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The long-term pattern of adult mortality and the highest attained age

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Summary. Recent new data on old age mortality point to a particular model for the way in which the probability of dying increases with age. The model is found to fit not only modern data but also some widely spaced historical data for the 19th and 17th centuries, and even some estimates for the early mediaeval period. The results show a pattern which calls for explanation. The model can also be used to predict a probability distribution for the highest age which will be attained in given circumstances. The results are relevant to the current debate about whether there is a fixed upper limit to the length of human life.

Keywords: Aging; Extreme values; Lifespan; Logistic model; Longevity; Mortality

1. Introduction

1.1. Background

At every age, there is a probability of dying within 12 months. From soon after 30 years of age this probability starts to rise by about 10% with each successive year of age. In round numbers, the probability of dying (for modern males) increases from 0.001 at age 30 to 0.1 at age 80 years. This inexorable increase follows quite closely the ‘law of mortality’ which was discovered by Gompertz (1825). By 80 years the rate of increase is noticeably slower and there is controversy about what happens at higher ages still. There are alternative models, which we shall discuss.

In the 19th century, the probabilities of dying at ages 30–80 years were much higher than they are today. However, until relatively recently it was widely believed that, although there had been spectacular improvements at lower ages, there had been little change in the probabilities of dying above 80 years of age. Many believed that, somewhere above 100 years old, there is a maximum lifespan which has remained unchanged since ancient times.

In a very influential paper, Fries (1980) expressed his belief as a physician that there is a natural limit to the length of human life, even in the absence of disease, and that the maximum lifespan does not change. However, the average length of life has risen dramatically since the mid-19th century. This combination of a rising average and a fixed upper limit means that the shape of the survival curve has become more rectangular. Fries thought that the expectation of life at birth can be expected to increase to an ‘ideal average life span’ of about 85 years. Chronic illnesses can be postponed by changes in life style. Adult vigour can be extended to higher ages, but still within the same maximum lifespan. As a result, morbidity will become compressed into a period of senescence before the end of life. A research strategy on aging should be fundamentally shifted, towards postponing chronic illness and maintaining vigour.

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These views were controversial from the beginning and there has been a lively debate which is still in progress. We have space to mention only a few references, to give the flavour.

Manton *et al.* (1991) said that, although traditionalists might not expect the average length of life to rise above 85 years, there were nevertheless reasons for thinking that, with healthier life styles and continuing medical progress and intervention, it might be possible to raise the expectation of life in the USA to 95 or even 100 years.

Olshansky *et al.* (1990, 1991) were more sceptical, arguing that it would require very large falls in death-rates at high ages to have much further effect on the expectation of life at birth. Longer life might often be attainable only at the cost of a prolonged period of frailty and dependence. More research was needed to improve the quality of life of the elderly by ameliorating non-fatal diseases.

Vaupel, quoted in Baringa (1991), challenged Fries. Swedish data showed that mortality rates at ages 85 years and over had been falling for 50 years. Data on the length of life of a million fruit-flies showed that their probability of dying starts by increasing with age but then levels off. There was no evidence of a biological limit to life. Gavrilov and Gavrilova (1991) reached the same conclusion independently.

1.2. *The maximum length of life*

If there is a virtually fixed upper limit to the length of human life, then further falls in death-rates will make the survival curve even more rectangular and deaths will eventually be compressed into a narrower band of ages. In contrast, if there is no fixed upper limit, or if a fixed limit exists but is far higher than the ages which have so far been attained, then there will be less rectangularization and less compression of the ages at death, but people may live even longer.

These different possible outcomes will be reflected in different patterns for the probabilities of dying above age 80 years. In principle there are three main possibilities. The first is that the probability of dying within 12 months may reach 1 at a finite age. There will then be a definite, fixed upper limit to the length of human life. Many people believe there to be such a fixed, definite limit. The French demographer Vincent (1951) thought that there were enormous odds against anyone exceeding the age of 110 years. Fries (1980) and the biologist Hayflick (1994) placed the upper limit at about 115 years, and indeed Hayflick used this supposed limit as a reason for rejecting various medical theories. Most recently the mathematical statisticians Aarsen and de Haan (1994) have inferred (though from very limited data) that there is an upper limit to life which lies, with 95% confidence, between 113 and 124 years.

The second possibility is that the probability of dying within 12 months does not reach 1 at a finite age, but nevertheless tends asymptotically to 1. In this case there is no definite, fixed upper limit, but as age increases the probability of further survival eventually becomes so tiny that for practical purposes it can be ignored. This is the scenario which is implied by the original law of Gompertz (1825) and also by the more recent models of Weibull (1951) and Heligman and Pollard (1980).

The third possibility is that the probability of dying within 12 months may continue to increase monotonically as age increases but may tend to a limit which is less than 1. This is what is implied by the 'logistic model', which was first applied to mortality by Perks (1932) and later, under different names and notations, by several others. In this model there is no definite, fixed upper limit to life and there is not even an age when the probability of further survival is negligible. However high the age, there will always be a substantial probability of

surviving for at least 1 more year. This model tends towards a Poisson process, so the survivors will gradually approach (but not quite reach) a state like that of radioactive atoms awaiting decay, with a half-life. Nevertheless, all the survivors will die in the end and there will be a probability distribution for the highest age which will be attained in a given group of survivors. This will depend on the absolute size of the group.

Each of these theories has its supporters among biologists, so there is at present no consensus in that quarter. These various models, if considered solely from a theoretical point of view without any reference to actual data, provide almost unlimited scope for argument about whether there is an upper limit to life.

1.3. *New data*

Previous studies of the force of mortality at high ages have been based on data for individual countries. More detailed analyses can be made when there are larger numbers, obtained by pooling the data for similar countries. In 1990 Peter Laslett set up the Cambridge Group project on the maximal length of life, and its members (Kannisto, Thatcher and Vaupel) assembled and computerized a new database. This is part of the Archive on Population Data on Aging, funded by the US National Institute on Aging and the Danish research councils. The database now contains all the available official statistics on deaths at ages 80 years and over in 30 countries since 1960, and in many cases earlier. The full data are currently held at the University of Odense and at the Max Planck Institute for Demographic Research at Rostock. Copies will shortly be placed in the Danish State Archive and published in other ways so that they will be available to both institutions and individual research workers on request.

