



# Perspectives on Mortality Forecasting

IV. Causes of Death

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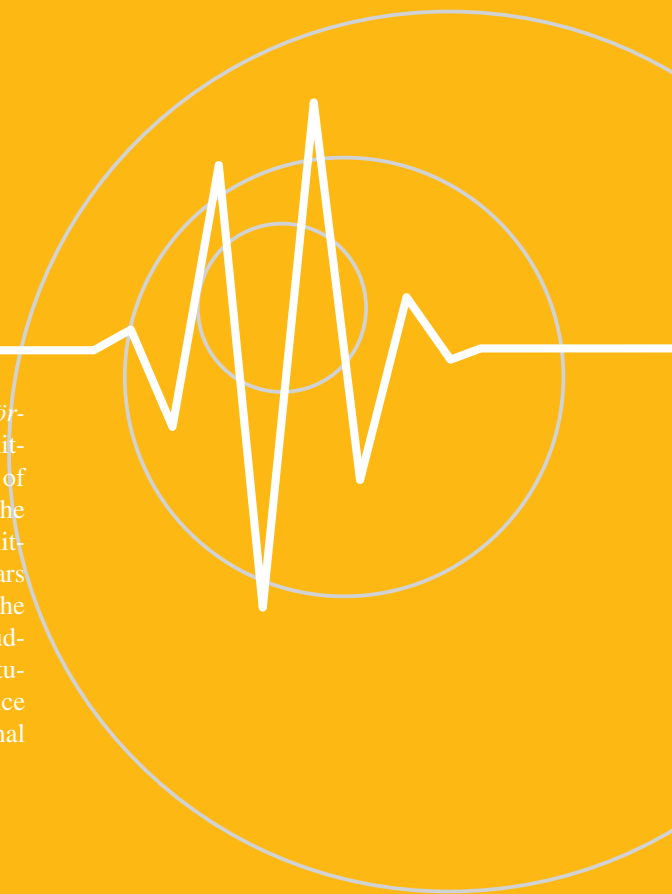
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Kaare Christensen

Swedish Social Insurance Agency

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Swedish Social Insurance Agency  
Adolf Fredriks Kyrkogata 8  
SE-103 51 STOCKHOLM, Sweden  
Telephone: +46 8 786 90 00  
E-mail: [huvudkontoret@forsakringskassan.se](mailto:huvudkontoret@forsakringskassan.se)

Graphic Production: Kristina Malm  
Cover Design: Jenny Övrebö  
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# Preface

Mortality projections are an essential input for projections of the financial development of pension schemes and health and social policy planning. Governments and insurance companies all over the world rely on good mortality projections from efficient administration of their pension commitments. However, during recent decades, demographers have continually underestimated improvements in life expectancy for persons 60 and older. The demographic models used in projecting mortality are usually statistical models based on historical data. The question is, it is possible to improve mortality modelling, and if so, what do demographers need to do to achieve this result?

This is the question that provided the impetus for forming the Stockholm Committee on Mortality Forecasting. The Swedish Social Insurance Agency (formerly National Social Insurance Board, RFV) is the national agency in Sweden responsible for providing a financial picture of Sweden's public pension system. The Swedish Social Insurance Agency has a long-standing interest in the development of modelling of pension schemes and participates actively in the international dialogue among experts in this area. The Stockholm Committee on Mortality Forecasting was created by RFV to bring together scholars from different disciplines working on issues in projecting mortality. The aim of the Committee is to survey the state of the art and to provide an impetus for the advancement of knowledge and better practice in forecasting mortality.

This is the fourth volume in a series presenting papers from workshops on mortality organized by the Stockholm Committee on Mortality Forecasting. It addresses the question of how information on changes in patterns in the cause of death can be used to improve mortality forecasting. While the increase in life expectancy was largely propelled by the decline in infant and child mortality up until the middle of the twentieth century, it has since then been sustained by the decline in old age mortality. The improvement in life expectancy among the elderly is mainly due to progress in combating chronic diseases. Mortality in cardiovascular and cerebrovascular diseases and some forms of cancer has declined. While improved medical care – earlier detection, improved surgery, and better therapies – is a major factor behind this, it is not the only one. Changes in working conditions, life styles and improvements early in life have contributed to the reduction of mortality in chronic diseases, as have other factors. In this volume, the focus is on the changes in the patterns of cause of death.

The article by Graziella Caselli, Jacques Vallin, and Marco Marsili discusses the usefulness of making extrapolations of past trends in major diseases. They discuss the problems related this method. In spite of clear drawbacks in

using this information for extrapolation, they do not categorically reject it since it can provide a fairly realistic overview of what is behind trends and in doing so alert policy makers of possible effects if these trends continue. Other means of making use of causes of death information for forecasting is discussed as well, including making use of information from other countries.

Måns Rosén starts from an epidemiological perspective, discussing the relationships between incidence, prevalence and mortality. In addition to examining possibilities of extrapolating past trends in cause specific mortality, Rosén brings up the central discussion of whether prolongation of life leads to compression or prolongation of morbidity. While many studies in the past have concluded that compression is dominating, some recent Swedish studies indicate that this may not be the case. Thus it may well be that improvements in medical care and therapy will lead to an increase in the demand for health expenses but not necessarily health care in this group.

The third and final chapter, by Richard Willets, discusses how analyses of mortality by cause of death will influence forecasts in the UK. His conclusion is that, despite well-documented difficulties in making cause of death projections in the past, there still appears to be a good case for continuing to do so. This is particularly the case when predicting mortality among the not very old elderly, say those below 80 years of age. It can also be used to test “extreme” scenarios. Thus while using information on causes of death in making mortality forecasts has proven to be difficult, there still is a substantial potential to be gained.

Tommy Bengtsson

Kaare Christensen

Edward Palmer

## **Members of the Stockholm Committee on Mortality Forecasting**

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Uppsala University and Swedish Social Insurance Agency, Sweden

Professor Tommy Bengtsson, Committee Secretary  
Lund University, Sweden

Professor Juha M. Alho  
University of Joensuu, Finland

Professor Kaare Christensen  
University of Southern Denmark, Odense

Professor Nico Keilman  
University of Oslo, Norway

Professor James W. Vaupel  
Director of Max Planck Institute for Demography, Rostock, Germany



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# How Useful Are the Causes of Death When Extrapolating Mortality Trends. An Update<sup>1</sup>

**Graziella Caselli\*, Jacques Vallin\*\* and Marco Marsili\*\*\***

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\*Professor of Demography, Director of the Department of Demography, University of Rome “La Sapienza”, \*\* Research Director at INED, Paris, and \*\*\*Researcher at ISTAT, Rome

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 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

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<sup>1</sup> This paper is an update of Caselli and Vallin 1999a (in French) and Caselli and Vallin 1999b (in English).

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.

## **Extrapolation of Mortality by Cause Risks Absurdity**

Figure 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 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)**

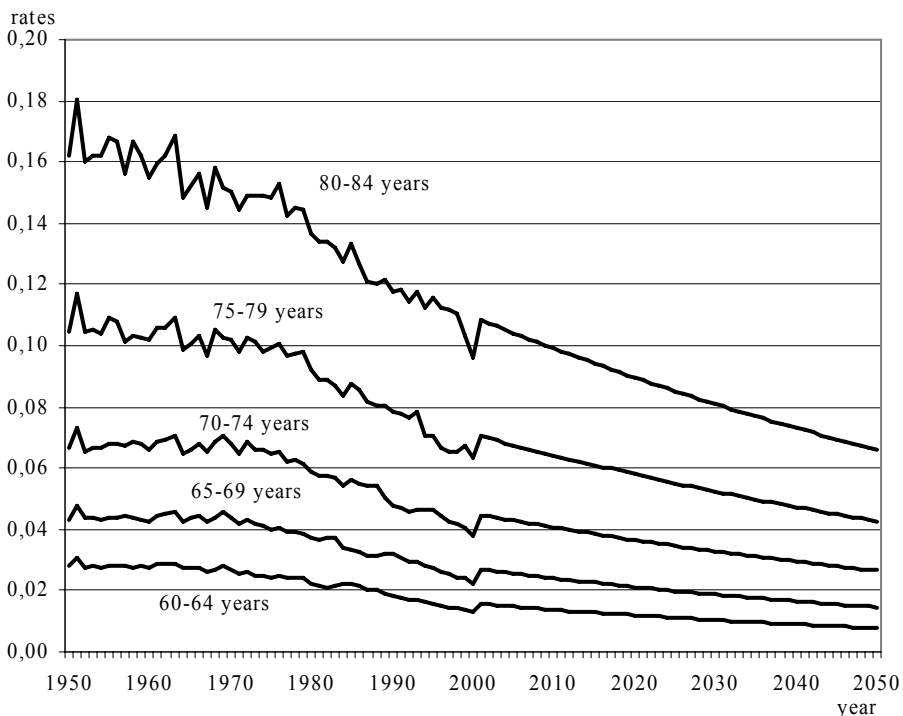
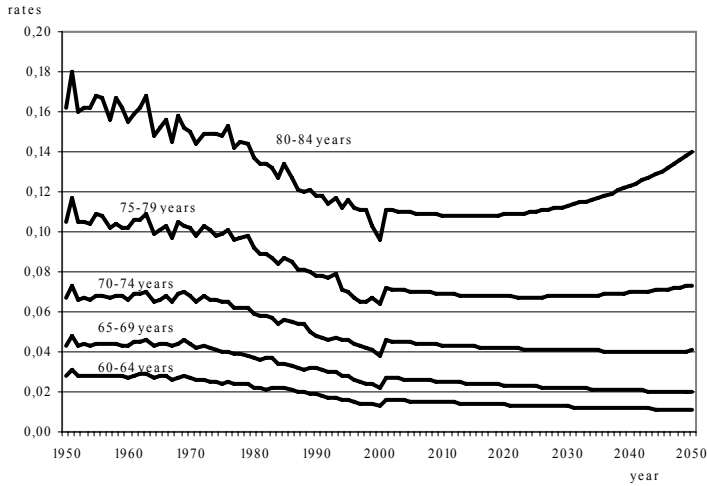


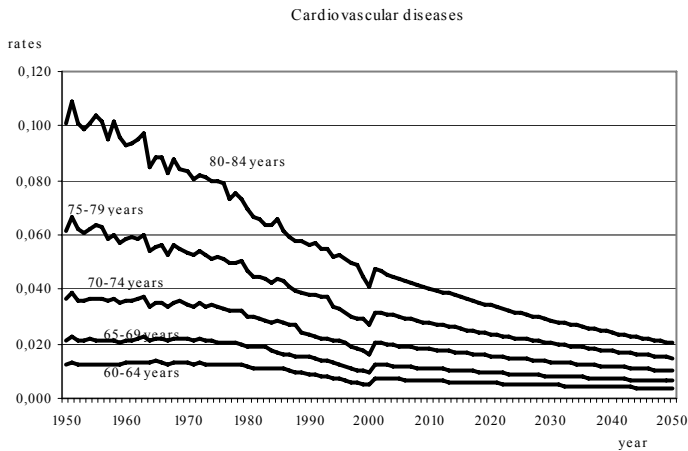
Figure 2, on the other hand, illustrates the results of summing similar type extrapolations performed separately for each group of causes. A systematic increase in total mortality immediately occurs at older ages between 75 and 85 years, while the trend of reduction for ages between 60 and 75 years is 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 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 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 (Figure 3).

**Figure 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)

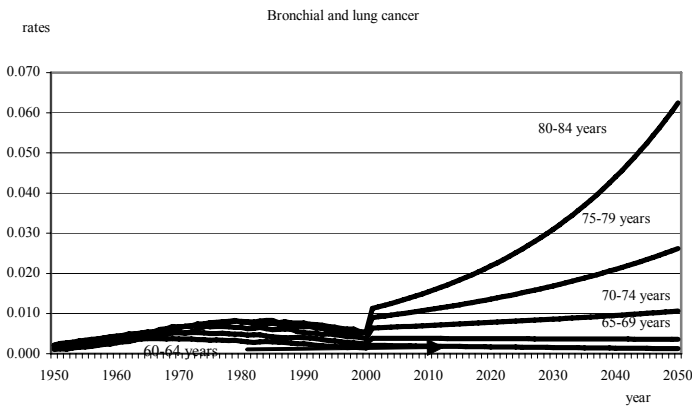


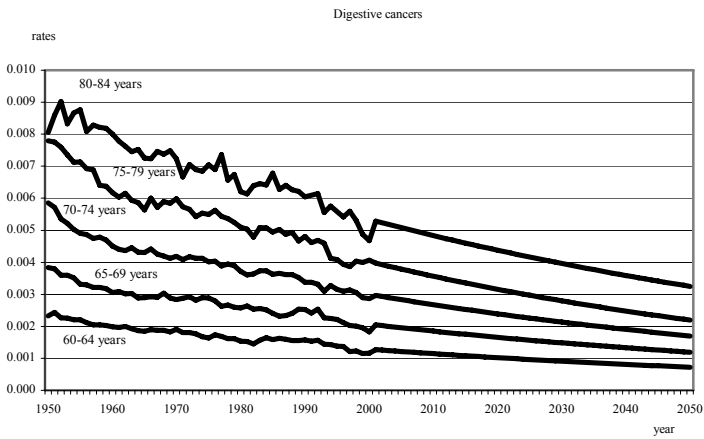
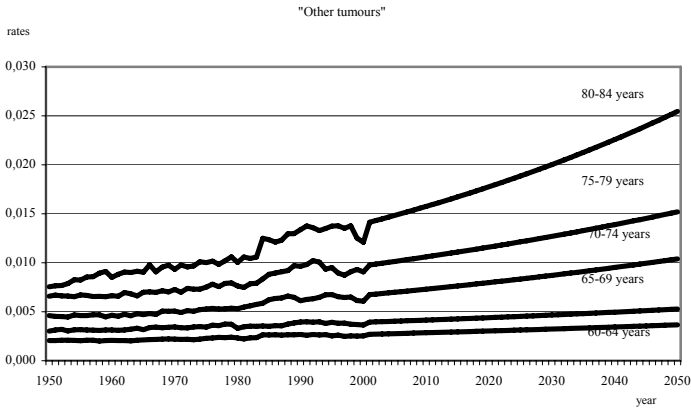
According to this outline, the impact of bronchial and lung cancers on total mortality comparative rates at 60-84 years would rise from 9.9% in 2000 to 30.9% in 2050, while that of cardiovascular diseases would fall from 45.7 to 23.4% (Table 1)!

