.7 Results compared, in terms of standardized mortality rates at 60–84 years, “linear” and “least-squares” models, for mortality for “all causes” and the “sum of rates by cause” based on observed data for 1950–2000 (England & Wales, males)

Figures 18.7, 18.8, and 18.9 report the different outcomes obtained to date regarding standardized mortality rates at 60–84 years, referring alternatively to the periods 1950–2000 and 1981–2000.

Based on data observed for 1950–2000, the improvement gained by using the “least-squares” method to adjust the curve, generates overall within the limits of the extrapolation period explored here, a trend in the sum of mortality rates by cause which is clearly less preposterous than that obtained with a strictly “linear” model even though still far removed from that yielded by the direct extrapolation of mortality for all causes. According to the sum of the extrapolations by cause, the

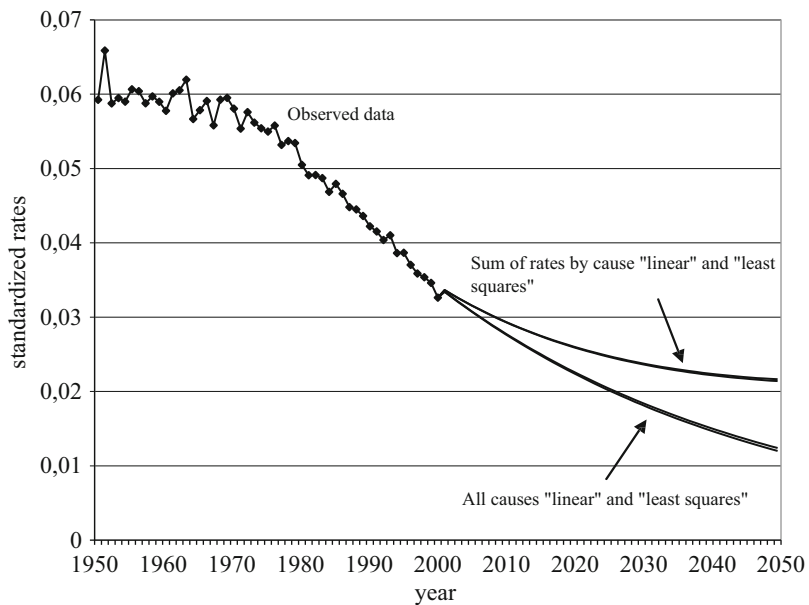


Fig. 18.8 Compared results, in terms of standardized mortality rates at 60–84 years, “linear” and “least-squares” models, for mortality for “all causes” and the “sum of rates by cause” based on observed data for 1981–2000 (England & Wales, males)

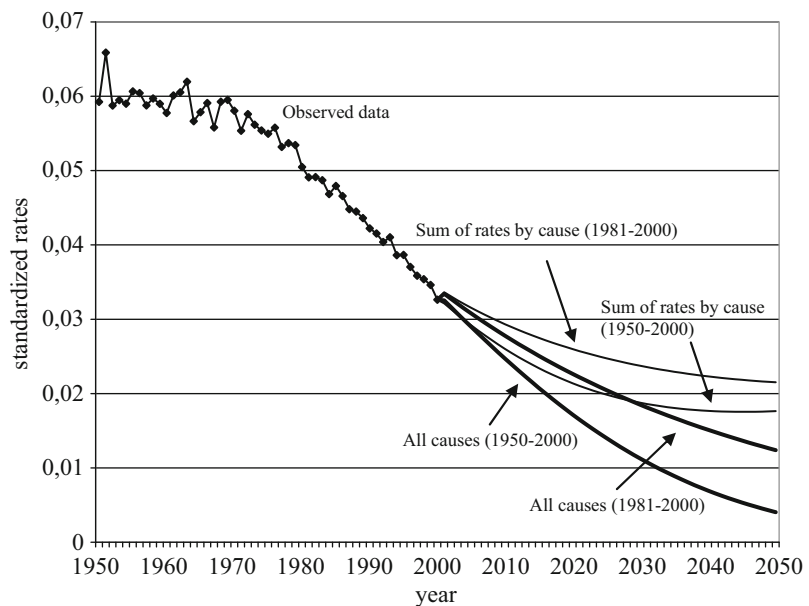


Fig. 18.9 Results, in terms of standardized mortality rates at 60–84 years, of the Lee-Carter model for mortality for all causes and the sum of the rates by cause, according to the reference period used (1950–2000 and 1981–2000) (England & Wales, males)

mean number of years lived between 60 and 85 years rises from 18.1 in 2000 to 20.6 for 2050, compared with 24.0 obtained with the direct extrapolation of mortality for all causes.