Results from the archive are being published in a series of monographs (Kannisto, 1994, 1996; Jeune and Vaupel, 1995). The latest in the series is *The Force of Mortality at Ages 80 to 120* (Thatcher *et al.*, 1998). In this book, six possible models for mortality above age 80 years are fitted to each of eight data sets, each comprising pooled data for 13 industrialized countries. The data sets are for the overlapping periods 1960–1970, 1970–1980 and 1980–1990, and for the cohorts born in 1871–1880, for males and females separately. As an indication of size, the 13 countries include almost 40 million people who reached 80 years of age and over 120 000 who reached 100, during the period 1960–1990. They cover over 32 million deaths at ages 80 years and over in this period. The data used in these analyses, which provide one of the foundations for the present paper, are given in full in Thatcher *et al.* (1998).

At these high ages, the new data are found to be clearly closer to the logistic model than to the Gompertz, Weibull and Heligman and Pollard models. This finding is taken as the starting point for the present paper.

1.4. *Aims and outline of the present paper*

The first substantive question to be addressed is *why* mortality should follow the logistic model. We shall describe three relevant theories, namely the so-called ‘fixed frailty’ model (Beard, 1971; Vaupel *et al.*, 1979), the stochastic process model of Le Bras (1976) and a recent controversial theory based on genetics (Mueller and Rose, 1996).

Next, we shall investigate how well a three-parameter form of the logistic model fits data for the past as well as the present, and at all adult ages, not just high ages. For this, the model is fitted to some widely spaced historical life-tables, from Halley’s life-table onwards, and also to some even earlier estimates for the mediaeval period. The model fits these data rather well.

Moreover, the parameters reveal a long-term pattern of adult mortality which on one interpretation implies that the so-called ‘rate of aging’ has been remarkably stable over many centuries.

The fitted parameters can also be used to tackle two difficult problems. The first is to estimate the expectations of life at high ages in the historical periods on a uniform basis. The second is to estimate the probability distributions of the highest ages which one may reasonably suppose to have been attained in England from the Middle Ages onwards. We are here dealing with the highest ages which were actually attained in the circumstances of the time, not with the potential ages which might have been attained if the circumstances had been different.

As a final illustration, we show the implied trends over the centuries of the mode of the distribution of the highest attained ages in stationary populations of various sizes.

2. The logistic model

2.1. Notation

We shall use the terminology and notation which is traditional in this field. The probability that a person who has just reached age x will die within 12 months is denoted q_x , in the actuarial notation. The probability that he or she will die in the instant between age x and age $x+dx$ is $\mu_x dx$, where μ_x is known as the force of mortality. In the literature, this is sometimes described as the instantaneous death-rate, and it is also the same as the hazard rate in survival theory. It is sometimes written as $\mu(x)$ and sometimes described as the mortality function.

For adult mortality, the numerical values of μ_x can be estimated or derived in several ways, from age-specific death-rates, from the survival curves in life-tables or cohort data, or from the probability of dying within 12 months (q_x). Conversely, q_x can be derived from μ_x . The relevant formulae are given in Appendix A.

2.2. Statement of the model

The logistic model assumes that the force of mortality μ_x is a logistic function of the age x . The most general model of this type has four independent parameters and can be expressed in the form

$$\mu_x = \frac{\kappa z}{1+z} + \gamma, \quad (1)$$

where

$$z = \alpha \exp(\beta x) \quad (2a)$$

$$= \exp\{\beta(x - \phi)\}, \quad \phi = -\ln(\alpha)/\beta. \quad (2b)$$

However, for studying long-term historical trends and predicting highest attained ages, this fully general model is found to be less useful in practice than a simpler but more robust version, with only three parameters. When the general model (1) was fitted by Thatcher *et al.* (1998) to the new data for 13 industrialized countries, the fitted parameters showed that κ is near 1. This leads us to consider the three-parameter model

$$\mu_x = \frac{z}{1+z} + \gamma, \quad (3)$$

where z is defined as before. If we use equation (2a) the parameters are α , β and γ . If we use equation (2b) they are β , γ and ϕ . These two ways of defining the parameters are completely equivalent and each will be found to have its advantages.

Model (3) has two important properties. When z is small (as at ages up to 70 years or so) it implies that

$$\mu_x \approx \alpha \exp(\beta x) + \gamma, \quad (4)$$

which is the ‘law’ proposed by Makeham (1860), and this was found to be still useful on much more recent data from many countries by Gavrilov and Gavrilova (1991). This also includes as a special case the original law of Gompertz, namely

$$\mu_x \approx \alpha \exp(\beta x). \quad (5)$$

The second important property is that when γ is small compared with $z/(1+z)$, which was true at high ages even in the historical periods (and at almost all ages today, when γ is negligible), model (3) implies that

$$\text{logit}(\mu_x) \approx \ln(\alpha) + \beta x. \quad (6)$$

This is the approximate relationship for old age mortality which was first noted by Kannisto (1992), used independently by Himes *et al.* (1994) and since overwhelmingly confirmed as a good approximation by Thatcher *et al.* (1998). What is just as important for the present application is that model (6) is a robust model which has been found to give good results when fitted to data below age 100 years and then extrapolated to higher ages, which is exactly the operation that we shall require. The same advantage will therefore apply to model (3).

Accordingly, we shall adopt equation (3) as the preferred model for investigating the long-term historical trends of adult mortality.

2.3. Comparisons with other models

It is of interest to see how the logistic model (3) and the Gompertz model (5) compare with the two other contending models which were mentioned in Section 1. One of these is the model of Weibull (1951), namely

$$\mu_x = \alpha x^\beta. \quad (7)$$

The other is the law of mortality used for graduation by Heligman and Pollard (1980). This has three terms and eight parameters, but at high ages the first two terms can be neglected and the third can be written in the form

$$\text{logit}(q_x) = \alpha + \beta x, \quad (8)$$

where α and β are constants. We may note that, as $x \rightarrow \infty$, $q_x \rightarrow 1$ and hence $\text{logit}(q_x) \sim -\ln(1 - q_x)$. Also we have $-\ln(1 - q_x) \approx \mu(x + \frac{1}{2})$, from expression (23) in Appendix A. On substituting these approximations in equation (8) we see that in the Heligman and Pollard model the force of mortality tends asymptotically to the straight line $\mu_x = \alpha - \frac{1}{2}\beta + \beta x$. This can also be proved more formally.

When these four models are fitted to actual data, they are all relatively close to the data at the ages where most of the deaths are concentrated, and hence relatively close to each other. At higher ages, the four models necessarily diverge because they tend to different asymptotes (an exponential, a power function, a sloping straight line and a horizontal straight line).