**Figure 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)**



**Figure 3**      **Continued**





**Table 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)

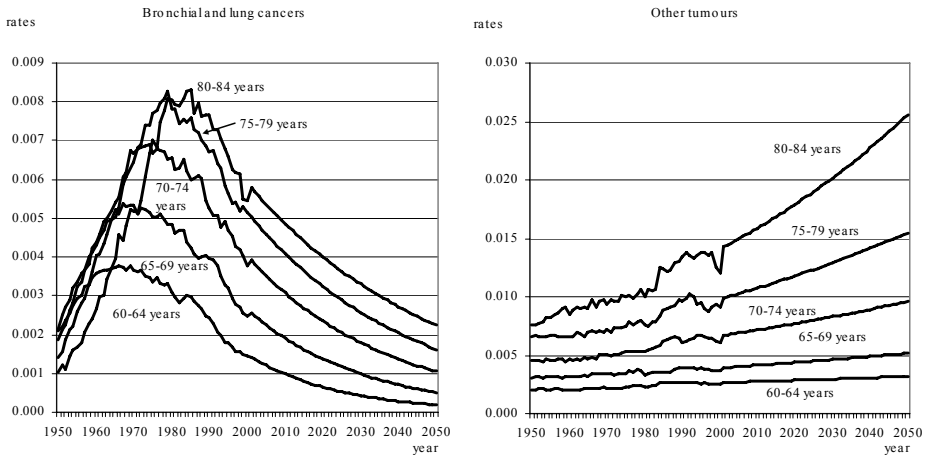
Group of causes of death	2000	2050	
		Base 1950-2000	Base 1981-2000
Cardiovascular diseases	45.7	23.4	16.2
Bronchial and lung cancers	9.9	30.9	3.6
Digestive cancers	7.7	4.0	5.6
Other tumours	16.9	24.0	37.6
Other diseases and violent deaths	19.8	17.7	37.1
<b>TOTAL</b>	<b>100.0</b>	<b>100.0</b>	<b>100.0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

No doubt this example is too extreme. Obviously, for England & Wales no one would dream of extrapolating bronchial and lung cancer mortality trends for 2001-2050 by a linear adjustment of the entire period 1950-2000, when in fact a reversal trend occurred in the early 1970’s.

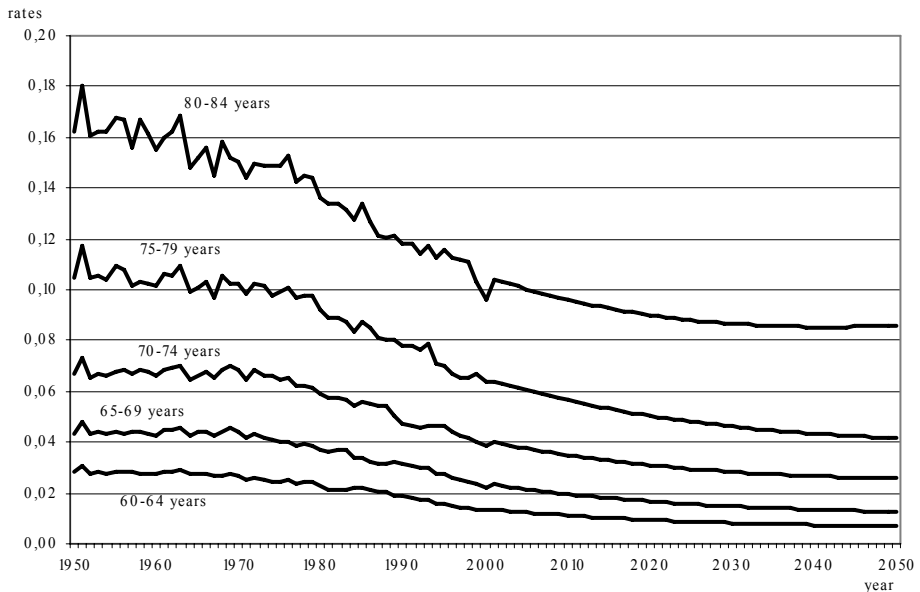
Thus fresh calculations were made, restricting the adjustment of past trends to the period 1981-2000. The results are visibly improved for bronchial and lung cancer, as this time mortality for this cause decidedly follows a downward trend for all age groups (Figure 4). However, the problem is still not 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 (Figure 5). There is no doubt, given this scenario, that the total number of years lived between 60-84 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.



**Figure 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)



**Figure 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)



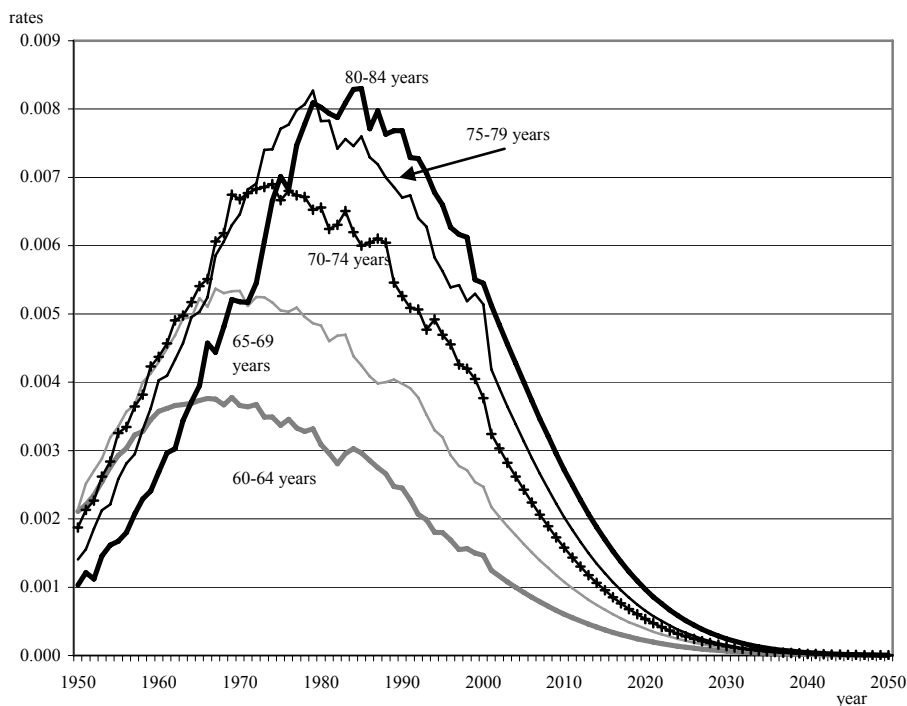
## Would More Sophisticated Methods Be Any Better?

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.

### *a) A better adjustment of chronological series of rates by age*

Here a choice was made between four classic functions (straight line, parabole, 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 (Figure 6).

**Figure 6** Extrapolation of age group mortality rates for bronchial and lung cancers, using a “least-squares” adjustment of 1950-2000 data (England & Wales, males)



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 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.

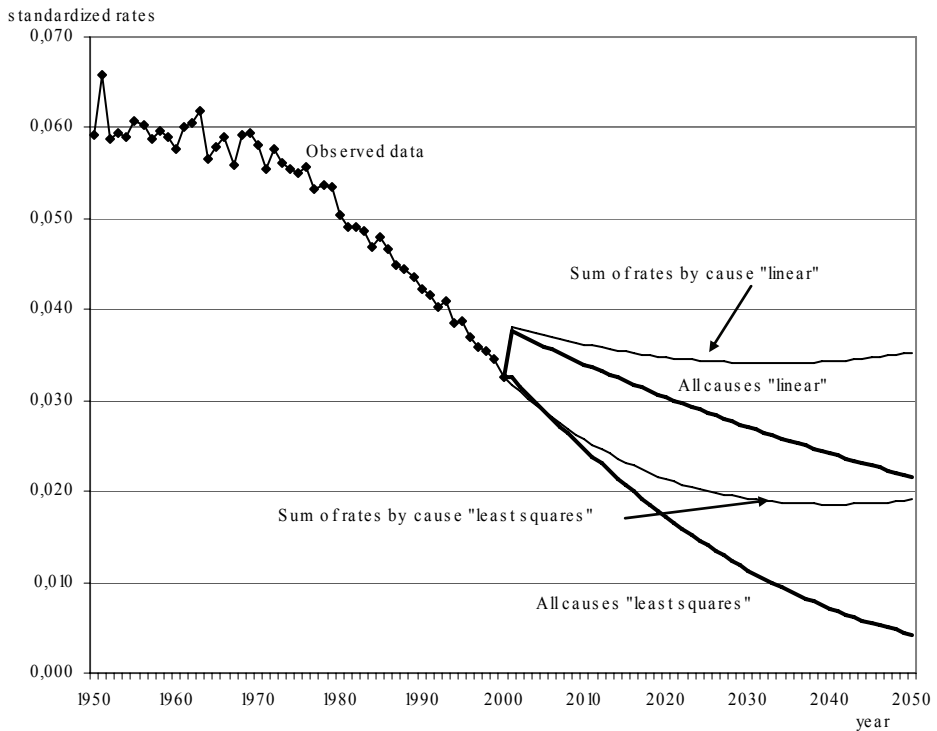
**Table 2** Percent of each group of cause as part of the standardized mortality rates for all causes at 60-84 years, in 2000 and in 2050, after a “least-squares” extrapolation of 1950-2000 rates and then 1981-2000 rates (England & Wales, males)

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
<b>TOTAL</b>	<b>100.0</b>	<b>100.0</b>	<b>100.0</b>

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 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”.

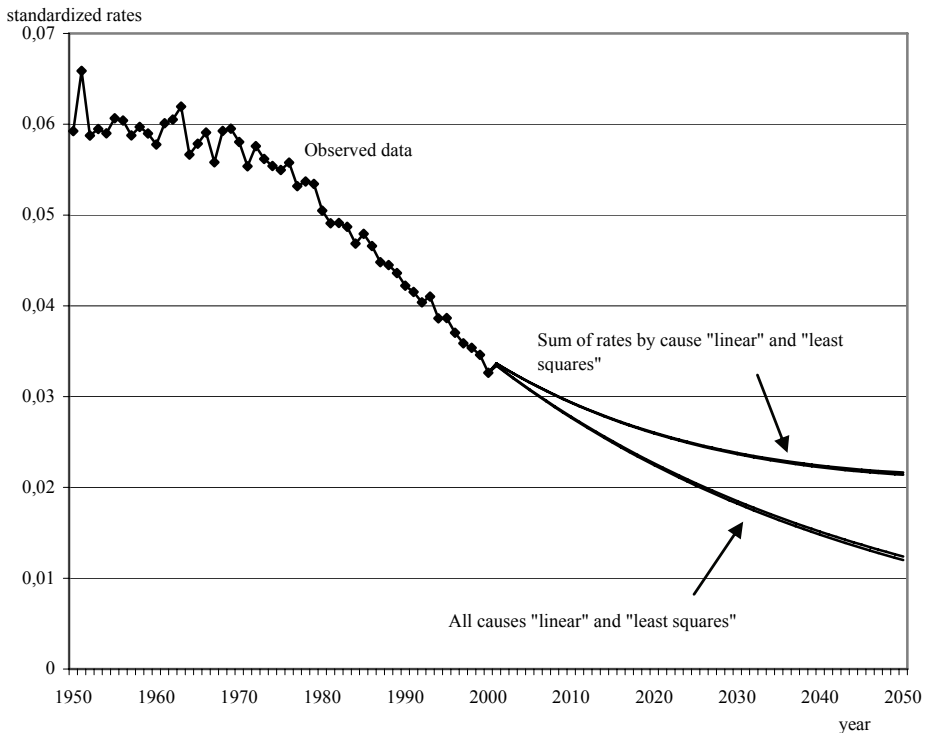
Figures 7 and 8 report the different outcomes obtained to date regarding standardized mortality rates at 60-84 years, referring alternatively to the periods 1950-2000 and 1981-2000.

**Figure 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 to 2000 (England & Wales, males)



Based on data observed for 1950 to 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 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.

**Figure 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 to 2000 (England & Wales, males)



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” (Figure 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”.

*b) “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 3 are more or less the same as those in Table 2.

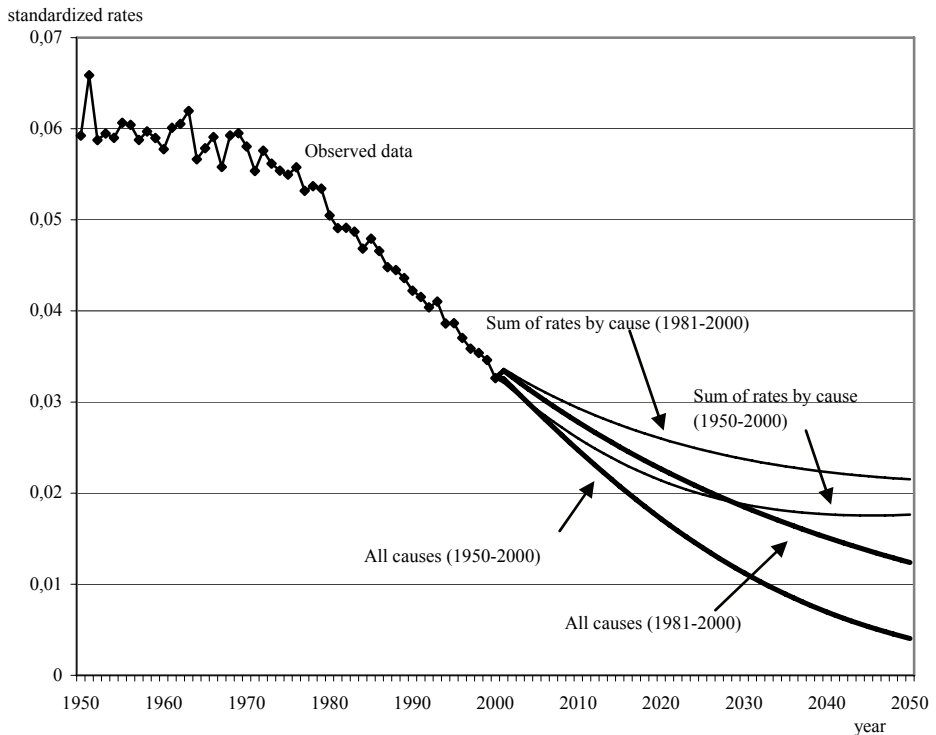
**Table 3** Percent of each group of causes as part of the standardized mortality rates for all causes at 60-84 years, in 2000 and in 2050, after extrapolating with the Lee-Carter method 1950-2000 rates and 1981-2000 rates (England and Wales, males)

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
<b>TOTAL</b>	<b>100.0</b>	<b>100.0</b>	<b>100.0</b>

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. 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.

**Figure 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)**



*c) “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

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

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 4), is very similar to that obtained for the previous two attempts (Tables 2 and 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 trace increases (17% compared with 6%). Nonetheless, regarding the number of



years lived between 60 and 85 years (Table 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.