Nonetheless, it should be pointed out that the reference period used for the adjustment can notably change the end result. If this is limited to the most recent period, the role (favourable) played by trends in cardiovascular diseases is more quickly obliterated than that (unfavourable) played by “other tumours” (Fig. 18.8). Surprisingly, in 2050, by summing the extrapolations by cause the average number of years lived would be exactly the same as in the previous instance (20.2 years), and this time, too, it is lower than that obtained by a direct extrapolation of mortality for all causes (22.1).

One could, while maintaining the same approach, whereby a model is elaborated using a period component of age rates, attempt a further refinement, by choosing for each cause of death not only the best adjustment function but also the reference period which would best reflect recent trends. The limits of such an approach emerge fairly quickly, which risks being over-subjective and in any case fails to solve the problem of the impossibility of foreseeing an eventual reversal of the upward trends in “other tumours”.

18.2.2 “Age-Period” Adjustment (Lee-Carter Model)

In order to continue, more complex models are needed, which take into account other aspects of mortality, possibly able to anticipate trends already germinating in certain available data sources. First of all, using the model proposed by Ronald Lee and Lawrence Carter (1992), we will perform our extrapolations using a combination of past information on age and period. This stochastic model may be denoted by:

$$\ln(m_{x,t}) = a_x + b_x k_t + (e_{x,t})$$

where, of course, $m_{x,t}$ is the mortality rate at age x at times t , a_x , b_x , and k_t , the model’s parameters, and $e_{x,t}$ the stochastic error, so that the average $E(e_{x,t})$ is equal to zero and the variance $V(e_{x,t})$ is constant. When the model is adjusted by the least-squares method, the interpretation of the parameters is very simple: the adjusted value of a_x is strictly equal to the average of $\ln(m_{x,t})$ for the period, so that b_x represents change in mortality age structure and k_t period trends. Regardless of whether the extrapolation is based on overall data observed between 1950 and 2000 or only on those for the most recent period (1981–2000), the outcomes obtained for each group of causes is little different from those obtained using the classic adjustment of the least squares: cause profiles in 2050 in Table 18.3 are more or less the same as those in Table 18.2.

As before, when looking back, two facts are quite remarkable. On the one hand the result obtained by directly extrapolating mortality for all 18.1 years lived between 60 and 85 years in 2000 rises to 22.0 years in 2050, when referring to the period 1981–2000, instead of only 23.9 when referring to the period 1950–2000.

Table 18.3 Percent of each group of causes as part of the standardized mortality rates for all causes at 60–84 years, in 2000 and in 2050, after extrapolating with the Lee-Carter method 1950–2000 rates and 1981–2000 rates (England and Wales, males)

Group of causes of death	2000	2050	
		Base 1950–2000	Base 1981–2000
Cardiovascular diseases	45.7	4.0	16.2
Bronchial and lung cancers	9.9	0.8	3.6
Digestive cancers	7.7	7.9	5.6
Other tumours	16.9	54.4	38.1
Other diseases and violent deaths	19.8	32.9	36.5
TOTAL	100.0	100.0	100.0

However, on the other hand, a cause by cause extrapolation considerably reduces over time these differences, until by summing the rates by cause extrapolated, in 2050 we obtain, respectively, 20.1 and 20.8 years lived, depending on the reference period considered. This occurs, as previously was the case, so that with this model as with the standard adjustment of the least squares, a marked increase may be foreseen in mortality for other tumours. Finally, this model, despite the fact that it is much more sophisticated, contributes little more than that offered by the standard adjustment of the least squares.