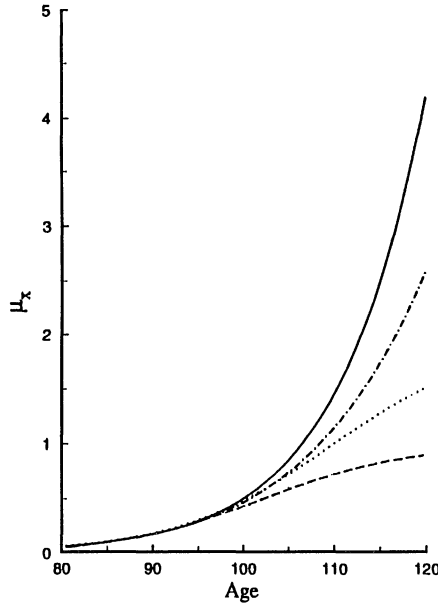


Fig. 1. Four models for the force of mortality: —, Gompertz; - - -, Weibull; · · · · ·, Heligman and Pollard; - · - · -, logistic

Fig. 1 illustrates the size of this divergence in a practical case, when the four models are fitted to the same data at ages 80–98 years and then extrapolated to 120 years. In this particular example the data were for the deaths of over 8 million females in 13 industrialized countries in 1980–1990, but this is immaterial. Whatever the data set, the fitted models always diverge.

As already stated, the new pooled data for 13 industrialized countries are close to the logistic model. If we use one of the other models, we shall overestimate the force of mortality at the highest ages and hence underestimate the highest age which will be attained in a population of given size.

2.4. Explanatory theories

2.4.1. The fixed frailty model

The first theory which was put forward to explain why μ_x should be a logistic function of age was the fixed frailty model, originally proposed by Beard in 1959 (see Beard (1971)). It is based on the assumption that the population is heterogeneous. The i th member of a birth cohort is subject to a personal hazard function which follows Makeham’s law

$$\mu_i(x) = \alpha_i \exp(\beta x) + \gamma, \tag{9}$$

where α_i (the frailty) varies from individual to individual but is fixed from (say) 30 years of age onwards. The constants β and γ are assumed to be the same for all individuals. The relative variation in the α_i between individuals is assumed to follow a gamma distribution with variance σ^2 . As the cohort becomes older, those with high frailty are more likely to die first and there is an effect which may be described as survival of the fittest. The result is that the mix of values of μ_i gradually changes. It can be shown that the average value of $\mu_i(x)$ among the survivors follows the general logistic model (1), with κ given by

$$\kappa = \beta/\sigma^2. \quad (10)$$

This model was later discovered independently by Vaupel *et al.* (1979). It is often called the ‘gamma–Makeham’ model.

2.4.2. *The Le Bras stochastic process*

Le Bras (1976) assumed that aging can be modelled by a stochastic process, in which individuals progress by jumps at random times through a succession of steadily deteriorating states. He considered a cohort which starts as homogeneous, with all the members in the same state. As they grow older, the members gradually become spread over many states and the cohort becomes increasingly heterogeneous. No assumption is made about how the force of mortality depends on age. Nevertheless, when the cohort reaches age x there will be an average force of mortality $\bar{\mu}(x)$ which, on certain assumptions, follows the logistic model (1).

A slightly generalized version of the Le Bras model was developed by Gavrilov and Gavrilova (1991), pages 247–250. In any interval of time dt , the individuals in the i th state will (in their notation) have a probability $(\mu_0 + i\mu) dt$ of dying and a probability $(\lambda_0 + i\lambda) dt$ of jumping to the $(i+1)$ th state, where μ_0 , μ , λ_0 and λ are constant parameters. It can be shown that, if this model is to fit the data, a modern person would have to pass through about 100 000 states before becoming a centenarian. Also μ_0 must be small compared with λ_0 , μ must be small compared with λ and the parameters in equation (1) will satisfy

$$\begin{aligned} \beta &\approx \lambda, \\ \kappa &\approx \lambda_0. \end{aligned} \quad (11)$$

Thus κ will depend on the initial rate of transition between states from the homogeneous opening start, whereas β will depend on how fast this rate of transition changes between successive states.

2.4.3. *Biological theories of aging*

There is an enormous literature on biological theories of aging and here we can mention only those points which are known to the author and which seem particularly relevant to the models of mortality discussed in this paper.

For life to continue, the internal workings of the body have to be kept in balance (homeostasis). This is achieved by compensating mechanisms in the heart, lungs, kidneys, liver etc. If the balance is disturbed, the body draws on its reserves of capacity to restore homeostasis. In young adults, the functional capacity of the organs is 4–10 times that needed to sustain life. After about 30 years of age, however, the capacity of the organs starts to decline steadily. Measurements of vital capacity, maximum heart rate, maximum oxygen consumption, the rate of cell renewal, etc., all decline with age, and with them the ability to restore homeostasis.

Why does the reserve capacity decline with age? According to evolutionary theory, the body must spend some of its resources on the immediate needs of survival (e.g. the ability to find food and to avoid predators), some on reproduction and some on the maintenance and repair of its bodily functions as they suffer damage. This damage can arise either from external causes, or from wear and tear or from the accumulation of DNA mutations and damage at the molecular and cellular level. (Whenever a cell divides its DNA molecules must be copied, and on each occasion there is a very small chance of damage.) However, the more the resources which must be spent on immediate survival and reproduction, the less are the

resources which are available for repair and maintenance. A balance must be struck between the two.

This balance is struck by natural selection. If a species is to survive, it is imperative that enough of its members should live sufficiently long to reach the age of reproduction. What happens after that does not matter very much, from the point of view of evolution. Accordingly, when we pass the age of reproduction, we are left with a body with a repair mechanism which was primarily developed to reach the age of reproduction, not to live beyond it.

In fact, the deterioration in reserve capacity starts after the beginning of the reproductive period but well before its end. Two processes then come into play. In the first, genes which have positive effects on fitness early in life can sometimes have negative effects on survival late in life (antagonistic pleiotropy). In the second, detrimental mutations which act only late in life are not efficiently eliminated by selection and can accumulate (mutation accumulation). Medawar (1952) described the post-reproductive period as a 'genetic dustbin'. Both these processes are made possible by the decline in the force of selection with age. The physiological and biochemical manifestations of aging have evolved as the result of a reduced investment in maintenance and repair brought about by the two effects mentioned (Williams, 1966; Kirkwood, 1981). The highly condensed summary given in this paragraph is taken from Stearns (1992), p. 182.