**Table 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

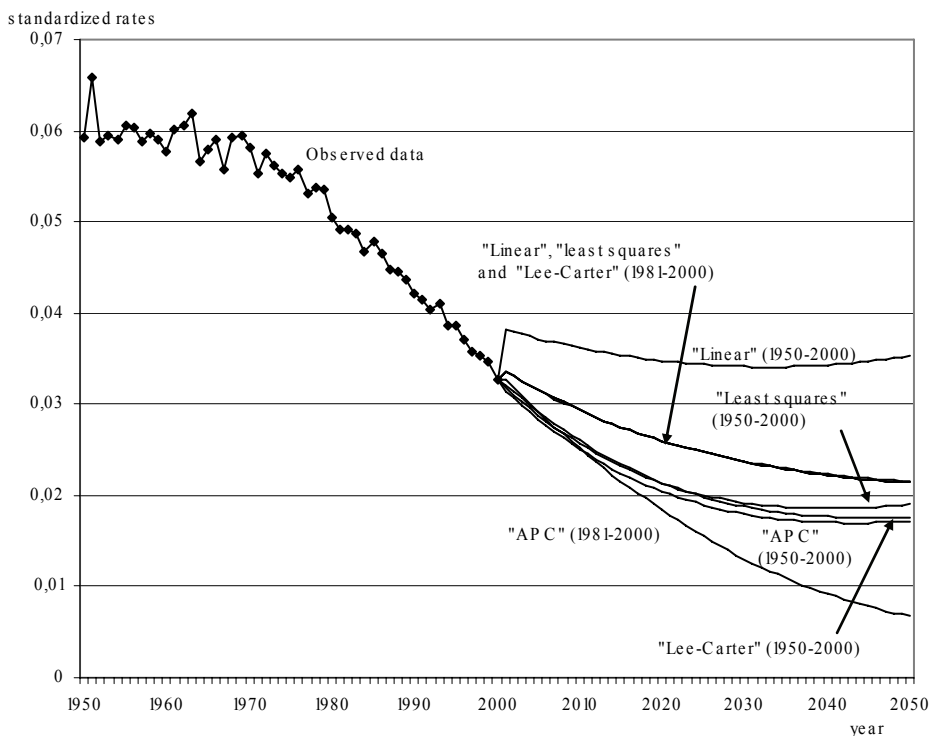
Figure 10 compares trend estimates of the sum of standardized mortality rates by cause for each of the four models used here, applied alternatively to the two periods 1950-2000 and 1981-2000.

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.

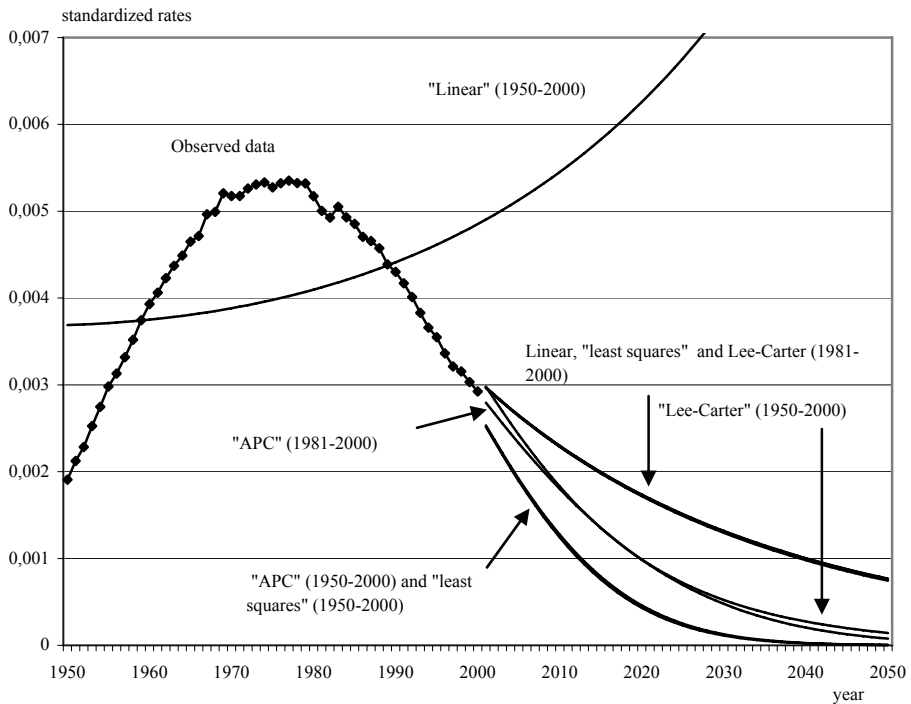
**Figure 10** A comparison, in terms of standardized mortality rates at 60-84 years, of the four approaches used (“linear”, “least-squares”, “Lee-Carter”, and “APC” models), of the sum of the rates by cause, according to the reference period used for the extrapolation (1950-2000 and 1981-2000) (England & 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 Figure 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 ten 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.

**Figure 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)



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 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 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).

**Table 5 Trends from today to 2050 in the number of years lived between 60 - 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
<b>Reference period: 1950-2000</b>				
<b><i>Linear model:</i></b>				
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
<b><i>Least-squares model</i></b>				
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
<b><i>Lee-Carter model</i></b>				
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
<b><i>APC model</i></b>				
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
<b>Reference period: 1981-2000</b>				
<b><i>Linear model:</i></b>				
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
<b><i>Least-squares model</i></b>				
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
<b><i>Lee-Carter model</i></b>				
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
<b><i>APC model</i></b>				
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

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 Figure 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.

## **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 (Figure 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 6).

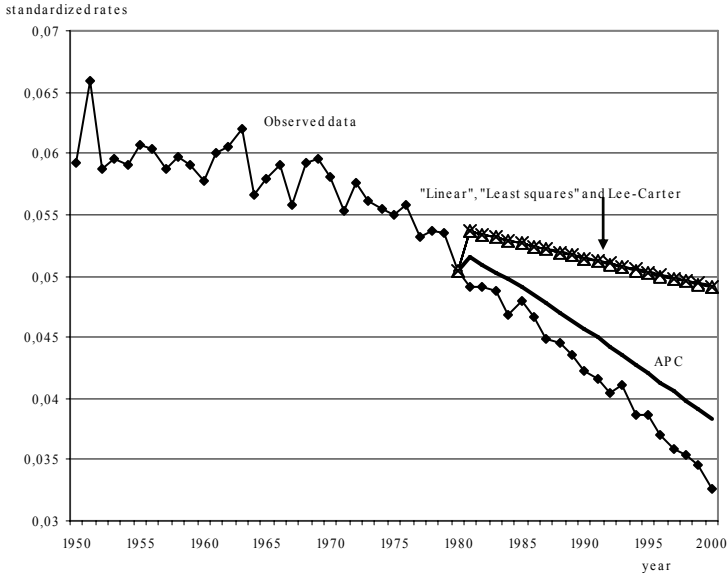
**Table 6** Number of years lived between 60 and 85 years in 2000: comparison between observed values and those obtained by extrapolating the data for 1950-1980, according to the four models (England & Wales, males)

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

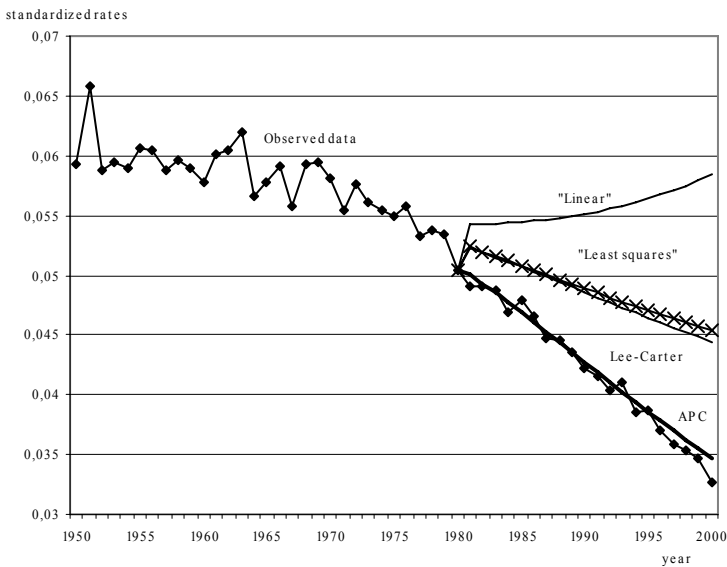
Considering the first three models, the picture is not better when causes of death are considered (Figure 12b): despite differences in outcome among the models, none of them corresponded at all to the reality. Each of them underestimated the fall in mortality. This underestimation, as expected, is totally 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 6). Moreover, the APC 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 (Figure 12b), while for the year 2000 survival between 60 and 85 years differed by half a year.

**Figure 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,**

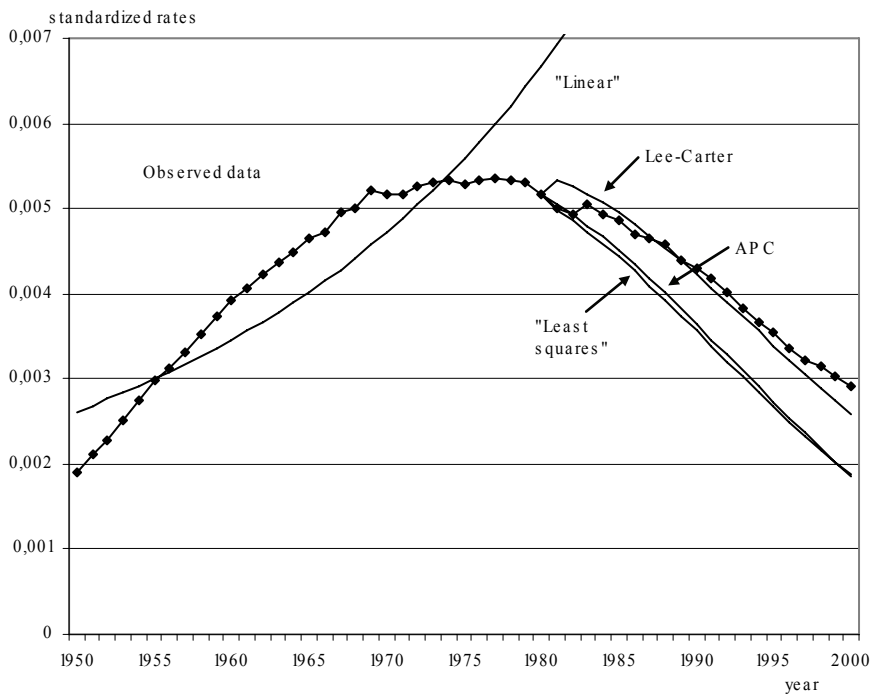


**b) Sum of the extrapolations for mortality for all causes**



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 (Figure 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.

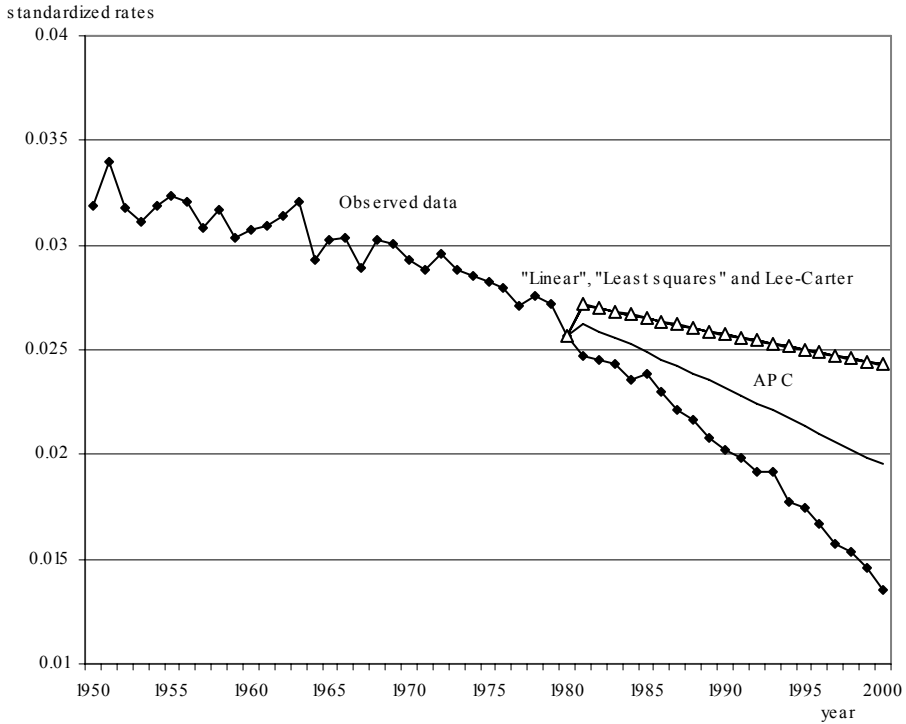
**Figure 13** Extrapolations for 1981-2000 of trends for 1950-1980 in bronchial and lung cancer mortality, according to the four models, compared with real trends (England & Wales, males)



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 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 (Figure 14). Otherwise what one finds for diseases of the cardiovascular system is a perfect overlapping of the results of the first three models for the extrapolation of mortality for all causes (Figure 12a).



**Figure 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)



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.

## 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**

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## References

- Benjamin B. and E. Overton (1985). "Prospects for mortality decline in England and Wales." *Population Trends*, 42, 22-29.
- Burgio A. and L. Frova (1995). "Projections de mortalité par cause de décès: extrapolation tendancielle ou modèle âge-période-cohorte." *Population*, 50(4/5), 1031-1052.
- Caselli G. (1996). "Future longevity among the elderly." In: G. Caselli and A. Lopez (eds.) *Health and mortality among elderly populations*. Oxford: Clarendon Press, 235-265.
- Caselli G. (1993). "L'évolution à long terme de la mortalité en Europe." In: A. Blum and J-L. Rallu (eds.) *European Population, II. Demographic Dynamics/ Démographie européenne, II. Dynamiques démographiques*. Paris: INED/John Libbey, 111-164. (Also published in English: OPCS: Studies on medical and population subjects, n° 56, London, 1994).
- Caselli G. and R. Capocaccia (1989). "Age, period, cohort, and early mortality: an analysis of adult mortality in Italy." *Population Studies*, 43, 133-153.
- Caselli G. and V. Egidi (1992). "New frontiers in survival: the length and quality of life." In: *Human resources in Europe at the dawn of the 21<sup>st</sup> century/ Le capital humain européen à l'aube du 21<sup>e</sup> siècle*. Luxembourg: Eurostat, 95-120 (Proceedings of an International Conference, Luxembourg, 27-29 novembre 1991).
- Caselli G. and J. Vallin (1999a). "Est-il utile de prendre en compte les causes de décès pour extrapoler les tendances de la mortalité?" In: C. Wattelar and J. Duchêne (eds.) *Le défi de l'incertitude. Nouvelles approches en perspectives et prospectives démographiques*. Louvain-la-Neuve: Institut de démographie de l'UCL, Academia-Bruylant et L'Harmattan, Chaire Quételet, 85-110.
- Caselli G. and J. Vallin (1999b). "How useful are the causes of death when extrapolating mortality trends?" In: V. Egidi (ed.) *Towards an integrated system of indicators to assess the health status of the population*. Rome: ISTAT, 121-150.
- Hobcraft J., J. Menken and S. Preston (1982). "Age, period, and cohort effects in demography: a review." *Population Index*, 48(1), 4-43.