18.2.3 “Age-Period-Cohort” Adjustment (APC Model)

Are further refinements necessary when using an “APC” model based on the combined effects of age, period and cohort? APC models have been used chiefly to interpret past mortality trends (Osmond and Gardner 1982; Hobcraft et al. 1982; Osmond 1985; Caselli and Capocaccia 1989; Wilmoth et al. 1990). Their application in mortality forecasts is more recent (Caselli 1996) or limited to certain specific causes. Burgio and Frova (1995), based on the fact that, generally speaking, the mortality risk, m , may be expressed as a function $m = f(Z\theta)$ of factors $Z = (z_1, \dots, z_n)$ and the parameters $\theta = (\theta_1, \dots, \theta_k)$, hypothesised that the logarithms of the mortality rates could be adjusted using a polynomial function of age, period and cohort:

$$\ln (y_{t,x}^*) = a + a(x) + p(t) + c(t - x)$$

with:

$$\ln (y_{t,x}^*) = a + \sum_i b_i x^i + \sum_j c_j t^j + \sum_k d_k (t - x)^k,$$

for

Table 18.4 Percent of each group of causes as part of the standardized mortality rates for all causes at 60–84 years, in 2000 and 2050, after extrapolating with the “APC” model rates for 1950–2000 and for 1981–2000 (England & Wales, males)

Group of causes of death	2000	2050	
		Base 1950–2000	Base 1981–2000
Cardiovascular diseases	45.7	3.4	9.9
Bronchial and lung cancers	9.9	0.0	1.5
Digestive cancers	7.7	7.3	16.6
Other tumours	16.9	55.6	46.6
Other diseases and violent deaths	19.8	33.6	25.4
TOTAL	100.0	100.0	100.0

$$i = 1, \dots, h_1, j = 1, \dots, h_2 \text{ and } k = 1, \dots, h_3$$

In this function, $y_{t,x}^*$ denotes the theoretical value of mortality rates at age x during the year t (total or by cause) and $a, b_1, \dots, b_{h_1}, c_1, \dots, c_{h_2}, d_1, \dots, d_{h_3}$ are the parameters estimated by the least-squares method.

While this adequately describes past trends, it is not directly applicable to forecasts, to the extent that it does not pretend to prognosticate short-term fluctuations, translated by variations of the “period” parameter. For this reason the authors subdivided this parameter into two additive components, a basic movement, described by the straight line uniting the points relative to the first and last observations, and deviations in this trend. To perform the extrapolation they simply prolonged the basic movement, presuming deviations equal to zero in the basic trend.

The cause profile for 2050, for the reference period 1950–2000 (Table 18.4), is very similar to that obtained for the previous two attempts (Tables 18.2 and 18.3). What can be noted is a slightly larger impact of “other tumours” (55.6%) compared with a lesser impact of “cardiovascular diseases” (3.4%). On the other hand, results differ when, in the projection by cause, the more recent reference period 1981–2000 is taken. An important role is played by “other tumours” (47% as opposed to 38%), compared with a lesser impact of “other diseases” (25% as opposed 37%), while that of tumours of the digestive tract increases (17% compared with 6%). Nonetheless, regarding the number of years lived between 60 and 85 years (Table 18.5), the outcome of the APC approach for the years 1981–2000 is particularly interesting. Only with the APC model is the number of years lived according to the sum of the extrapolations by causes (23.2 years) close to that obtained with the direct extrapolation of mortality for all causes (24.0 years). It can be clearly seen that the APC model, which takes into account cohort effects, is better able to embrace the complexities of more recent trends.

Figure 18.10 compares trend estimates of the sum of standardized mortality rates by cause for each of the four models used here, applied alternatively to the two periods 1950–2000 and 1981–2000.

Table 18.5 Trends from today to 2050 in the number of years lived between 60 and 84 years, according to the model and the reference period used (England & Wales, males)