These processes are believed to account for the Gompertz-type increase in the force of mortality after the age of reproduction. However, there are now some biological reasons for believing that this deterioration may not continue indefinitely. Extensive work on fruit-flies has shown that their force of mortality increases sharply with age but then reaches a plateau, or even shows a fall (Carey *et al.*, 1992; Curtsinger *et al.*, 1992). Geneticists have now produced some mathematical models to explain how such a plateau can arise, from particular combinations of the effects of selection, accumulated mutation and antagonistic pleiotropy (Mueller and Rose, 1996). One of the findings of their study is that it is possible for the plateau to be reached when the probability of dying q_x is less than 1, which implies that the force of mortality can tend to a finite limit. This theory is controversial and will no doubt receive critical examination. If their arguments (or an amended version) command support and can be applied to humans, they would supply a biological reason for the upper limit for μ_x in the logistic model.

2.4.4. *Comments*

No doubt all the theories outlined in Sections 2.4.1–2.4.3 contain elements of truth. The population *is* heterogeneous. Genes *are* important. Also, although responses to life-threatening events and bodily developments will depend on genetic make-up, the actual incidence and timing of the events may resemble a stochastic process. However, none of these theories seems fully satisfactory as it stands and there is scope for further research on the reasons why the logistic model fits the mortality data.

An even more difficult question is why the data should be fitted by the particular model (3), i.e., to put the question another way, why is the parameter κ in the more general logistic model (1) apparently quite close to 1, when age is measured in years? Equations (10) and (11) suggest that possible relevant factors include the degree of heterogeneity in the population and/or the pattern of transitions between states, whereas the biological theories suggest that it depends on the balance between different types of genes. In the present paper, we shall simply regard $\kappa = 1$ as a convenient working hypothesis.

3. Long-term trends

3.1. Historical data

In this section, we shall be concerned with trends in mortality (at ages 30 years and over) over very long periods indeed. The simplest way to obtain a picture of the main changes which have taken place over the centuries is to examine a selection of mortality data at very widely spaced dates. For this, the most convenient sources of data are published life-tables.

We shall begin with *English Life Tables No. 14*, which relate to England and Wales in the period 1980–1982. These tables were prepared by the Government Actuary and published by the Office of Population Censuses and Surveys (1987). They are based on deaths in the three years 1980–1982 combined with population estimates derived from the 1981 census of population. In the three years 1980–1982 there were about 834 000 deaths of males and about 828 000 deaths of females at ages 30 years and over.

We now jump back over 140 years to *English Life Table No. 1*. This was compiled by William Farr from the 1841 census and the then newly established civil registrations of births and deaths in England and Wales. It was published by the Registrar General (1843). The table was based on the deaths in England and Wales in the single year 1841, during which there were about 71 000 deaths of males and about 74 000 deaths of females at ages 30 years and over.

Another 150 years back and we have Halley's life-table, based on the ages at death in Breslau in 1687–1691. This has been extensively discussed by, among others, Pearson (1978) and Hald (1990). Pearson concluded that Halley's life-table provided the best of the available estimates of mortality for this historical period. It is estimated, from the information quoted by Hald, that there were about 2675 deaths at ages 30 years and over in Breslau in the five years 1687–1691.

We now come to a very recent development. Wrigley *et al.* (1997) have published a new life-table for the period 1640–1689, compiled by family reconstitution from the entries in parish registers for married couples who were born and married in 26 parishes in England. Their database included 3133 deaths at ages 30 years and over, which is slightly more than Halley's. On comparing their results with his, we find that they are remarkably close. This is a very encouraging agreement on the force of mortality in the second half of the 17th century.

Before the 16th and 17th centuries no life-tables are generally accepted as reliable, except for small groups like monks. For the late mediaeval period (the 13th–15th centuries) there are some life-tables constructed by Russell (1948) from the information which was recorded when feudal tenancies changed hands.

For the early mediaeval period, a life-table was constructed by Acsadi and Nemeskeri (1970), table 130, from ages at death estimated from forensic examinations of 2300 adult skeletons in Hungarian cemeteries of the 10th–12th centuries. These cannot be regarded as very reliable, because a recent study has shown that the forensic methods used by Acsadi and Nemeskeri tended to overestimate adult ages below 60 years and to underestimate ages above 60 years (Molleson and Cox, 1993). However, this does not mean that the life-table cannot be used. If the method underestimated high ages, we can at least expect that the highest ages attained in the mediaeval period will have been higher than those predicted from Acsadi and Nemeskeri's life-table.

Acsadi and Nemeskeri (1970) also constructed life-tables for the Roman era, based on inscriptions on tombstones, as distinct from the examination of skeletons, but we shall not make use of these. Doubts have been expressed about the accuracy of the ages and about the representativeness of those who were given tombstones. Also, the resulting estimates of the

Table 1. Historical estimates of the force of mortality compared with values fitted by the logistic model (3)†

<i>Age group (years)</i>	<i>Observed force of mortality</i>	<i>Fitted force of mortality</i>	<i>Relative error (%)</i>	<i>Observed force of mortality</i>	<i>Fitted force of mortality</i>	<i>Relative error (%)</i>
<i>A, Hungary, 10th–12th centuries</i>						
30–35	0.0235	0.0232	–1.2			
35–40	0.0291	0.0281	–3.6			
40–45	0.0337	0.0351	4.2			
45–50	0.0402	0.0453	12.6			
50–55	0.0696	0.0598	–14.1			
55–60	0.0814	0.0804	–1.3			
60–65	0.1033	0.1090	5.5			
65–70	0.1485	0.1481	–0.3			
70–75	0.1877	0.1996	6.4			
75–80	0.3008	0.2651	–11.9			
80–85		0.3442				
85–90		0.4341				
90–95		0.5295				
95–100		0.6237	—			
			–0.4			
<i>B, England, 1640–1689</i>				<i>C, Halley, Breslau, 1687–1691</i>		
30–35	0.0171	0.0171	0.2	0.0164	0.0183	11.8
35–40	0.0205	0.0188	–8.2	0.0195	0.0198	1.4
40–45	0.0195	0.0214	9.6	0.0233	0.0220	–5.6
45–50	0.0244	0.0253	3.7	0.0282	0.0255	–9.7
50–55	0.0307	0.0313	1.9	0.0342	0.0308	–9.9
55–60	0.0459	0.0403	–12.1	0.0383	0.0390	1.9
60–65	0.0513	0.0540	5.3	0.0474	0.0516	8.8
65–70	0.0701	0.0743	6.0	0.0630	0.0705	11.9
70–75	0.1129	0.1039	–7.9	0.0995	0.0986	–0.9
75–80	0.1445	0.1461	1.1	0.1589	0.1392	–12.4
80–85	0.1974	0.2040	3.3		0.1958	
85–90		0.2794			0.2708	
90–95		0.3716			0.3638	
95–100		0.4757	—		0.4700	—
			–0.3			–0.3

(continued)

force of mortality do not look very credible: at ages 30–40 years they are in line with the early mediaeval estimates but at ages 70–90 years they are below the modern estimates.