- Lee R.D. and L. Carter (1992). “Modelling and forecasting US mortality.” *Journal of the American Statistical Association*, 87(419), 659-675.
- Meslé F. (1993). “Analyse comparée des projections nationales en Europe.” In: J. Vallin (ed.) *L’avenir de l’espérance de vie*. Paris: INED, 5-22. (Congrès et Colloques n° 12).
- Osmond C. (1985). “Using age, period, and cohort models to estimate future mortality rates.” *International Journal of Epidemiology*, 14(1), 124-129.
- Osmond C. and M. Gardner (1982). “Age, period, and cohort models applied to cancer mortality rates.” *Statistics in Medicine*, 1.
- Poppel F. van and J. de Beer (1996) “Evolution of standard mortality projections for the elderly.” In: G. Caselli and A. Lopez (eds.) *Health and mortality among elderly populations*. Oxford: Clarendon Press, 288-312.
- Vallin J. (1992). “Quelles hypothèses peut-on faire sur l’évolution future de la mortalité ?” In: G. Tapinos (ed.) *La France dans deux générations : population et société dans le premiers tiers du XXI<sup>e</sup> siècle*. Paris: Arthème Fayard, 98-114.
- Vallin J. (1989). “L’avenir de l’espérance de vie vu à travers les projections de l’INSEE.” *Population*, 44(4/5), 930-936.
- Vallin J. and F. Meslé (1989). “À long terme, l’écart d’espérance de vie entre hommes et femmes devrait diminuer.” *Population*, 44(6), 1244-1251.
- Wilmoth J. (1996). “Mortality projection for Japan: a comparison of four methods.” In: G. Caselli and A. Lopez (eds.) *Health and mortality among elderly populations*. Oxford: Clarendon Press, 266-287.
- Wilmoth J., J. Vallin and G. Caselli (1990). “When does a cohort’s mortality differ from what we might expect ?” *Population, English selection*, 2, 93-126. (Also published in French: “Quand certaines générations ont une mortalité différente de celle que l’on pourrait attendre.” *Population*, 44(2), 335-376).

# Forecasting Life Expectancy and Mortality in Sweden – Some Comments on Methodological Problems and Potential Approaches

**Måns Rosén**

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Centre for Epidemiology, National Board of Health and Welfare, Sweden

## Introduction

Since mortality is affected by innumerable factors in society, an interdisciplinary approach seems most appropriate. My contribution and point of departure starts from an epidemiological perspective and from the overall objective of the Swedish Centre for Epidemiology, i.e. to monitor public health in Sweden<sup>1</sup>. An advantage of epidemiology is the close link to public health and medicine as well as its focus on analyses of risk factors and the search for causal chains between risk factors, diseases and mortality. Mortality forecasting is a well-established discipline in demography, but maybe less developed within epidemiology. Still, there have been attempts to forecast mortality within the field of epidemiology (see e.g. Wilhelmsen, Lappas and Rosengren 2004; Gunning-Schepers 1989; Gunning-Schepers, Barendregt, and Van Der Maas 1989; Kruijshaar, Barendregt and Hoeymans 2002; Conroy, Pyörälä, Fitzgerald, Sans, Menotti, De Bacquer et. al. 2003). Usually, epidemiologists have focused on estimating mortality for specific causes of death (Wilhelmsen et al. 2004; Conroy et al. 2003) but there are also attempts to predict total mortality (Gunning-Schepers 1989; Kruijshaar et al. 2002). A common application has been to predict coronary heart mortality based on data on risk factors, e.g. smoking, level of cholesterol and blood pressure in the population (Wilhelmsen et al. 2004; Conroy et al. 2003). Knowledge of risk factor patterns is therefore an essential element in epidemiology. The risk factor approach will be discussed later. First, some comments on the outline of this paper.

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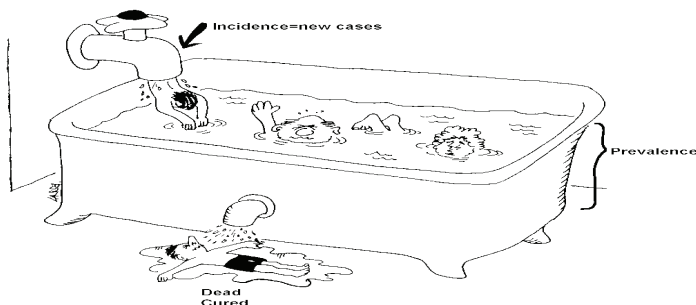
<sup>1</sup> National Board of Health and Welfare (2003).

I will start by introducing the two most widely used measures in epidemiology, i.e. incidence and prevalence, and the relationships between those two measures and mortality. Second, I will give some general comments on pros and cons with different options for mortality forecasting. The different options discussed are extrapolating mortality trends, predicting disease-specific causes of death, predicting mortality trends based on potential elimination of causes of death or predicting mortality based on risk factors or other developments in the community. Third, some methodological problems will be discussed. Finally, I will advocate a risk factor based approach and speculate about future mortality and longevity based on our attempts to monitor public health in Sweden.

## The Relationships between Incidence, Prevalence and Mortality

Incidence is defined as the number of new cases of a disease during a specified time period while prevalence is the total number of people with a disease at a specific point in time. It is well illustrated by a bath tube (Figure 1) where the water coming through the tap is the incidence and the water in the bath tube is the prevalence. The prevalence is affected by the incidence, but also by the number of people cured or deceased.

**Figure 1** The relationships between incidence, prevalence, mortality and cured



Those who die will no longer belong to the population at risk while those who are cured still belong to the population at risk. The cured survivors will have a probability of contracting a new disease. Primary prevention may influence incidence while prevalence is more of a measure of the total disease burden for society.

Mortality is affected by the incidence, which could be divided into different components from demographic characteristics (number of people, population growth and changing age distribution) to the risk of having/attracting a disease (based on risk factor patterns). Mortality is also influenced by the chance of surviving a disease. All these components are important to consider in mortality forecasting.

## **Extrapolating Mortality Trends or Predicting Disease-specific Causes of Death**

Life expectancy has increased impressively during the past 150 years. In Sweden, life expectancy for a man has improved from 35 years in the beginning of the 1800s to 61 years in the 1920s and up to 77.91 years in 2003. For women, life expectancy is 82.43 years in 2003. This success story seems to be never ending. In the 1980s, many believed there was little potential for improvement, but they were wrong. Still, it seems unlikely and atheoretical to believe this can persist forever.

History can also show us the danger of only extrapolating existing trends. A recent and dramatic story is the development of mortality and life expectancy in Russia<sup>2</sup>. Between 1970 and 1985 life expectancy in Russia was quite stable around 68 years (WHO). Between 1985 and 1987 it rose to 70 years followed by a substantial drop to about 64 years. Several studies have analysed the reasons to this dramatic and rapid change. The main explanations suggested are economic and social instability as well as changes in alcohol consumption (Shkolnikov et al. 2001; Notzon et al. 1998; Nemtsov 2002). The anti-alcohol campaign, launched in 1985, and the market reforms launched in 1992 were associated with large and rapid changes of alcohol consumption in Russia (Nemtsov 2002).

Trends in life expectancy among women in Denmark and the Netherlands can serve as other examples of the danger of only extrapolating trends. Since 1970, there is a steady increase in life expectancy for men both in Denmark and the Netherlands (WHO). However, extrapolating trends from the early 1970s would highly overestimate the longevity of women in Denmark and the Netherlands. Danish women increased their life expectancy substantially from about 76 years in 1970 to 78 years in 1977 followed by no increase at

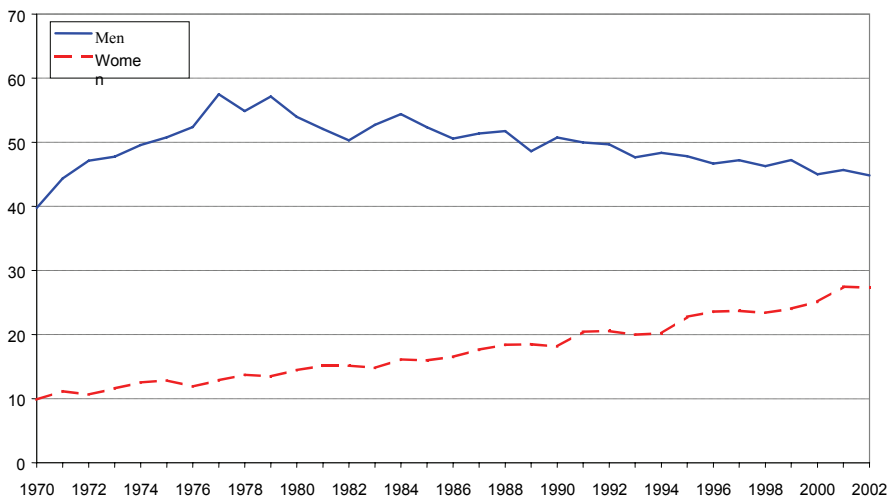
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<sup>2</sup> WHO Europe Health for All database, see <http://www.who.dk>; Shkolnikov, McKee and Leon (2001); Notzon, Komarov, Ermakov, Sempos, Marks and Sempos (1998); Nemtsov (2002).

all up to 1995. After 1995 life expectancy among women in Denmark has started to increase again. For women in the Netherlands, life expectancy increased substantially up to 1990, but has thereafter not followed the increasing trends of many other western European countries. These changes in trends indicate clearly that the risk factor patterns of women in these two countries have been different than in other European countries.

The danger of extrapolating mortality trends is also evident when studying some disease-specific causes of death in Sweden. Lung cancer mortality among men increased substantially from the 1950s up to around the end of the 1970s followed by a decrease in both incidence and mortality (Figure 2).

**Figure 2 Trends in lung cancer mortality in Sweden, 1970-2002**



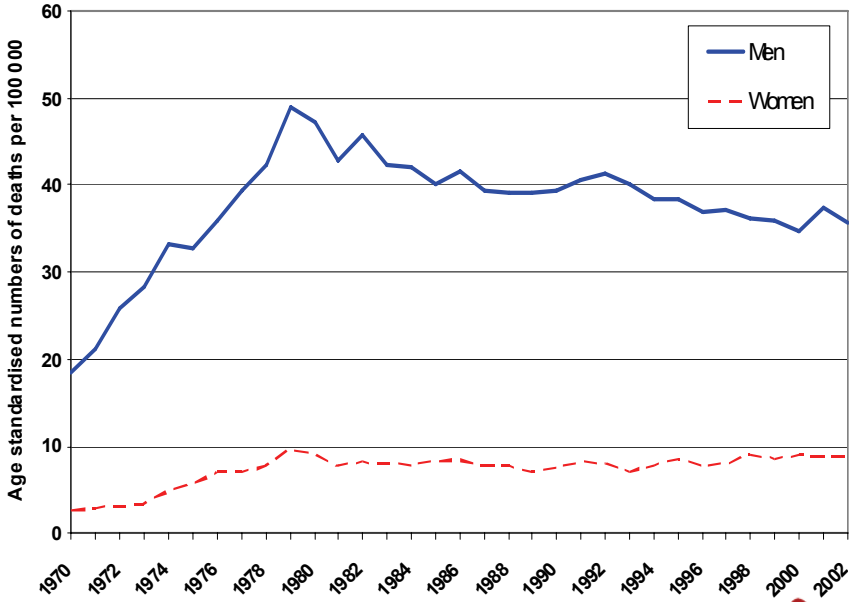
This trend break could easily have been anticipated if declining smoking rates had been considered. Smoking rates among men started to decline in the early 1960s accompanied with a trend break for lung cancer about 20 years later. Smoking rates among women have increased up to the late 1970s followed by a small decrease in smoking rates. So far, no shift in lung cancer rates among women can be seen. However, lung cancer rates among women seem to be levelling off.

Alcohol-related mortality rose dramatically after the abolishment of the Swedish rationing system in 1955 and it was first around 1980 a decreasing alcohol mortality trend was noticed (Figure 3). This trend break was probably due to intensified efforts in society as a whole. Cohorts born in the 1960s and



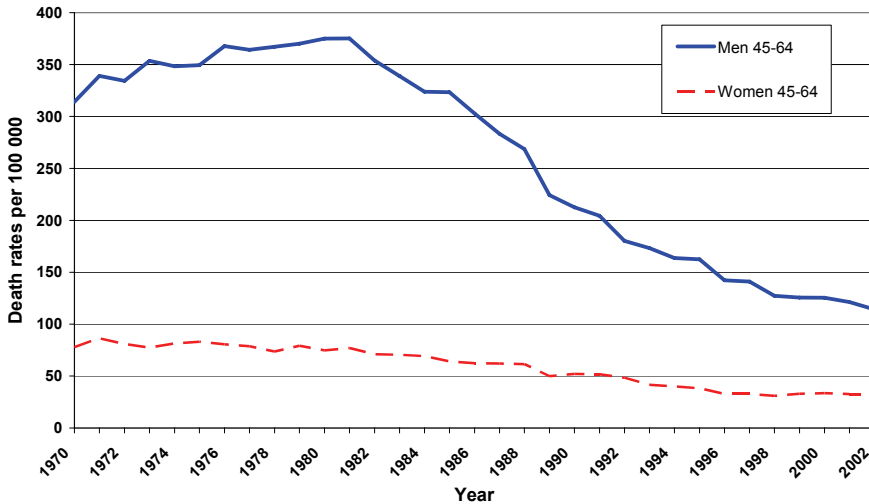
1970s also seem to be very healthy cohorts with low smoking rates and moderate alcohol consumption.

**Figure 3 Alcohol-related mortality in Sweden 1970-2002**



The development of acute myocardial infarction and other coronary heart diseases among middle-aged men is another example of a trend break (Figure 4). This trend break took place in the beginning of the 1980s and was due to several changes in risk factors, especially the decline in smoking rates among men. The level of serum cholesterol has also decreased in the Swedish population contributing to a decreasing trend in coronary heart mortality. All these examples clearly indicate caution in respect to merely extrapolating mortality trends.

**Figure 4 Ischaemic heart disease mortality in Sweden 1970-2002, 45-64 years**



## Predicting Mortality Based on Potential Elimination of Causes of Death

To gain an idea of how great the potential is for increasing life expectancy one can do hypothetical calculations of how much it would increase if a disease no longer led to death (Curtin and Armstrong 1988; Haglund and Rosén 2001). In the Swedish Public Health Report of 2001 such calculations have been made (Haglund and Rosén 2001). The results are summarised in Table 1, which shows that the elimination of cardiovascular disease as a cause of death is the single most important step to prolong life expectancy followed by cancer. For cardiovascular disease more than 5 years could be gained for men by eliminating this disease group. Many may be surprised by the small gains obtained by eliminating traffic accidents (3 months for men and 1 month for women) or infectious diseases (1 month).

Social factors play an important role in the etiology of diseases and for mortality predictions. Upper white-collar workers have the lowest mortality. If the death risk for the whole population between 25 and 74 were reduced to the same level as for upper white-collar workers, men's life expectancy would have been 2 years and 5 months longer and women's life expectancy one year and 5 months longer (Haglund and Rosén 2001).