Reference period and <i>model</i> used	Observed values	Extrapolated values		
	2000	2015	2030	2050
Reference period: 1950–2000				
<i>Linear model:</i>				
Total all causes	18.1	18.2	19.0	20.0
Sum of specific rates by cause	18.1	17.7	18.0	18.1
<i>Least-squares model</i>				
Total all causes	18.1	20.3	22.3	24.0
Sum of specific rates by cause	18.1	19.8	20.5	20.6
<i>Lee-Carter model</i>				
Total all causes	18.1	20.2	22.2	23.9
Sum of specific rates by cause	18.1	19.6	20.5	20.8
<i>APC model</i>				
Total all causes	18.1	20.3	22.3	24.1
Sum of specific rates by cause	18.1	19.8	20.7	20.8
Reference period: 1981–2000				
<i>Linear model:</i>				
Total all causes	18.1	19.5	20.7	22.0
Sum of specific rates by cause	18.1	19.0	19.7	20.2
<i>Least-squares model</i>				
Total all causes	18.1	19.5	20.8	22.1
Sum of specific rates by cause	18.1	19.1	19.8	20.2
<i>Lee-Carter model</i>				
Total all causes	18.1	19.5	20.7	22.0
Sum of specific rates by cause	18.1	19.0	19.7	20.1
<i>APC model</i>				
Total all causes	18.1	20.3	22.3	24.0
Sum of specific rates by cause	18.1	20.1	21.8	23.2

Compared with the results of the “linear” model applied to the entire period 1950–2000, this is largely unaware of a further acceleration in the 1980’s mortality decline among the elderly, particularly regarding cardiovascular diseases. This predicts almost constant mortality levels, if not a slight increase toward 2040, while all the other cases on the figure (comprising the “linear” model applied to the period 1981–2000) all appear to have grasped the drop in mortality for this cause although the intensity tends to vary. In other words, at this level of appreciation, choosing the right reference period is very important.

Nonetheless, if further refinement is sought, two aspects may be noted. Even when applied to the entire period 1950–2000, the results of the “APC”, “Lee-Carter” and “least-squares” models are not different from each other and the same as for the “linear” model when restricted to the most recent period and, thus, offer greater resistance should a poor choice be made regarding the reference period.

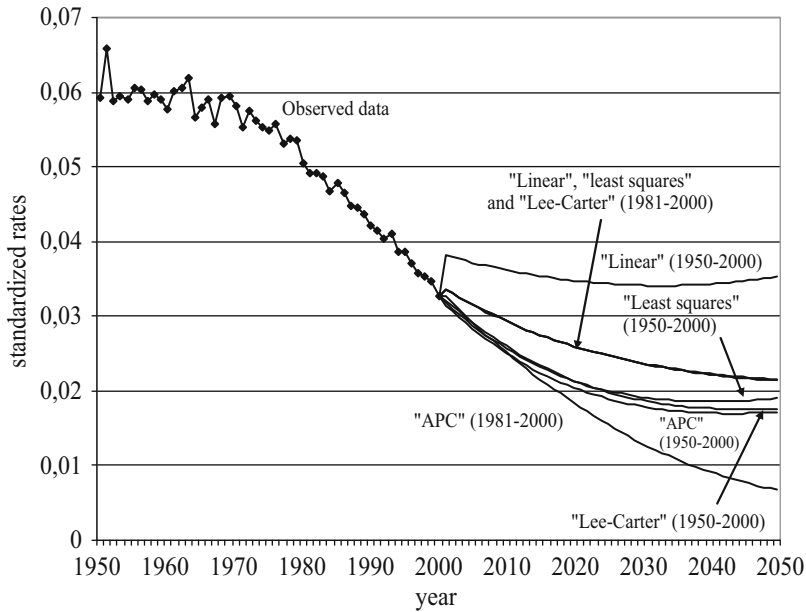


Fig. 18.10 A comparison, in terms of standardized mortality rates at 60–84 years, of the four approaches used (“linear”, “least-squares”, “Lee-Carter”, and “APC” models), of the sum of the rates by cause, according to the reference period used for the extrapolation (1950–2000 and 1981–2000) (England & Wales, males)

Finally, in each instance, whether for one reason or another, when attempting an extrapolation over the long term, undoubtedly it is advisable to use the most sophisticated model, the APC model, the only one to take into account the cohort effect and thus has the advantage of being able to detect the variety of changes which occur during the entire period. The divergence between the results obtained arises when accounting for recent or current reversal of certain tendencies. The actual performance of the different projections may be appreciated even more clearly if focus is given to how a specific cause has developed for which a fresh reversal has been recorded. This can be seen in Fig. 18.11, illustrating patterns for bronchial and lung cancers. Leaving aside the obvious absurdity of the application of the “linear” model to the entire period 1950–2000, it can be seen at which point this model is distinguished from the other three. When the reversed trend has been evident for 10 years or more, the results of all the projections are fairly similar. Of course what can be seen are the same nuances noted above for the sum of the rates by cause, but these are more attenuated. The trend, less pronounced in causes such as “other tumours”, is more decisive at this level.