For examining the very-long-term trends in the force of mortality at ages 30 years and over, we shall use the ‘snapshots’ provided by the seven sets of data in the life-tables described above. We shall treat males and females separately where this is possible, i.e. in 1980–1982 and 1841. For each of these seven data sets, the average force of mortality over 5-year age groups has been derived by the method described in Appendix A. The results are given in the columns headed ‘Observed’ in Table 1, and they are plotted in Fig. 2. Observed values are not given for the very highest age groups, where the life-tables are constructed by approximate methods.

Many important features of the long-term changes in the force of mortality can be seen from Fig. 2. Starting from the top, line A gives the estimates for the 10th–12th centuries, which of course are the least reliable of our data. However, no-one will be surprised to find that the estimated force of mortality in these centuries was higher than in the 17th century (lines B and C).

Table 1 (continued)

Age group (years)	Observed force of mortality	Fitted force of mortality	Relative error (%)	Observed force of mortality	Fitted force of mortality	Relative error (%)
<i>D, England and Wales, 1841, males</i>				<i>E, England and Wales, 1841, females</i>		
30–35	0.0108	0.0110	2.2	0.0107	0.0107	0.1
35–40	0.0123	0.0119	–3.2	0.0118	0.0114	–3.5
40–45	0.0140	0.0133	–4.7	0.0131	0.0125	–4.4
45–50	0.0159	0.0157	–1.2	0.0145	0.0145	–0.3
50–55	0.0181	0.0196	8.4	0.0162	0.0177	9.2
55–60	0.0254	0.0260	2.4	0.0220	0.0231	5.0
60–65	0.0375	0.0364	–2.9	0.0331	0.0321	–2.9
65–70	0.0553	0.0532	–3.8	0.0493	0.0470	–4.6
70–75	0.0815	0.0797	–2.2	0.0736	0.0712	–3.3
75–80	0.1201	0.1205	0.4	0.1097	0.1094	–0.3
80–85	0.1771	0.1807	2.1	0.1638	0.1674	2.2
85–90	0.2617	0.2643	1.0	0.2448	0.2502	2.2
90–95	0.3884	0.3710	–4.5	0.3674	0.3583	–2.5
95–100		0.4933			0.4847	
			–0.5			–0.2
<i>F, England and Wales, 1980–1982, males</i>				<i>G, England and Wales, 1980–1982, females</i>		
30–35	0.0010	0.0007	–23.0	0.0006	0.0007	21.2
35–40	0.0014	0.0015	8.4	0.0009	0.0010	10.4
40–45	0.0024	0.0027	15.4	0.0016	0.0016	–1.2
45–50	0.0043	0.0047	8.9	0.0028	0.0025	–10.4
50–55	0.0079	0.0080	0.8	0.0047	0.0040	–13.8
55–60	0.0138	0.0133	–3.5	0.0076	0.0067	–11.2
60–65	0.0227	0.0220	–3.3	0.0119	0.0114	–4.5
65–70	0.0365	0.0358	–1.9	0.0187	0.0193	3.0
70–75	0.0587	0.0577	–1.7	0.0308	0.0326	5.8
75–80	0.0930	0.0916	–1.5	0.0527	0.0548	3.9
80–85	0.1432	0.1422	–0.7	0.0919	0.0908	–1.2
85–90	0.2110	0.2141	1.5	0.1567	0.1469	–6.3
90–95	0.2900	0.3092	6.6	0.2374	0.2290	–3.5
95–100	0.3894	0.4236	8.8	0.3215	0.3390	5.4
			1.1			–0.2

†Average values of μ_x in 5-year age groups.

The fall in the force of mortality between the 17th century (lines B and C) and 1841 (lines D and E) is quite clear, at least up to 80 years of age. However, this fall is small compared with the large relative changes between 1841 (lines D and E) and 1980–1982 (lines F and G). By far the most important single reason for this change was the virtual conquest of infectious diseases, following improvements in living conditions, water-supplies, sanitation, hygiene, vaccination and later medical improvements. This period also shows the emergence of the sex ratio of mortality, which appears as the gap between lines F and G. There is, of course, a vast amount of detailed information available about the changes in mortality since 1841, including the changes in causes of death, which has been admirably summarized by the Office for National Statistics (1997).

3.2. Fitting the model

Figs 2–9 give a very clear picture of how the force of mortality has changed over the centuries and how well the logistic model (3) fits.

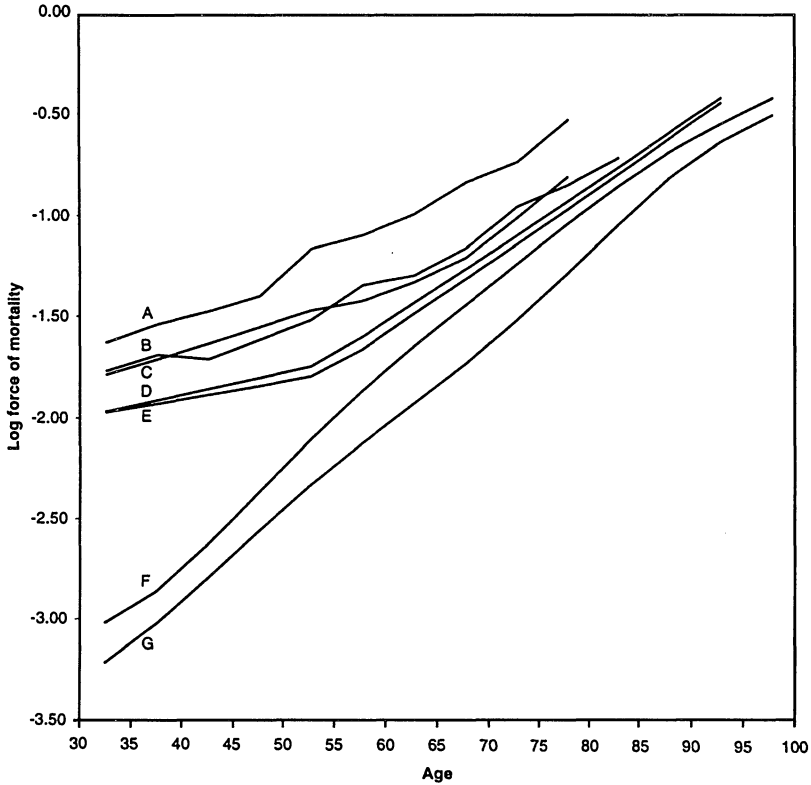


Fig. 2. Observed values of the force of mortality: A, Hungary, 10th–12th centuries; B, England, 1640–1689; C, Halley, Breslau, 1687–1691; D, England and Wales, 1841, males; E, England and Wales, 1841, females; F, England and Wales, 1980–1982, males; G, England and Wales, 1980–1982, females