**Table 1** Changes in life expectancy in months through elimination of certain diseases or risk factors in Sweden 1993-1997

<b>Disease/risk factor</b>	<b>Men</b>	<b>Women</b>
Infections	1	1
Cancer	34	37
Cardiovascular diseases	64	48
Injuries and accidents	12	6
Traffic accidents	3	1
Tobacco	16	8
Social inequalities	29	17

*Source:* Haglund and Rosén (2001)

## **Predicting Mortality Based on Development of Risk Factors**

Predicting mortality based on social developments and predictions on risk factor changes seems most appropriate since these are the driving forces for mortality. The major problems are the lack of knowledge we have concerning all risk factors affecting all diseases. The three most important risk factors for coronary heart disease (CHD) are smoking, hypertension and high blood cholesterol levels. However, 247 risk factors for CHD have been suggested in the scientific literature (Hopkins and Williams 1981). It is impossible to make predictions for all these and many of them are not very well evidence based. Still, the three major risk factors explain quite a large proportion of CHD deaths and it is therefore much easier to predict the future CHD trends than to predict mortality for other causes of death, e.g. cancer where the knowledge base is more limited. Since about half of all deaths are caused by cardiovascular disease, it seems meaningful to make mortality predictions based on the risk factor development of this disease group.

## **Methodological Problems in Predicting Mortality Based on Risk Factor Predictions**

In this paper I advocate a risk factor prediction approach to mortality forecasting. I hope the earlier presentation convincingly has shown the advantages of this approach in comparison with extrapolating mortality trends.

However, several methodological problems still exist. Four problems could be high-lighted. Relative risks vary over time and by regions, latency times differ, co-morbidity and competing causes of death complicate the predictions and the lack of appropriate risk factor data limit the possibilities.

In the case of coronary heart disease, longitudinal studies from different parts of the world have displayed the same major and independant risk factors, but with varying relative risks (Wilhelmsen et al. 2004; Conroy et al. 2003; Empana, Ducemetiere, Arveiler et al. 2003). The Framingham risk functions based on U.S. populations overestimate the absolute coronary heart disease risk of middle-aged men when they are applied to different European populations (Empana et al. 2003). A problem in estimating mortality trends is the long latency times between exposure to risk factors and when the individuals are strucked by the disease. For smoking and lung cancer latency time is usually more than 20 years of smoking. These kinds of considerations must be taken into account when making mortality predictions. However, the greatest problem in mortality modelling is usually lack of reliable risk factor data. Our own experiences of testing the Dutch mortality model (Gunning-Schepers 1989) on Swedish data showed the lack of risk factor data even in a data affluent society like Sweden.

## **Future Mortality and Longevity**

As a simple exercise, Rosén and Haglund (2002) estimate future life expectancy in Sweden, not based on sophisticated dynamic population models, but merely on assumptions about risk factor developments and general knowledge about public health, recent successes in health care and the potential of eliminating certain causes of death (Table 2).

Social differences in mortality are large even in economically well-developed countries like Sweden. The reasons for these differences are multi-factorial and are most likely due to an accumulation of health risks during the whole life-cycle. Lower socio-economic groups have usually lower birth weights, have been brought up in more disadvantaged areas, have less education, smoke more, eat more unhealthy products, have more often monotonous work or are more often unemployed. However, history has shown that lower socio-economic groups will eventually reach the life expectancy of higher socio-economic groups, but that they are always 10-20 years behind. Eliminating the present social differences in health seems therefore a realistic scenario.

**Table 2** Future estimated life expectancy (years) in Sweden

<b>Intervention</b>	<b>Men</b>	<b>Women</b>
Life expectancy, year 2000	77.4	82.0
Eliminating social differences	+2.5	+1.5
Improved lifestyle	+1.5	+1.5
Improved medical care	+1.5	+0.5
Optimist supplement	+1.0	+1.0
Estimated life expectancy	84.0	87.0

*Source:* Rosén and Haglund (2002)

It is also obvious that eliminating cardiovascular disease has the greatest impact on longevity. This is an area where we have evidence based knowledge of risk factors and great potential for primary prevention. Since the 1990s medical technologies have had a success story in developing life-saving interventions in the field of coronary heart disease. All together, this implies a high potential for improving longevity by reducing mortality for cardiovascular disease. We estimated a gain of 1.5 years due to improved lifestyle, mainly reduced smoking rates, and further gains due to improved medical technologies of 1.5 years for men and 0.5 years for women. The larger estimated gain for men is due to the fact that medical interventions will influence cardiovascular disease most, which is a larger burden for men than women. Finally, we added an optimist supplement of one year for improvements not foreseen by our estimates.

## **Implications for the Future**

Mortality forecasting plays an important role for development and maintenance of national and private insurance schemes. However, there are also other social and economic consequences of changing mortality trends. A lively discussion has been whether prolonging lives may lead to compression or expansion of morbidity (Thorslund, Lennartsson, Parker and Lundberg 2004). Many studies in the past have indicated decreasing morbidity and improved functional status among the elderly, i.e. supporting the hypothesis of compression of morbidity. Recent studies in Sweden show, however, deteriorating health in some aspects among the elderly (Thorslund et al. 2004; Rosén and Haglund 2005). This development supports the hypothesis that we now are going from healthy survivors to sick survivors due to improvement in health care (Rosén and Haglund 2005). Since the late 1980s,

new and very effective life-saving drugs and treatments have been developed, especially in the field of cardiovascular disease. This has had tremendous effect on survival among patients with acute myocardial infarction, heart failure and diabetes. Those surviving will, however, live with their chronic diseases and demand more care than earlier “healthy” survivors.

## References

Conroy R.M., K. Pyörälä, A.P. Fitzgerald, S. Sans, G. Menotti, D. De Bacquer et al. (2003). “Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project.” *European Heart Journal*, 24, 987-1003.

Curtin L.R. and R.J. Armstrong (1988). “United States Life Tables Eliminating Certain Cases of Death. U.S. Decennial Life Tables for 1979-81.” Vol 1, No 2. *DHHS Pub. No (PHS) 88-1150-2*, Public Health Service. Washington DC: U.S. Government Printing Office.

Empana J.P., P. Ducemetiere, D. Arveiler et al. (2003). “Are the Framingham and PROCAM coronary heart disease risk functions applicable to different European populations? The PRIME Study.” *European Heart Journal*, 24, 1903-1911.

Gunning-Schepers L. (1989). “The health benefits of prevention: simulation approach.” *Health Policy*, 12, 1-255.

Gunning-Schepers L., J.J. Barendregt and P.J. Van Der Maas (1989). “Population interventions reassessed.” *Lancet*, 1, 479-481.

Haglund B. and M. Rosén (2001). “Public health in the future.” *Scandinavian Journal of Public Health*, suppl. 58, 231-239.

Hopkins P.N. and R.R. Williams (1981). “A survey of 246 suggested risk factors.” *Atherosclerosis*, 40, 1-52.

Kruijshaar M.E., J.J. Barendregt and N. Hoeymans (2002). “The use of models in the estimation of disease epidemiology.” *Bulletin World Health Organisation*, 80, 622-628.

National Board of Health and Welfare (2003). *A finger on the pulse. Monitoring public health and social conditions in Sweden 1992-2002*. Stockholm: Centre for Epidemiology, National Board of Health and Welfare.

Nemtsov A.V. (2002) “Alcohol-related human losses in Russia in the 1980s and 1990s.” *Addiction*, 97, 1413-1425.

Notzon F.C., Y.M. Komarov, S.P. Ermakov, C.T. Sempos, J.S. Marks and E.V. Sempos (1998). “Causes of declining life expectancy in Russia.” *JAMA*, 279, 793-800.

Rosén M. and B. Haglund (2005). "From healthy survivors to sick survivors - implications for the twenty-first century." *Scandinavian Journal of Public Health*, 33(2), 151-155.

Rosén M. and B. Haglund (2002). "Hur länge kommer vi att leva?" *Framtider*, 1, 16-19.

Shkolnikov V., M. McKee and D.A. Leon (2001). "Changes in life expectancy in Russia in the mid-1990s." *Lancet*, 357, 917-921.

Thorslund M., C. Lennartsson, M.G. Parker and O. Lundberg (2004). "De allra äldstas hälsa har blivit sämre. Könsskillnaderna är stora - kvinnorna mår sämre visar nya data." *Läkartidningen*, 101, 1494-1499.

WHO Europe Health-for-All-Database, see <http://www.who.dk>

Wilhelmsen L., G. Lappas and A. Rosengren (2004). "Risk of coronary events by baseline factors during 28 years follow-up and three periods in a random population sample of men." *Journal of Internal Medicine*, 256, 298-307.



# How Analysis of Mortality by Cause of Death is Currently Influencing UK Forecasts

**Richard Willets**

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Managing Director, Willets Consulting Limited

The purpose of this paper is to examine the potential benefits of cause of death analysis in the context of projecting future mortality rates in the UK.

In the first section of the paper the main features of recent mortality trends in the UK are briefly described. In the second methods currently used to project mortality in the UK are outlined. Current issues and topics for research are also discussed. In section three potential causes of the “UK cohort effect” are listed and the role of cigarette smoking, in particular, is discussed. A model of mortality which includes a year of birth component is discussed in section four. It is argued that models such as this can be used to analyse mortality from different causes and this analysis can have important benefits. Conclusions and implications are given in section five.

Throughout the paper most emphasis is placed on understanding and modelling mortality trends for older adults. This part of the age range is currently the focus of most research in the UK and has the greatest financial significance in terms of its impact on pension schemes and public finances.

## **1. Mortality Improvement in the UK**

In common with many developed countries round the world, the UK has recently experienced substantial reductions in mortality rates. The pace of improvement, especially at older ages, has accelerated strongly as the figures in Table 1 demonstrate.

**Table 1      Reduction in the mortality rate for males aged 65-74 in the England & Wales population since 1901**

<b>Time period</b>	<b>Reduction in the mortality rate for males aged 65-74</b>
1901 to 1969 (68 years)	19.4%
1969 to 1986 (17 years)	21.6%
1986 to 1996 (10 years)	19.4%
1996 to 2002 (6 years)	21.1%

*Source:* The Office for National Statistics

Table 1 shows that broadly the same fall (circa 20%) in the rate of mortality for males aged 65-74 has occurred in successive periods of 68, 17, ten and then six years. The pace of change at the beginning of the 21<sup>st</sup> century has therefore been more than ten times as rapid as that seen in the first seven decades of the 20<sup>th</sup> century.

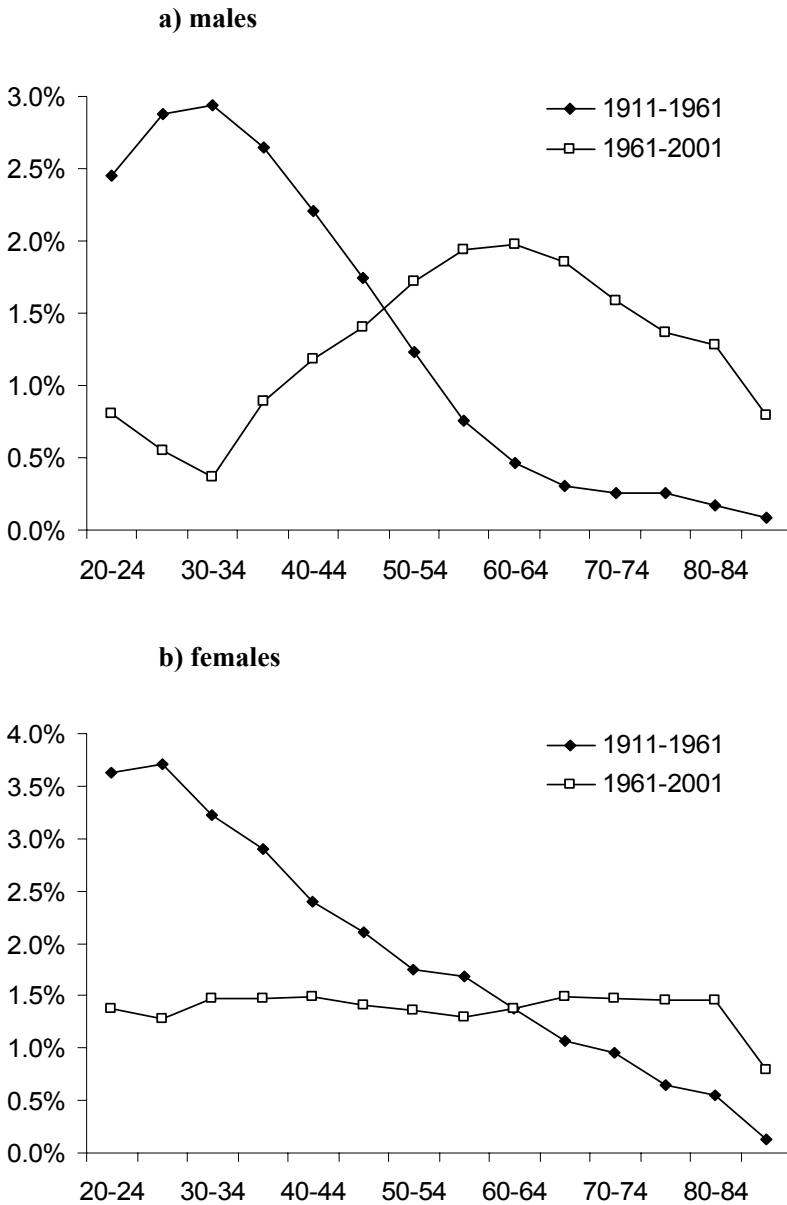
This simple example illustrates the extent to which the pace of change in mortality rates at older ages has improved over time. More generally, we have seen a trend towards faster improvements at older ages, but less rapid change at younger ages. This feature of mortality change has applied to many developed countries and is sometimes referred to as the “aging of mortality improvement” (Wilmoth 1997).

Figures 1a and 1b illustrate this trend of more rapid improvement at increasingly advanced ages by comparing average annual rates of mortality improvement<sup>1</sup> over the past four decades with rates for the previous 50 years.

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<sup>1</sup> Throughout this paper the term “mortality improvement rate” is taken to mean the rate of change in the mortality rate at a given age from year to the next, i.e.  $1 - m(x,t) / m(x,t-1)$ , where  $m(x,t)$  is the central mortality rate for age  $x$  and time  $t$ .