However, coming back to our question: is it worth considering the cause of death? This exercise, which is purely a forecast, does not suffice to provide an answer. Nonetheless, two comments are worth making. If a long reference period is opted for (1950–2000), one blatant result is that, by taking into account the causes of death, the

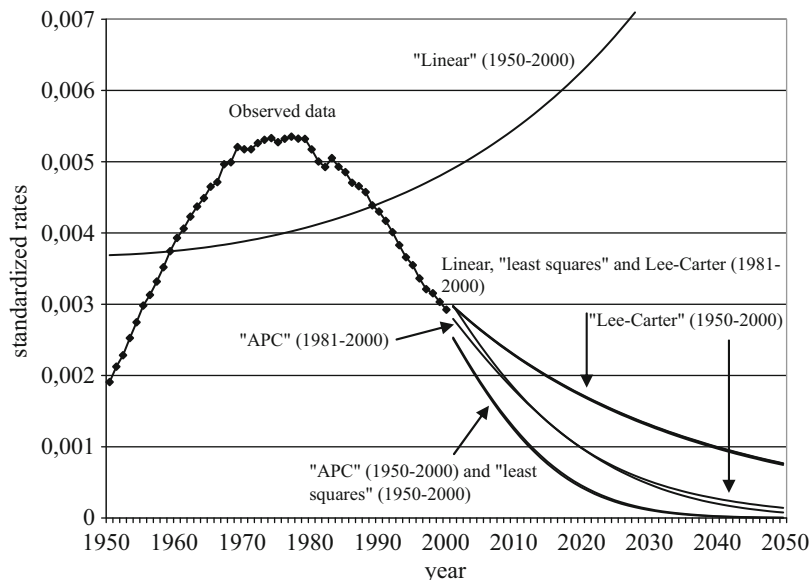


Fig. 18.11 A comparison of comparative mortality rates at 60–84 years, of the four approaches used (“linear”, “least-squares”, “Lee-Carter” and “APC” models), for mortality from bronchial and lung cancers, according to the reference period used for the extrapolation (1950–2000 and 1981–2000) (England & Wales, males)

results of the “linear” model are more pessimistic than others, with a “stagnation” in the number of years lived between 60 and 85 years around 18.1 (in 2050), compared with 20.0 years obtained by directly extrapolating rates for all causes (Table 18.5). With the other three approaches used only slight differences arise when the cause of death is considered, with the number of years lived between 60 and 85 years just topping 20.6–20.8 in 2050. It should be noted that for each of the three models, the sum of the extrapolated rates by cause is even less favourable than that obtained by directly extrapolating mortality for all causes (24 years instead of 20.6 and 20.8).

If the reference period is confined to the end of the observation period (1981–2000) the situation is reversed for the “linear” model which generates a sizeable increase in the number of years lived between 60 and 85 years (20.2 in 2050), but, again, this result is visibly lower than the result obtained by extrapolating mortality for all causes (22.0 in 2050). On the other hand, with the “least-squares” and “Lee-Carter” models the outcome of the projection by cause is not very different from that which is got using the longer period of reference and, for these models, too, the number of years lived is lower than that for all causes. Results for the more recent reference period regarding the application of the APC model are decidedly more interesting. As will be recalled, values for years lived in 2050 differ little among each other according to whether we consider the sums of rates extrapolated by cause or the extrapolation of mortality for all causes (23.2 compared with 24.0).

These results may be easily explained. In the first instance (the long reference period), major importance is given to the role played by reversed mortality from bronchial and lung cancers. This is quite well accounted for relatively speaking by the more sophisticated extrapolation by cause models, but not by the “linear” model, which by spreading the effects of the changing situation over the entire period, ignores the substantial decline in mortality for this cause. More importantly, it completely overlooks this decline among the oldest old that has only occurred quite recently (see Fig. 18.3). In the second instance (more recent, shorter period), where reversed mortality from bronchial and lung cancers is “recognized” by all of the models, differences mainly arise with regard to how they perceive the role played by “other tumours”, which neither the “least-squares” nor the “Lee-Carter” models were able to apprehend fully, while only the APC model managed to grasp these changes.