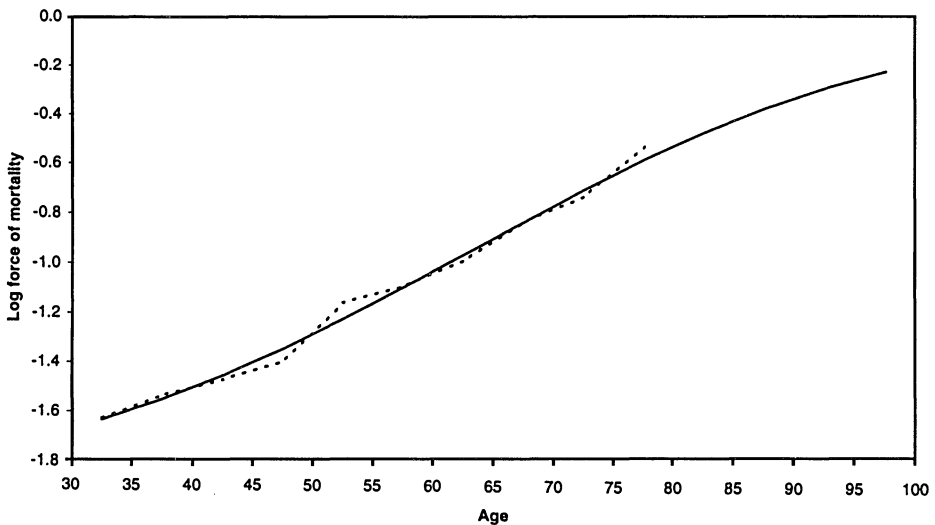


Fig. 3. Force of mortality in Hungary, 10th–12th centuries: , observed values; ——— , fitted logistic model

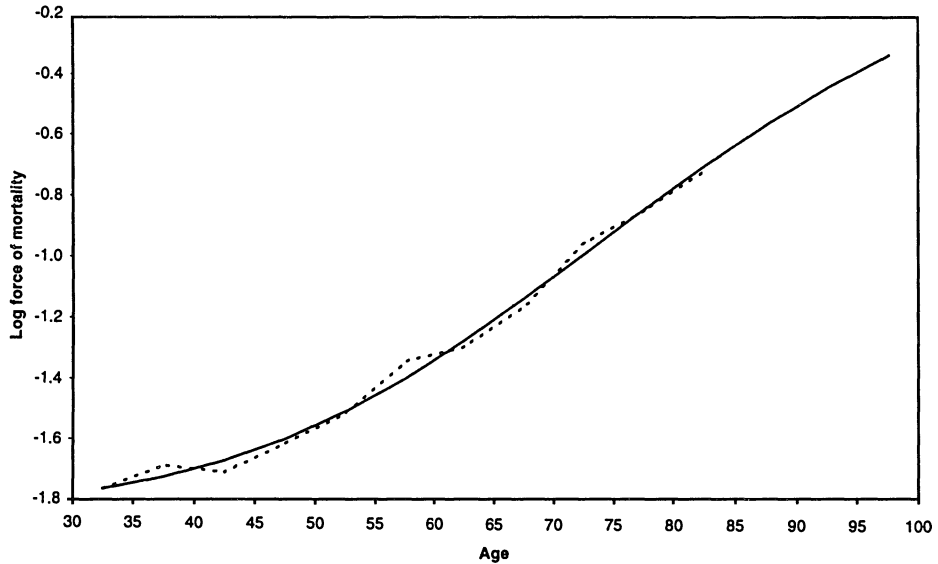


Fig. 4. Force of mortality in England, 1640–1689: , observed values; ———, fitted logistic model

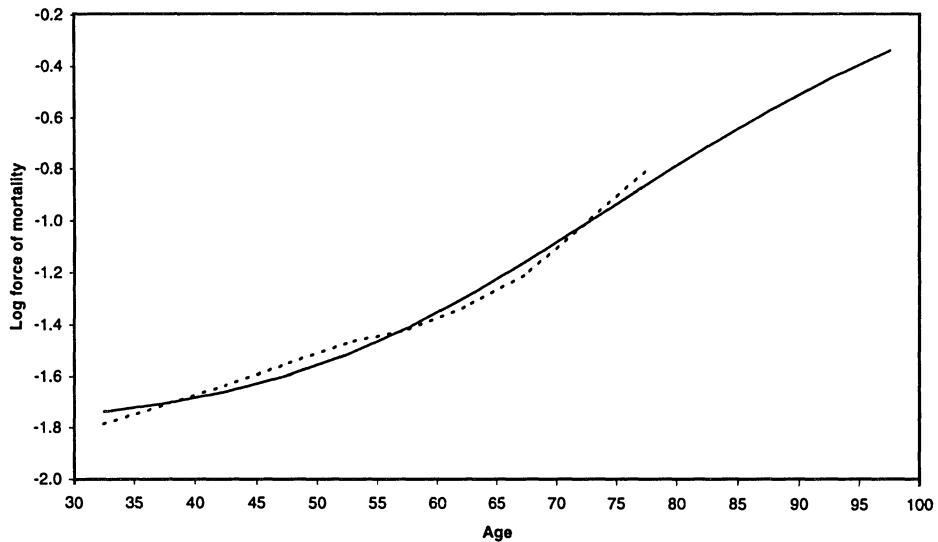


Fig. 5. Force of mortality in Breslau, 1687–1691: , observed values; ———, fitted logistic model

The model was fitted by the method of maximum likelihood, as described in Appendix B. The data which were used are given in full in the columns headed 'Observed' in Table 1; these were derived by the author directly from the life-tables as published in the references cited in Section 3.1, using equation (21) in Appendix A. The fitted parameters of the models are given in Table 2.