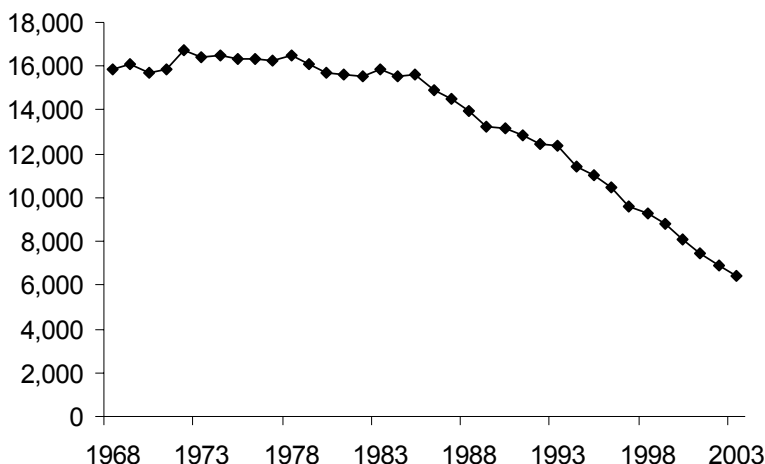
**Figure 1** Average annual mortality improvement rates, England & Wales population, 1911-2001



Source: The Office for National Statistics

In terms of individual causes of death, the single most important driver of these accelerated improvements has been the substantial reduction in heart disease mortality seen in recent decades. This is illustrated by Figure 2a, which shows the crude rate of heart disease mortality for men aged 65 to 74.

**Figure 2a Heart disease deaths per 1,000,000, ages 65-74, England & Wales population, 1968-2003, males**



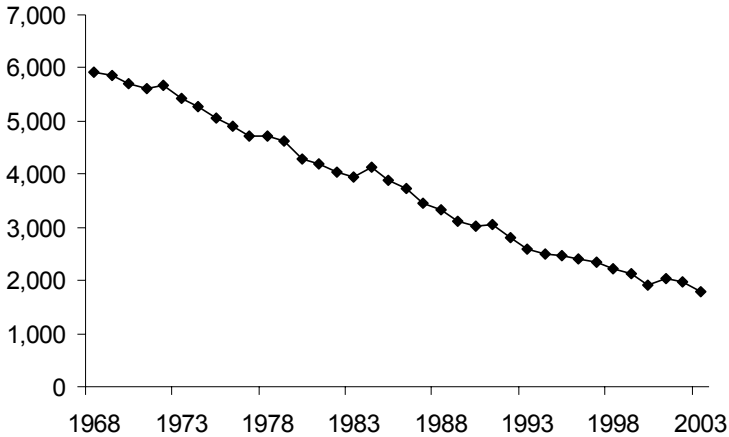
Source: The Office for National Statistics

Figure 2a shows that the death rate from heart disease for men aged 65-74 has fallen by almost 60% since 1985. The reduction is equivalent to an annual rate of improvement of 4.8% p.a.

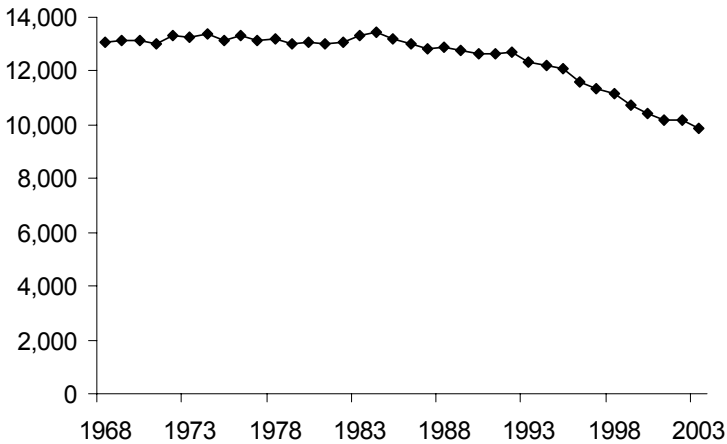
The major contributor to the decline in heart disease mortality is believed to be reduced cigarette smoking prevalence (Kelly and Capewell 2004). However, reduction in population blood pressure and cholesterol levels and improvements in treatment have also played a significant role. These positive trends have comfortably outweighed the impact of adverse trends in obesity, diabetes and lack of physical activity, which together contributed approximately 8,000 extra deaths in England & Wales between 1981 and 2000 (Unal et al. 2004).

There have also been substantial reductions in other leading causes of death, such as stroke and cancer, at these ages, as illustrated by Figures 2b and 2c.

**Figure 2b** Stroke deaths per 1,000,000, ages 65-74, England & Wales population, 1968-2003, males



**Figure 2c** Cancer deaths per 1,000,000, ages 65-74, England & Wales population, 1968-2003, males



Source: The Office for National Statistics

However, the major contributor to the recent rapid improvement in mortality at older ages has been heart disease. Willets et al. (2004) showed that over half of the recent mortality improvement for men in their 60s in England &

Wales was due to heart disease alone. The figures in Table 2 also show that most of the remainder of the improvement was due to reductions in stroke and cancer mortality.

**Table 2 Breakdown of contributions to the overall rate of mortality improvement over the period 1989-2001, by cause, England & Wales population, males**

<b>Cause</b>	<b>Ages 30-39</b>	<b>Ages 60-69</b>
Heart disease	+0.4%	+1.8%
Stroke	+0.3%	+0.3%
Cancer	+0.1%	+0.9%
AIDS	+0.3%	0.0%
Drugs/alcohol/liver disease	-0.8%	0.0%
Violent/accidental	-0.3%	+0.3%
All others	0.0%	+0.4%
Aggregate	-0.1%	+3.4%

It is also worth noting that at younger ages, such as the 30-39 age group, improvements in mortality due to heart disease, stroke and cancer have been more than offset by adverse trends in other causes, notably those linked to drug and alcohol abuse.

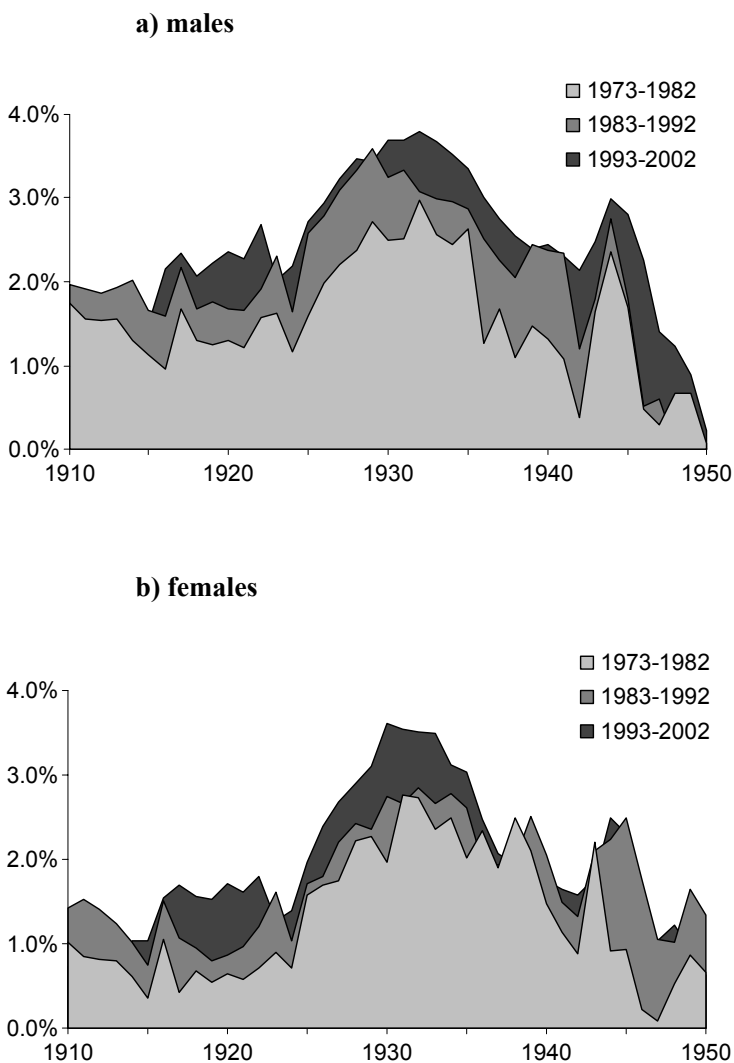
In addition to this trend of accelerating improvements at older ages, UK patterns of mortality change have also been influenced by the feature sometimes referred to as the “UK cohort effect”.

Figures 3a and 3b show how the pace of improvement (the average annual reduction in mortality rates) has varied by year of birth in successive ten-year periods. Three features are evident in both figures:

- The pace of improvement has been consistently higher for people born in the period 1925-1945 than for people born either side of this generation (this being the so-called “UK cohort effect”). In particular more rapid improvement has been a feature of mortality change for those born in the period 1925 to 1935, with a secondary peak around 1945.
- Many of the peaks and troughs in the rates of improvement are replicated over time, showing the enduring significance of year of birth in determining the pace of change at different ages.

- The pace of improvement within birth cohorts has increased over time. In other words, the pace of improvement has been accelerating in recent decades, after allowing for the impact of the cohort effect.

**Figure 3** Rate of mortality improvement by year of birth and ten-year period, England & Wales, smoothed using 7-year rolling averages

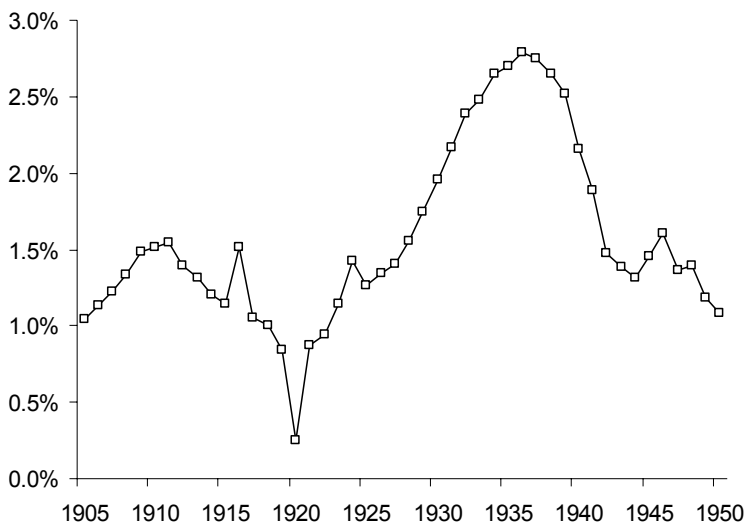


Source: The Office for National Statistics

It is also worth noting that a similar effect can be seen in other developed countries. Data from the Human Mortality Database maintained by the University of California, Berkeley (USA) and Max Planck Institute for Demographic Research (Germany) was analysed for 17<sup>2</sup> developed countries (all those included on the database excluding those in Eastern Europe). For each of the 17 countries data from 1950 to 2003 for ages 40 to 89 (subject to the years available in each case) were used to calculate average rates of mortality improvement by year of birth.

The results are illustrated by Figure 4. It is notable that the pace of improvement has been significantly more rapid for males born in or around 1935-1940 than generations born before or after this period<sup>3</sup>.

**Figure 4** Average annual rate of mortality improvement by year of birth for 17 developed countries, males, ages 40 to 89, data from 1950 to 2003, figures smoothed using rolling averages



Source: Human Mortality Database

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<sup>2</sup> Austria, Belgium, Canada, Denmark, England & Wales, Finland, France, Italy, Japan, Netherlands, New Zealand, Norway, Spain, Sweden, Switzerland, USA and West Germany.

<sup>3</sup> It is also worth noting that the trough in the pace of improvement for the “1920-born” generation is actually far lower than Figure 4 suggests. Without the impact of smoothing, the figure for this year is minus 7.8% p.a. This feature is likely to have been caused by the impact of the 1919 influenza pandemic.



## 2. Current Methodologies and Research in the UK

Mortality projections for the UK population are currently produced by the Government Actuary's Department (GAD). The projections assume that "current" rates of mortality improvement – based on the most-recent trends in aggregate mortality – will converge with target rates over a 25-year time-frame.

The latest projection, the so-called "2002-based" projection (GAD 2003), assumed target rates of improvement of 1.0% p.a. for both males and females.

The rates of improvement are projected on a cohort basis for generations born prior to 1947.

A number of variant projections are also made using alternative improvement scenarios.

Projections of future mortality for pensioners and annuitants in the UK are produced by the Continuous Mortality Investigation (CMI), a research group of the UK Actuarial Profession. The last official projection, published in conjunction with the "92 series" of mortality tables for pensioners and annuitants, is based on assumed rates of future improvement in historic trends by age group (CMI 1999). Rates of improvement in the so-called "CMIR17" basis were assumed to diminish over time, consistent with the idea of ultimate (or minimum) rates of mortality.

In 2002 interim "cohort" projections were published by the CMI (CMI 2002) which combined the rates of change in the CMIR17 basis with blocks of rapid improvement consistent with the projection of the UK cohort effect into advanced ages. Three variant projections were produced which differed in the extent to which the cohort effect was assumed to be projected forwards into the future.

Both the GAD and CMI projection methodologies project aggregate rates of mortality, rather than using a cause-of-death methodology.

Cause-of-death modelling has not, and is not, generally favoured as an approach for projecting future mortality rates in the UK. The 1976-based GAD projection of UK population mortality did model future improvements for ten distinct groups of causes of death. However, this methodology was not adopted for future projections and a major review of the projection methodology for the UK population (GAD 2001) concluded that: *"projections of mortality should not be carried out by cause of death"*.

A similar review paper published by the CMI (CMI 2004) sought feedback from the UK Actuarial Profession on the methodology to be adopted for future mortality projections. One question it asked was whether projections should be carried out on an aggregate or a cause-of-death basis. The response was overwhelmingly in favour of an aggregate methodology.

Arguments against cause-of-death projections included:

- interactions between different causes are difficult to model, especially at older ages;
- medical/research effort will shift as the relative importance of different causes changes over time;
- there are problems with classifying the true cause of death in the very old; and
- changing methods of cause classification over time can distort trends.

Much of the research on future mortality improvement currently being carried out in the UK is being driven by regulatory change in the UK insurance industry which favours a stochastic approach to modelling risk.

Major topics of research include all forms of stochastic mortality modelling, such as Lee-Carter and variants, and advanced methods of smoothing mortality surfaces such as p-splines (CMI 2005).

However, two big questions at the time of this writing – at least for the UK insurance and pensions industries – are:

- whether the UK cohort effect will be projected forwards into the future as the 1925-45 generation ages; and,
- whether the pace of improvement in mortality rates for older ages will continue to accelerate.

For instance, a Guidance Note recently published by the UK Actuarial Profession (2004) states that in determining the capital requirements of an insurance company:-

*“the ICA [Individual Capital Assessment] should consider firstly, with justification, how any historically observed trends (including cohort effects) might continue, or might continue to accelerate or decelerate.”*

It is difficult to see how such a justification could be obtained without a consideration of the underlying causes of mortality trends, such as the cohort effect.

### 3. Understanding the “UK Cohort Effect”

A number of possible causes for the UK cohort effect have been discussed. These include:

- patterns of cigarette consumption;
- the adverse impact of World War II;
- patterns in birth rates;
- the development of the UK Welfare State after World War II;
- the impact of diet in early life/maternal malnutrition; and
- early life exposure to infectious disease/lifetime levels of inflammation.