18.3 The Models Put to the Proof

While providing food for thought, a comparison of the different projections does not help us objectively in assessing how meaningful it is to take into account the causes of death nor the validity of the models used to do so. What it does show us are the differences among the results obtained and to suppose that this or that result is more or less plausible. To determine whether a quality leap has occurred one can estimate the model on an earlier period and compare the model’s projections with how reality has unfolded thereafter. This is our approach.

It turns out that for any extrapolation the period opted for is of paramount importance. We saw that if the period selected is too long, or too short, the risk is that the different trends underway will not be detected. Thus it was decided to apply the models to the period 1950–1980 and compare extrapolations for the period 1981–2000 to reality.

In this case it is clear that, regardless of the model used, apart from the APC model the extrapolation of mortality for all causes largely underestimated the drop in mortality (Fig. 18.12a). It is equally astonishing to see to what extent the results of the first three models are confounded: Absolutely nothing in from the trends in mortality rates by age for all causes in the 1960’s and 1980’s was captured by the refinements in these models. All yield a little less than 16 years to live between the ages of 60 and 85 years in 2000, instead of the 18.1 years observed (Table 18.6).

Considering the first three models, the picture is not better when causes of death are considered (Fig. 18.12b): despite differences in outcome among the models, none of them corresponded at all to the reality. Each of them underestimated the fall in mortality. This underestimation, as expected, is totally exaggerated in the “linear” model (just about 15 years to live between 60 and 85 years). Regarding “least squares” and “Lee-Carter” models, it is better to avoid working on a cause-by-cause approach and, thus, the projection was notably improved, although none of them succeeded in arriving at a realistic result (Table 18.6). Moreover, the APC