The published life-tables give estimates of q_x , and functions which can be derived from q_x . These are sufficient to estimate the parameters, but not to estimate their standard errors. For

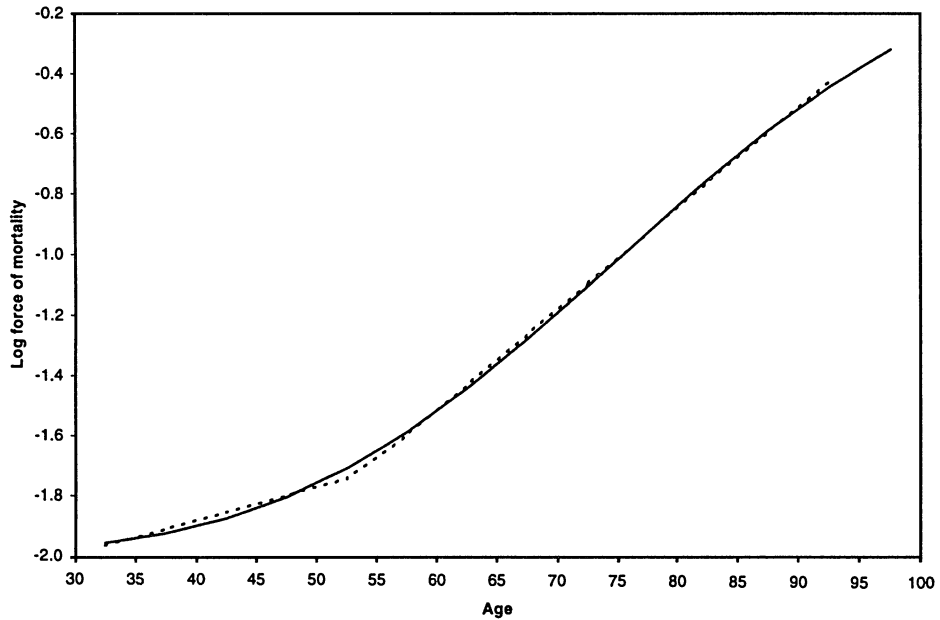


Fig. 6. Force of mortality in England and Wales, 1841, males: , observed values; ——— , fitted logistic model

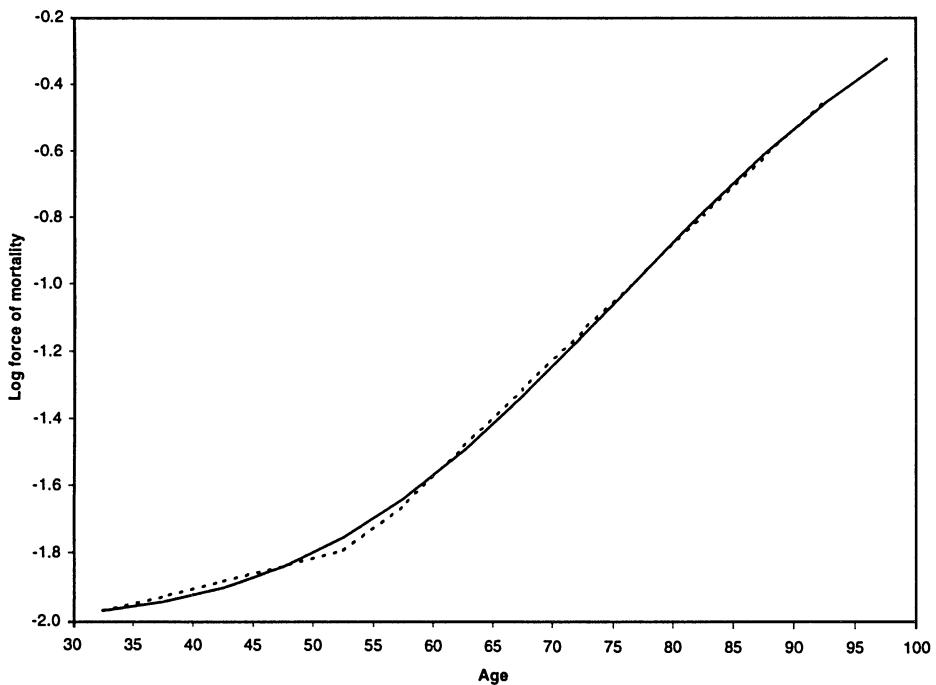


Fig. 7. Force of mortality in England and Wales, 1841, females: , observed values; ——— , fitted logistic model

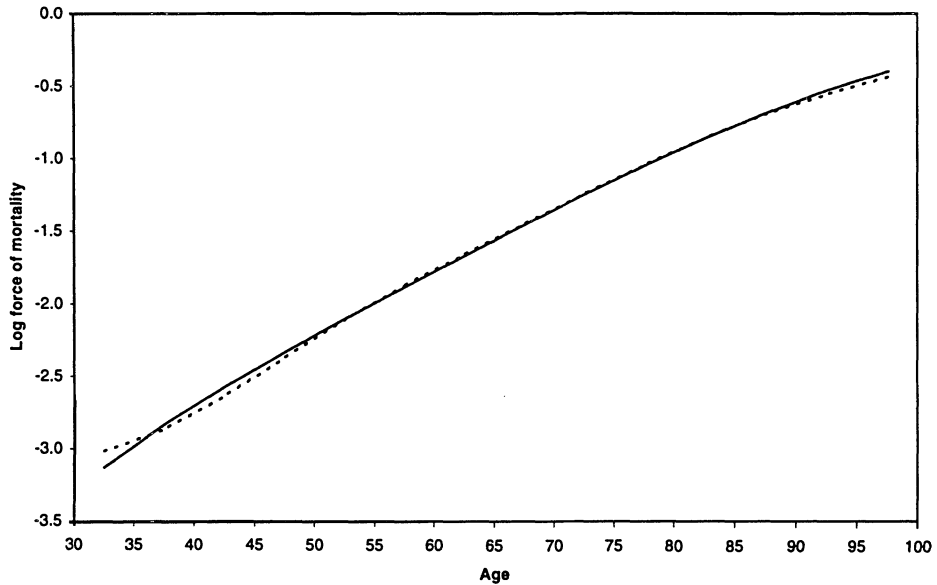


Fig. 8. Force of mortality in England and Wales, 1980–1982, males: , observed values; ———, fitted logistic model