These possible causes are discussed in more detail in Willets (2004). However, to illustrate the value of considering mortality trends for different causes of death, the impact of cigarette smoking will be analysed in more detail here.

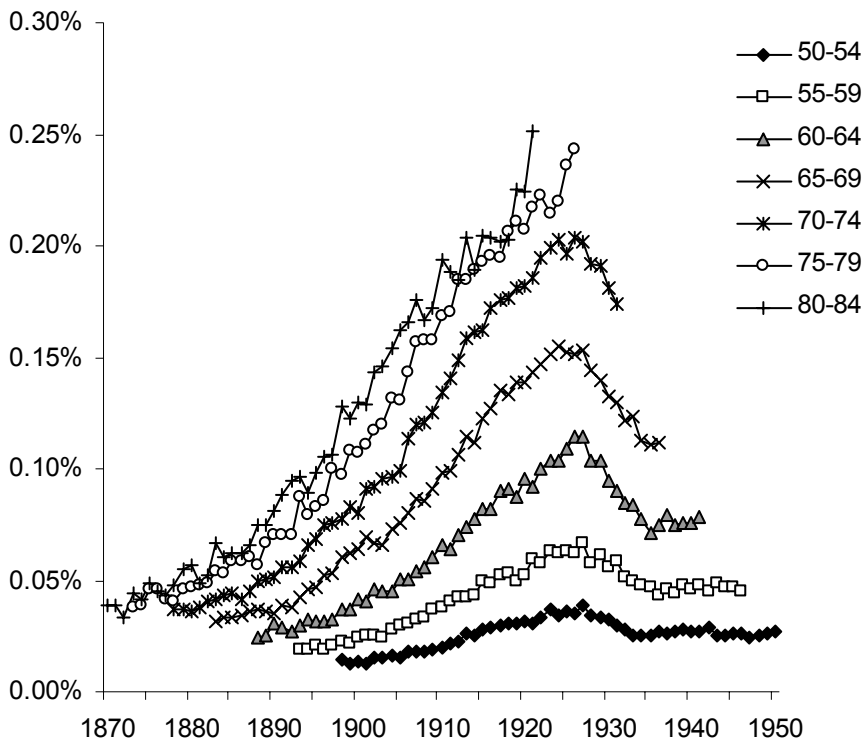
Cohort effects in lung cancer mortality rates have been well-documented in recent decades (see, for example, Caselli 1996). Indeed, in women especially, the trends in lung cancer mortality in the UK have been described as providing “*an almost perfect example of a cohort effect*” (Office for National Statistics 1997).

Figure 5 shows rates of lung cancer mortality for females in England & Wales by year of birth. It can clearly be seen that the rate of lung cancer deaths at each age group has peaked for those women born in or around 1925.

A similar pattern can be seen for males in England & Wales, with the peak rates of lung cancer mortality occurring for men born in or around 1905.

This data closely matches the pattern shown in figures for lifetime consumption of cigarette tar by year of birth (Lee et al. 1990). There is, therefore, strong evidence that trends in lung cancer mortality by year of birth are correlated with trends in cigarette consumption by year of birth. As a result it is a relatively straightforward task to project future rates of lung cancer mortality for mature generations of UK lives.

**Figure 5** Rate of lung cancer mortality for females in England & Wales by year of birth, using data from 1950 to 2003



Source: The Office for National Statistics

As a result of this clear link between lifetime smoking behaviour and mortality from one of the major smoking-related causes of death, it is sometimes argued that the UK cohort effect is unlikely to be projected forwards far into the future.

This argument is based on the suggestion that the UK cohort effect has been largely caused by past patterns in smoking, but that cigarette smoking prevalence has now stabilised in the UK (Office for National Statistics 2004). Furthermore it is argued that smoking-related causes of death (such as lung cancer) are less significant in relative terms at older ages.

In order to explore whether this theory is supported by experience, it is useful to consider historic trends in different causes of death.

#### 4. Modelling Mortality by Cause of Death

Tables 3a to 3c illustrate the pattern of mortality improvement in three major causes of death for females in England & Wales, namely lung cancer, heart disease and breast cancer. In each case the average annual rate of improvement has been derived for successive periods of ten years using log linear regression on cause-specific mortality rates.

**Table 3a** Average annual rate of lung cancer mortality improvement in successive 10-year periods, England & Wales population, females

Age group	1973-1983	1983-1993	1993-2003
40-44	+1.5%	-1.9%	+4.2%
45-49	<b>+3.7%</b>	+0.3%	+0.4%
50-54	+0.4%	+0.2%	+0.5%
55-59	-2.7%	<b>+3.8%</b>	-0.5%
60-64	-3.7%	+0.3%	+1.3%
65-69	-4.7%	-1.9%	<b>+3.5%</b>
70-74	-4.8%	-2.6%	+0.7%
75-79	-4.5%	-3.1%	-2.0%
80-84	-4.3%	-3.5%	-2.4%

Source: The Office for National Statistics

Table 3a clearly shows that the rate of improvement in lung cancer mortality has been particularly rapid for a group of females born in the same period, who are ten years older in each successive ten-year period. The shaded figures relate chiefly to women born in or around 1930. It is notable that the pace of improvement for this cohort has remained relatively constant over time and also that the pace of improvement for adjacent age groups has been far lower.

The equivalent figures for heart disease mortality are given in Table 3b.

**Table 3b** Average annual rate of heart disease mortality improvement in successive 10-year periods, England & Wales population, females

Age group	1973-1983	1983-1993	1993-2003
40-44	+4.4%	+2.9%	+2.2%
45-49	+3.6%	+5.4%	+1.4%
50-54	+0.6%	+5.3%	+5.0%
55-59	-0.1%	+4.8%	+6.6%
60-64	+0.4%	+3.2%	+7.1%
65-69	+0.7%	+2.2%	+7.2%
70-74	+1.1%	+2.1%	+6.3%
75-79	+1.2%	+1.6%	+5.2%
80-84	+1.5%	+1.0%	+4.7%

Source: The Office for National Statistics

The pattern of improvement is somewhat different for heart disease. It is evident that the pace of improvement has accelerated over time for all birth cohorts, but that the most rapid pace of change has applied, consistently, to a much wider range of birth years than was the case for lung cancer.

**Table 3c** Average annual rate of breast cancer mortality improvement in successive 10-year periods, England & Wales population, females

Age group	1973-1983	1983-1993	1993-2003
40-44	+0.8%	+0.9%	+3.6%
45-49	+0.5%	+1.6%	+3.9%
50-54	-0.6%	+1.6%	+3.5%
55-59	-1.1%	+1.7%	+2.8%
60-64	-0.7%	+0.6%	+2.9%
65-69	-0.5%	+0.2%	+3.2%
70-74	-1.1%	+0.3%	+2.6%
75-79	-0.6%	-0.5%	+1.3%
80-84	-1.0%	-1.1%	+1.9%

Source: The Office for National Statistics

In the case of breast cancer the pace of improvement has also accelerated over time, but was generally greatest for those aged under 50 in 1973-1983, those aged under 60 in 1983-1993 and those aged under 70 in 1993-2003.

Tables 3a to 3c only give an approximate indication of cohort effects, by considering age-related improvements in successive periods of time. A more formal analysis can be achieved by modelling mortality rates using an approach in which year of birth parameters are included.

Such a model was constructed using a database of England & Wales population experience for the period 1968 to 2003. This period covers the years when deaths were classified using ICD8, ICD9 and ICD10; three versions of the International Classification of Diseases.

Raw deaths data for years from 1968 to 2000 were taken from the *20<sup>th</sup> Century Mortality* database produced by the Office of National Statistics (ONS). More recent data relating to the period 2001 to 2003 were taken from the *21<sup>st</sup> Century Mortality* database (ONS).

Mid-year population estimates were also taken from the most up-to-date ONS publications which incorporate the most recent revisions resulting from the 2001 Census results (October 2004).

For each calendar year (1968 to 2003) death numbers, split by 5-year age groups (up to 80-84) by gender and cause of death, were divided by the equivalent mid-year population estimates. Hence, central mortality rates for 5-year age bands were derived.

Using these central mortality rates for each age group ( $x$ ) and calendar year ( $t$ ), mortality improvement rates were calculated for age groups between 40-44 and 80-84 inclusive, i.e.

$$\text{Improvement rate, } \delta(x, t) = 1 - m(x, t) / m(x, t-1)$$

Each improvement rate was then assigned to one central year of birth. For example, the improvement rate for the 60-64 age group, for calendar year 2003, was assigned to year of birth 1941.

Various models can be constructed to decompose the *rates of improvement* for different causes using a combination of age, period and cohort factors. Age-period-cohort models have been widely used by epidemiologists and demographers to model mortality rates (see, for example, Tabeau 2001). These models are commonly fitted to log mortality rates. However, for this

purpose, the rates of improvement themselves (i.e. the  $\delta(x, t)$  terms) have been modelled. This approach is felt to produce results which are relatively easy to interpret and adapt for the projection of future mortality rates.

One feature of age-period-cohort models is that they do not provide a unique solution because of the interdependence of the three terms. There are various strategies to overcome this “identification problem”. However, for this particular paper, it was decided to consider the results of a simplified version of the model only, i.e. one with just period and cohort terms:-

$$\delta(x, t) = \beta(t) + \gamma(t-x)$$

where  $t$  = calendar year,  $t-x$  = year of birth,  $\sum w\gamma(t-x) = 0$ ,  $w$  = a weighting factor for each cohort taken as the number of deaths observed for that cohort.

This approach can be justified because the model fits rates of improvement rather than log mortality rates.

The missing age term is of far less significance than would be the case with a traditional age-period-cohort model. In fact, this age term can be seen as equivalent, in very broad terms, to the  $b(x)$  term in the Lee-Carter model (Lee and Carter 1992), where:

$$\log m(x,t) = a(x) + b(x) k(t)$$

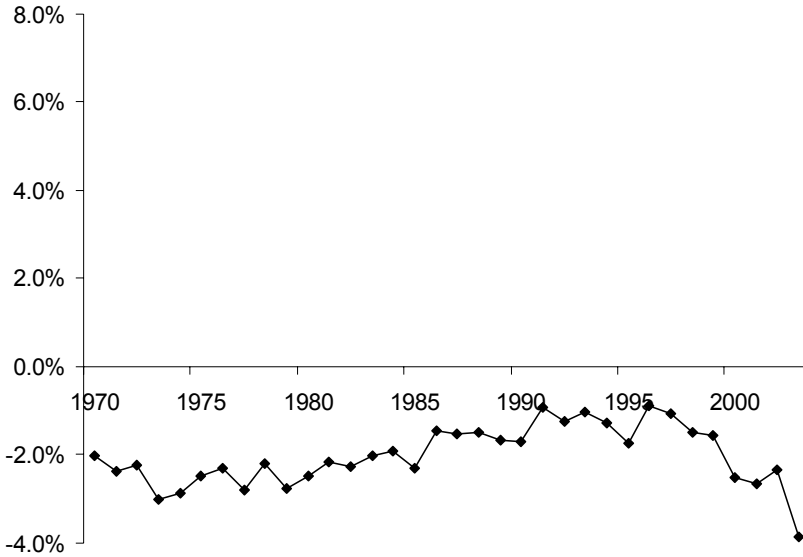
Most significantly there is not a clear pattern to the model residuals by age and time, which would indicate a poor fit.

The model can be fitted using a weighted least squares approach applied directly to actual and expected improvement rates or by using a maximum likelihood or minimum chi-squared function derived for the underlying mortality rates. All three approaches give similar results. In this instance results derived by applying the maximum likelihood approach have been used.

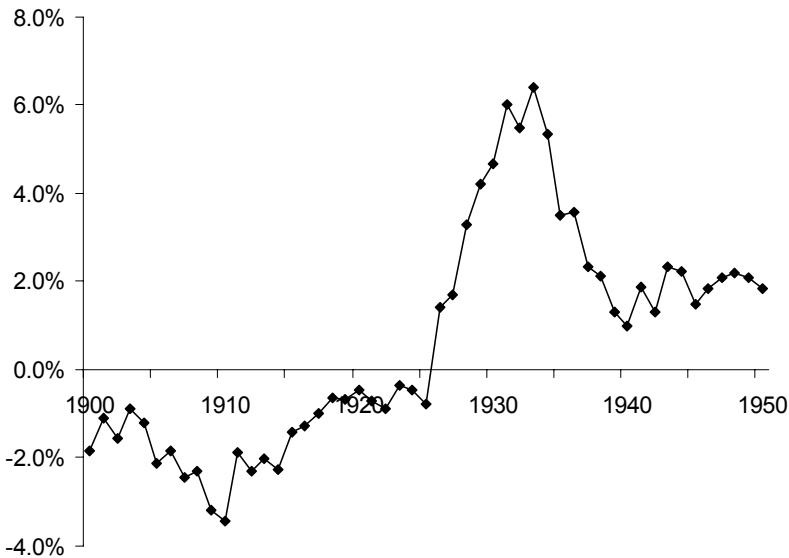
The two functions (i.e. period and cohort) derived from fitting the simplified model to cause-specific mortality data for females are given in Figures 6a to 6f. In each case rolling averages were used to identify underlying patterns in the data. In the case of the period function, the improvement rates for 1984 and 1993 were removed from the analysis as they were distorted by changes in the methodology used to assign a main cause to a death certificate. Likewise the rates for 2001 were also removed because ICD10 was first applied as a method of cause classification in this year.