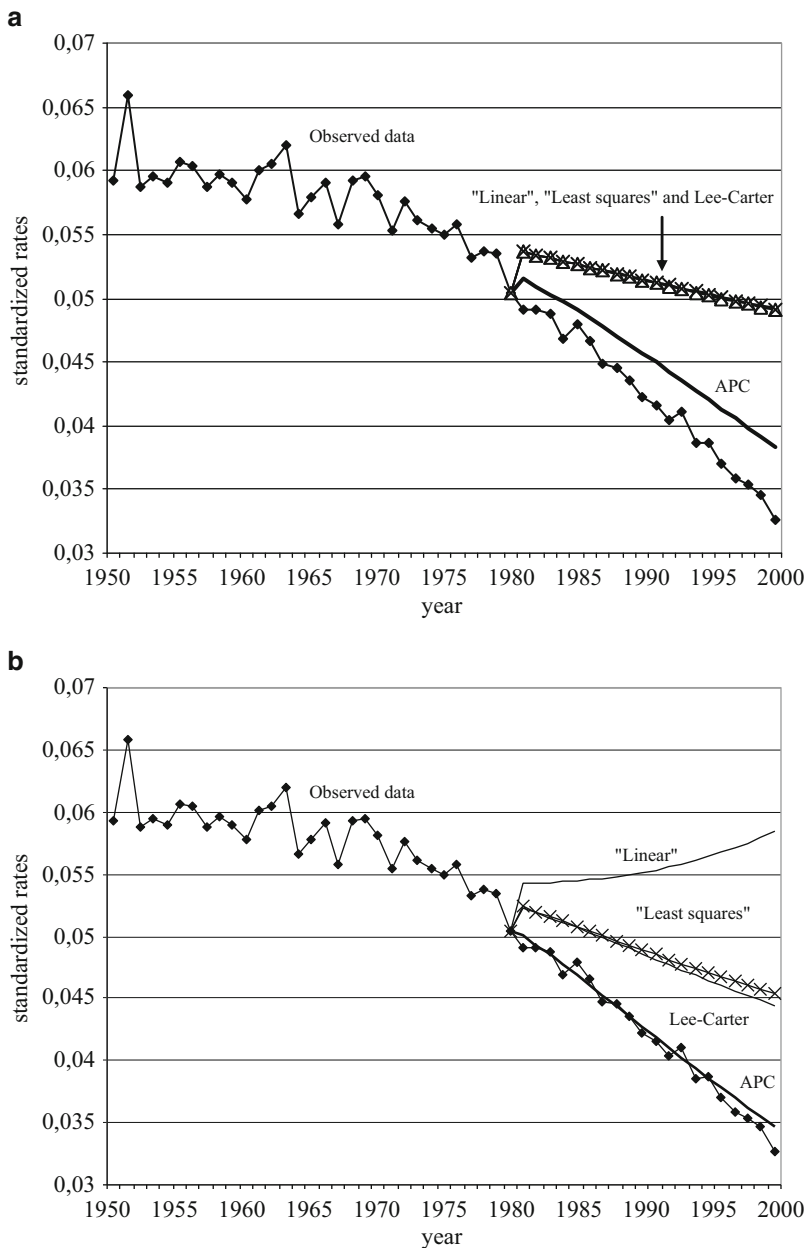


Fig. 18.12 Extrapolations for 1981–2000 of trends for 1950–1980 according to the four models, compared with real trends (England & Wales, males) (a) Direct extrapolation of mortality for all causes, (b) Sum of the extrapolations for mortality for all causes

Table 18.6 Number of years lived between 60 and 85 years in 2000: comparison between observed values and those obtained by extrapolating the data for 1950–1980, according to the four models (England & Wales, males)

Model	Observed values	Extrapolated values	
	2000	Total all causes	Sum of specific rates by cause
“Linear” model	18.1	15.7	14.9
“Least-squares” model	18.1	15.7	16.3
“Lee-Carter” model	18.1	15.8	16.2
“APC” model	18.1	17.2	17.6

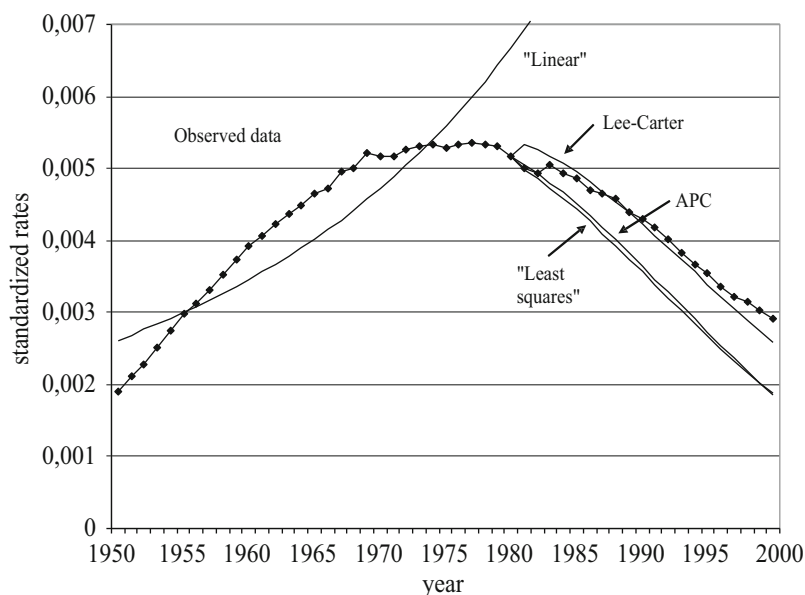


Fig. 18.13 Extrapolations for 1981–2000 of trends for 1950–1980 in bronchial and lung cancer mortality, according to the four models, compared with real trends (England & Wales, males)

model is the only one that approached reproducing reality. In particular when considering cause by death in the years 1981–2000 values often coincided with those observed (Fig. 18.12b), while for the year 2000 survival between 60 and 85 years differed by half a year.

However, when considering the results obtained by cause, it is clear that the APC model is not always better in capturing the renewed decline in mortality from bronchial and lung cancers (Fig. 18.13). The linear model naturally gave the most far-fetched results, extrapolating a preposterously high mortality rate, while on the other hand, the “Lee-Carter” projections best reflected the changing trends.