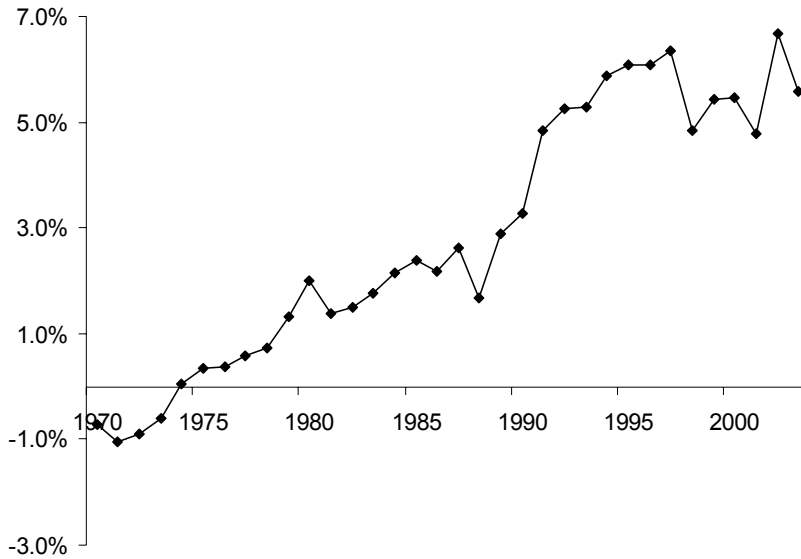
**Figure 6a** Period function  $\beta(t)$  derived by fitting a model of mortality improvement to lung cancer rates for females, England & Wales, 1968-2003



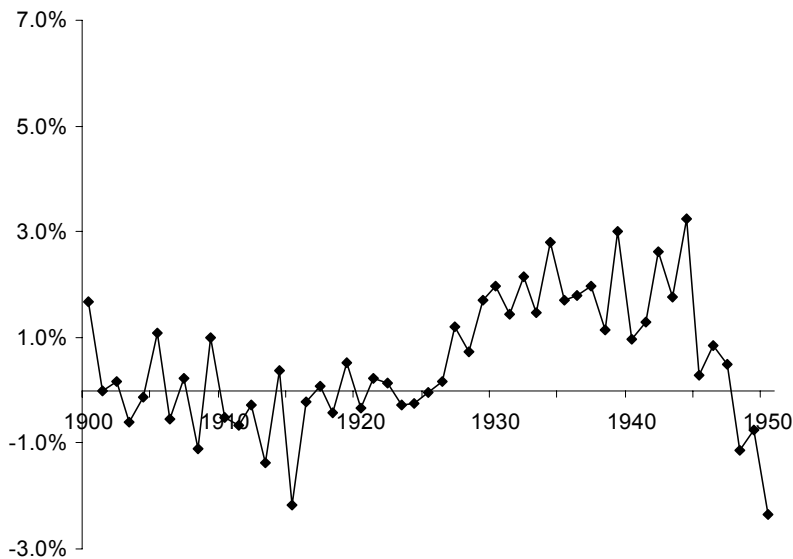
**Figure 6b** Cohort function  $\gamma(t-x)$  derived by fitting a model of mortality improvement to lung cancer rates for females, England & Wales, 1968-2003



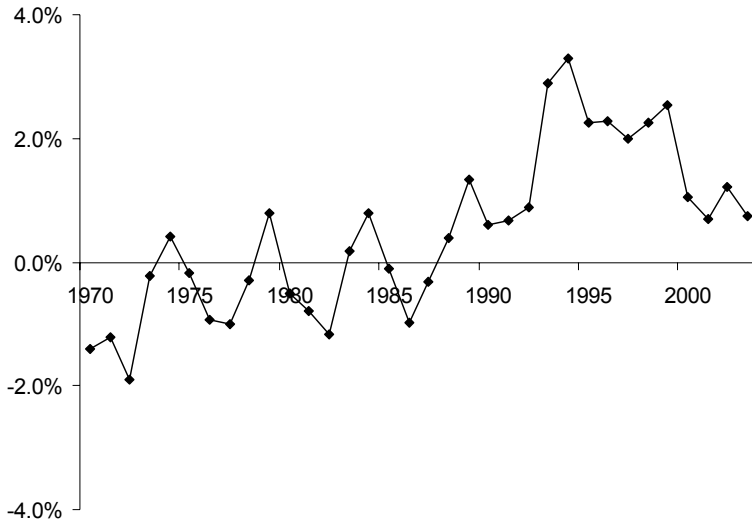
**Figure 6c** Period function  $\beta(t)$  derived by fitting a model of mortality improvement to heart disease rates for females, England & Wales, 1968-2003



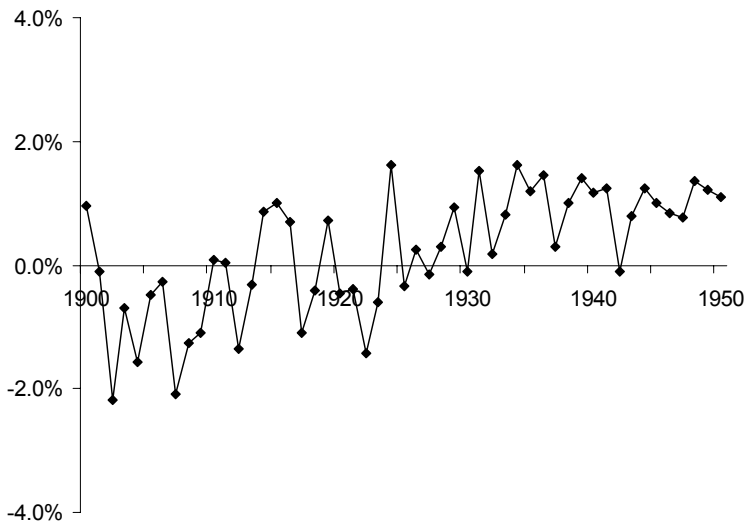
**Figure 6d** Cohort function  $\gamma(t-x)$  derived by fitting a model of mortality improvement to heart disease rates for females, England & Wales, 1968-2003



**Figure 6e** Period function  $\beta(t)$  derived by fitting a model of mortality improvement to breast cancer rates for females, England & Wales, 1968-2003



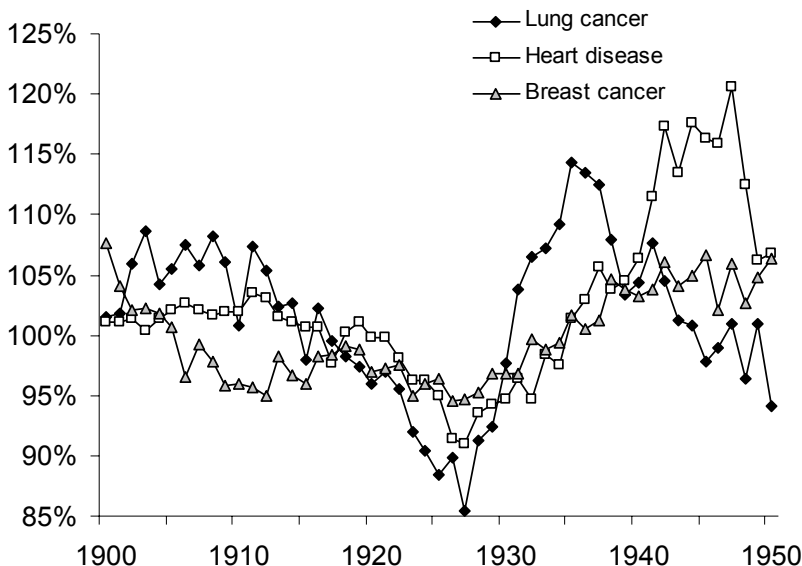
**Figure 6f** Cohort function  $\gamma(t-x)$  derived by fitting a model of mortality improvement to breast cancer rates for females, England & Wales, 1968-2003



It is interesting to note that the pattern of the cohort function is very different for lung cancer and heart disease. There is clear evidence that the cohort effect applies to later-born generations in the case of heart disease. This does not correlate well with trends in lung cancer or cigarette consumption by generation.

Another way of exploring how year of birth factors have influenced trends in different causes of death is to analyse how well a basic Lee-Carter model fits mortality rates for different birth years. This approach is illustrated by figure 7 in which fitted and actual rates are compared. For the three causes of death analysed the Lee-Carter model systematically over-estimated mortality rates for those born in 1935-1945, consistent with the impact of the cohort effect. However, it was again evident that this over-estimation applied to a significantly earlier generation in the case of lung cancer than for the other causes.

**Figure 7 Ratio of “expected” to actual mortality rates derived using a Lee-Carter model fitted to mortality rates for lung cancer, heart disease and breast cancer for females, England & Wales, 1968-2003, averaged by year of birth**



It is worth considering some of the characteristics of the causes of death in relation to cigarette smoking.

A review paper by Lee (2000) concluded that the relative lung cancer risk among current smokers was 10 to 20 times that of those who have never smoked. Furthermore, it was found that it generally took ex-smokers 20-25 years after giving-up to reduce the additional risk by 75%.

On the other hand, a similar review paper on heart disease risk (Lee 2001) concluded that the average relative risk of current smokers to those who have never smoked was 212%. Furthermore, it took ex-smokers 5-9 years after quitting to reduce this additional risk by 75%.

Thus, it can be argued, historic patterns of smoking are much more likely to cause cohort effects in lung cancer than heart disease mortality.

There is also evidence that breast cancer mortality improvements have been faster for those born after 1925 than for those born before date. This pattern is unlikely to be due to changing patterns of smoking behaviour as smoking is not considered to be a major risk factor in breast cancer. A review paper by McPherson et al. (2000) made the statement that “*smoking is of no importance in the aetiology of breast cancer*”.

The observed cohort effect may be partly due to the fact that the NHS Screening Programme for breast cancer was initiated in 1988. This was aimed – initially – at women aged 50 to 65, so would have most benefited those born in the 1930s and 1940s. However, it is notable that improvements in breast cancer mortality were also relatively high (compared with other age groups) for women aged in their 30s in the 1970s and in their 40s in the 1980s.

It can therefore be argued that prevalence of cigarette smoking from one generation to the next has certainly been one factor which has driven the UK cohort effect and that, as a result, there is a degree of inevitability in some element of likely future improvement, especially for mortality at older ages from conditions strongly linked to smoking.

However, trends in heart disease and breast cancer mortality suggest that smoking may not be the only factor. In Willets (2004) it is argued that there appear to be two ‘sub-cohorts’ of the 1925-45 cohort: an earlier group where the improvements are largely due to smoking and a later one where other factors, such as diet in early life or exposure to infectious diseases, may have played a greater role.

The key point for this paper is that analysis of mortality trends by cause of death can play a vital part in determining the factors driving mortality trends,

such as the cohort effect. Furthermore it is argued that such an understanding allows trends to be appropriately allowed for in the projection of future mortality rates.

## 5. Implications and Conclusions

In section four it was argued that in order to understand trends and observed features in aggregate mortality, trends in individual causes of death need to be analysed.

This understanding is necessary because subjective judgments are always made when projecting future rates of mortality, no matter what method is selected. Even the most mechanical method applied to aggregate mortality rates requires decisions to be taken. Specifically, the precise structure of the model needs to be decided and the period of past data on which to base the future projection needs to be chosen.

In projecting future mortality rates for UK pensioners, it is necessary to form a view on (at least) the following points in deriving a suitable methodology:

- Should the model be parameterised using year of birth or attained age components (or both)?
- If year of birth parameters are used, should their effect reduce with time, increasing age or neither?
- Should the general pace of improvement continue at its current pace or be assumed to accelerate or decelerate over time? Should it revert to a long-term average rate and, if so, what time period should be used to calculate that average?
- Should the pace of improvement for males and females converge over time? If so, how long should the period of convergence last?
- Should the pace of improvement be assumed to be faster for (for example, higher socio-economic class) pensioners than for the average population? If so, how much faster and at which ages?

An understanding of the forces driving historic trends is an essential element of making decisions of this nature.

In fact, despite the historical experience of using cause-of-death projections and the well-documented difficulties, there nevertheless appears to be a good argument for utilising cause-of-death projections in making forecasts.

In forecasting mortality rates for those under the age of (say) 80 it can be instructive to divide deaths into a small number of cause-groupings, perhaps those with very strong historic trends, and compare the results with equivalent aggregate projections.

Cause-of-death modelling can also be a good methodology to test “extreme scenarios”, which are becoming of increasing interest to insurance regulators and capital markets. Such an approach tends to be welcomed by users of such projections, who can see the methodology “grounded in reality.” It can also provide a suitable mechanism for allowing for expert medical opinion in different diseases.

## References

- Caselli G. (1996). "Future longevity among the elderly." In: G. Caselli and A.D. Lopez (eds.) *Health and mortality among elderly populations*. Oxford: Clarendon Press.
- Continuous Mortality Investigation (2005). "Projecting future mortality: towards a proposal for a stochastic methodology. Working Paper No. 15. London: CMI.
- Continuous Mortality Investigation (2004). "Projecting future mortality: a discussion paper." Working Paper No. 3. London: CMI.
- Continuous Mortality Investigation (2002). "An interim basis for adjusting the '92 series' mortality projections for cohort effects." Working Paper No. 1. London: CMI.
- Continuous Mortality Investigation (1999). CMI Report 17. London: CMI.
- Government Actuary's Department (2003). "'2002-based' UK population projections." London: GAD.
- Government Actuary's Department (2001). "National population projections: review of the methodology for projecting mortality." London: GAD.
- Human Mortality Database, University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at <http://www.mortality.org> or <http://www.humanmortality.de> (data downloaded on 26 May 2005).
- Kelly M.P. and S. Capewell (2004). "Relative contributions of changes in risk factors and treatment to the reduction in coronary heart disease mortality." Health Development Agency Briefing Paper. London.
- Lee P.N. (2001). "A review of the epidemiology of heart disease related to active smoking." See <http://www.pnlee.co.uk>.
- Lee P.N. (2000). "A review of the epidemiology of lung cancer related to active smoking." See <http://www.pnlee.co.uk>.
- Lee P.N., J.S. Fry and B.A. Forey (1990). "Trends in lung cancer, chronic obstructive lung disease and emphysema death rates for England & Wales, 1941-85, and their relation to trends in cigarette smoking." *Thorax*, 45, 657-665.



Lee R.D. and L. Carter (1992). “Modelling and forecasting US mortality.” *Journal of the American Statistical Association*, 87(419), 659-671.

McPherson K., C.M. Steel and J.M. Dixon (2000). “Breast cancer – epidemiology, risk factors and genetics.” *British Medical Journal*, 321, 624-628.

Office for National Statistics (2004). “Living in Britain; results from the General Household Survey.” London: HMSO.

Office for National Statistics (1997). “The health of adult Britain 1841-1994.” London: HMSO.

Tabeau E. (2001). “A review of demographic forecasting models for mortality.” In: E. Tabeau, A van den Berg Jeths and C. Heathcote (eds.) *Forecasting Mortality in Developed Countries: Insights from a statistical, demographic and epidemiological perspective*. European Studies of Population Vol. 9. Dordrecht: Kluwer Academic Publishers.

Unal B., J.A. Critchley and S. Capewell (2004). “Explaining the decline in coronary heart disease mortality in England & Wales between 1981 and 2000.” *Circulation*, 109, 1101-1107.

UK Actuarial Profession (2004). “Individual capital assessment.” Version 1.1. Guidance Note 46.

Willets R.C. (2004). “The cohort effect: insights and explanations.” *British Actuarial Journal*, 10(4), 833-877.

Willets R.C., A.P. Gallop, P.A. Leandro, J.L.C. Lu, A.S. Macdonald, K.A. Miller, S.J. Richards, N. Robjohns, J.P. Ryan and H.R. Waters (2004) “Longevity in the 21<sup>st</sup> century.” *British Actuarial Journal*, 10(4), 685-832.

Wilmoth J.R. (1997). “In search of limits.” In: K. Wachter and C. Finch (eds.) *Between Zeus and the Salmon: The biodemography of longevity*. Washington D.C: National Academy Press.



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This volume addresses the question of how information on changes in patterns in the cause of death can be used to improve mortality forecasting. Do these patterns contain information that help us in predicting mortality rates for the elderly over the next decades? When current diseases have been eliminated, what is waiting for us around the corner? These and similar questions are discussed in this volume.

**Stockholm Committee  
on Mortality Forecasting**

Professor Edward Palmer  
Professor Tommy Bengtsson  
Professor Juha M. Alho  
Professor Kaare Christensen  
Professor Nico Keilman  
Professor James W. Vaupel

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