Even given this success the “Lee-Carter” model may not be conferred universal acclaim as of yet. Indeed, although the decline in bronchial and lung cancer mortality was the main reason for the rapid improvement in mortality trends in the 1970’s and

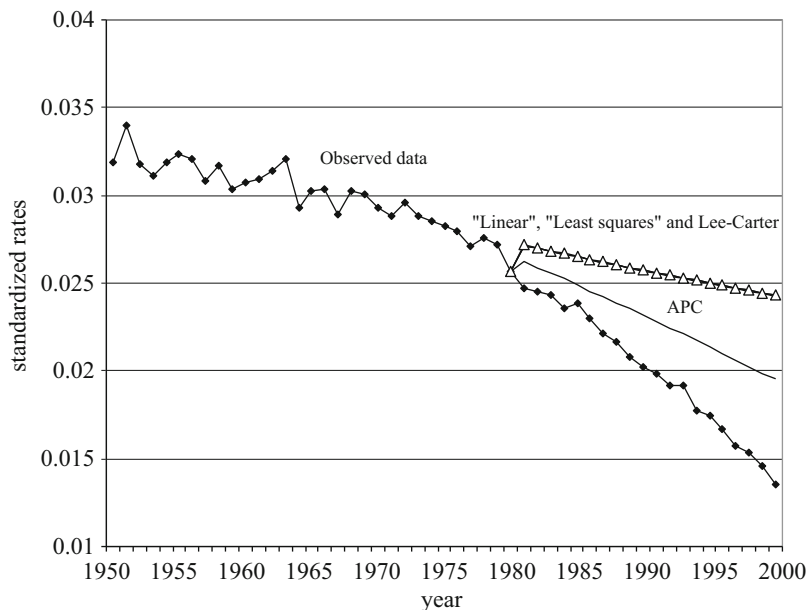


Fig. 18.14 Extrapolations for 1981–2000 of trends for 1950–1980 in mortality from cardiovascular diseases according to the four models, compared with real trends (England & Wales, males)

1980's, it was not the only reason. In fact, no single model, not even the APC model, is capable of fully apprehending this accelerated decline, because the “buds” of this even were not contained in any of the parameters of the models (Fig. 18.14). Otherwise what one finds for diseases of the cardio-vascular system is a perfect overlapping of the results of the first three models for the extrapolation of mortality for all causes (Fig. 18.12a).

In other words, there is no advantage in taking into account the causes of death to extrapolate mortality except in the case where future trends go strictly hand in hand with cohort phenomena, for example in the case of behaviour patterns with regard to smoking. In this case, the APC model performs best. No extrapolation model can foresee trends, the premises of which are not detectable in a reading of past trends.

18.4 Conclusion

Finally, if the aim is to foresee as realistically as possible mortality for all causes, by extrapolating past tendencies, we must make do with only extrapolating mortality rates for all causes. This is not to say that the idea of extrapolating mortality by cause is to be completely rejected. This can be useful from two points of view: to provide a fairly realistic overview of the consequences of cohort effects (in which case the APC model is out in front), as well as to alert policy makers on the effects to be

expected should past trends be prolonged over time (in which case the “linear” model suffices).

The extrapolation of past trends is not the only means of making forecasts. The future may also be fairly realistically based on observed data or that foreseen for elsewhere. Experiences of other countries may be used, where trends have already occurred similar to those one imagines will come to pass in the countries under focus. England was a precursor with regard to smoking habits and their experience may be used to anticipate reverse trends in bronchial and lung cancers, even if only based on current tobacco consumption. Moreover, the effects of recent policies may also be considered. A vaccination programme in a developing country may not be overlooked when estimating future mortality trends. One can, moreover take into account epidemiological facts which are already well-known, but whose effects on mortality are not yet evident. Perhaps even trends in the AIDS epidemic will help us estimate fairly precisely expected mortality over the next few years using only tendencies among the seropositive population. In each of these instances, working with a cause-by-cause model is to be favoured.

To make models, extrapolate trends, is all very well. However, the most complex method is not necessarily the best. The truth may be summed up by two sayings: The only good tools are those which are fashioned to suit the purpose and it is better to dream with your eyes open than make models with your eyes closed.

Acknowledgement The authors thank Dr. Maura Simone to her active contribution to the data processing required by this study.

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