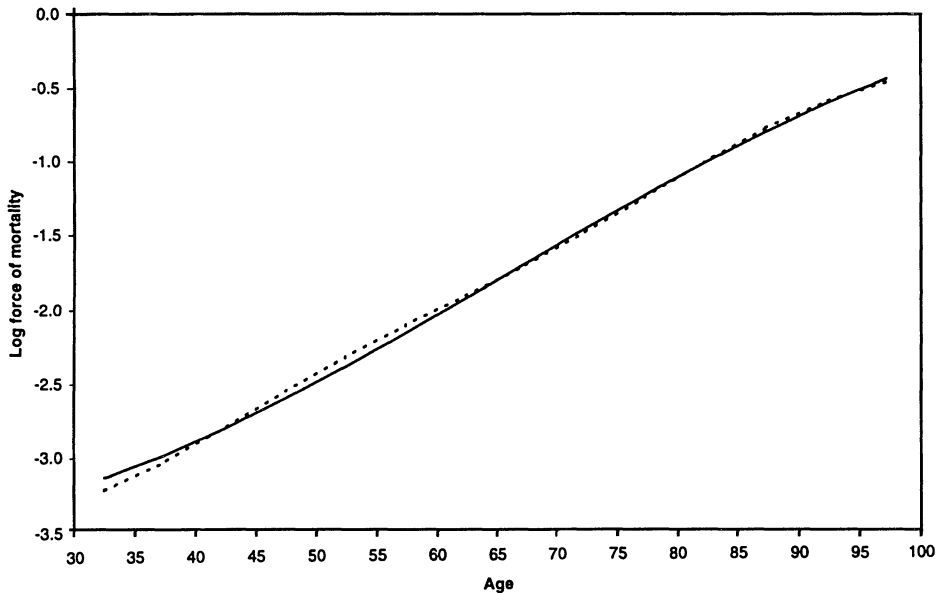


Fig. 9. Force of mortality in England and Wales, 1980–1982, females: , observed values; ———, fitted logistic model

these, we need more detailed information than is available about the absolute numbers who were at risk in the data from which the q_x were estimated. Accordingly, some simulated data on the numbers at risk were constructed by the method described in Appendix B and used to calculate the estimated standard errors in Table 2.

No single measure of goodness of fit is always appropriate. However, Table 1 shows in

Table 2. Parameters of the fitted logistic models†

<i>Data set</i>	<i>Deaths at ages 30 years and above</i>	<i>Values of the following parameters in model (3):</i>			
		$10^4\alpha$	β	γ	ϕ
A, Hungary, mediaeval	2300	8.7378 (3.6103)	0.0769 (0.0062)	0.0127 (0.0031)	91.63 (2.11)
B, England, 1640–1689	3133	1.8749 (0.5973)	0.0864 (0.0043)	0.0140 (0.0012)	99.28 (1.38)
C, Halley, Breslau, 1687–1691	2675	1.4397 (0.5218)	0.0888 (0.0049)	0.0158 (0.0012)	99.56 (1.50)
D, England and Wales, 1841, males	71000	0.5035 (0.0299)	0.1008 (0.0008)	0.0097 (0.0001)	98.15 (0.19)
E, England and Wales, 1841, females	74000	0.3231 (0.0190)	0.1050 (0.0008)	0.0097 (0.0001)	98.45 (0.18)
F, England and Wales, 1980–1982, males	834000	0.4657 (0.0048)	0.0992 (0.0001)	−0.0004 (0.0000)	100.59 (0.04)
G, England and Wales, 1980–1982, females	828000	0.1208 (0.0013)	0.1093 (0.0001)	0.0003 (0.0000)	103.62 (0.03)

†The parameters were fitted to the data in the second column of Table 1. The standard errors, in parentheses, use estimated data on numbers at risk (see Appendix B).

detail the relative differences between the observed and fitted values, described conventionally as ‘errors’. When these are large, they provide warnings that care must be taken.

In fact, the very largest of these relative errors (at ages 30–35 years in 1980–1982, but with opposite signs for males and females) are only quite so large because in these cases the absolute values of μ_x are very small. The other relative errors are not sufficiently large to disturb the very broad conclusions which we shall draw later in this section. For the later calculations in Section 4, about the highest attained age, it is not the relative errors but the cumulative sum of the absolute errors which is relevant.

3.3. *The long-term pattern*

Long-term changes in the pattern of mortality show up as trends in the parameters of the model. We can see from Table 2 that the parameter α has fallen dramatically. The parameter β , although rising, has only changed from 0.08 to 0.11 over this very long period. The parameter γ has become negligible. The parameter ϕ has increased. We need to consider the meaning of these parameters and the reasons for the changes. It is convenient to start with γ .

3.3.1. *The parameter γ*

When z is small we have $\mu_x \approx \gamma$, so γ will be close to the force of mortality at young adult ages. In fact, Tables 1 and 2 show that γ is almost as large as the average value of μ_x at ages 30–35 years. The observed fall in γ therefore reflects the virtual elimination of the causes from which people used to die at ages 30–35 years in the past, which overwhelmingly were infectious diseases and causes associated with childbirth.

3.3.2. *The parameter β*

The relative rate at which the force of mortality increases with age corresponds to the slope of the lines in Fig. 2. This relative rate of increase with age is sometimes used as a measure of a hypothetical ‘rate of aging’. In the Gompertz model (5) we have

$$\frac{1}{\mu} \frac{d\mu}{dx} = \beta, \quad (12)$$

so the relative rate of increase is the same at all ages. However, in the logistic model (3) things are more complicated. Even in the simple case where $\gamma = 0$, we find that

$$\frac{1}{\mu} \frac{d\mu}{dx} = \beta(1 - \mu). \quad (13)$$

Thus the relative rate of increase is close to β at young ages, where μ_x is small, but then it gradually slackens as age increases. By the very high age where $\mu_x = \frac{1}{2}$, the relative rate of increase has fallen to $\frac{1}{2}\beta$.

It is the parameter β which ultimately governs the relative rate at which the force of mortality increases with age. Table 2 shows that its values were about 0.10 for males and 0.11 for females in 1980–1982, still between 0.10 and 0.11 in 1841, about 0.09 for each of the independent estimates for the 17th century and (if the data can be believed) about 0.08 in the 10th–12th centuries. This is a remarkable stability over such a very long period, which calls for explanation. It would obviously be desirable for this finding to be checked, if further historical data can be analysed or collected, but at first sight these results appear to support the views of those who believe that there is an underlying pattern of aging, genetically determined.

3.3.3. The parameter ϕ

When $x = \phi$ in equations (2) and (3) we have $z = 1$ and hence $\mu_x = \frac{1}{2} + \gamma$, which is close to 0.5. Thus ϕ is simply the age at which the force of mortality reaches 0.5 (and hence the probability of dying q_x reaches about 0.4). It is also the age at which the logistic curve (3) has its point of inflection.

The value of ϕ on row A of Table 2 will be too low, because the forensic method underestimated high ages. However, this problem does not affect rows B, C, D and E, which all show estimates between 98 and 100. This means that these four logistic curves all pass quite close to the point where $\mu_x = \frac{1}{2}$ at 99 years. It is also related to a feature of the cohort mortality rates for England and Wales from 1841 to 1981, which also show a close approach to convergence at very high ages (Charlton (1997), p. 26). Our interpretation of these findings is that there was a very long period when the force of mortality above 90 years of age changed very little, despite the changes at lower ages. However, this long static period came to an end in the 1950s, when mortality rates at very high ages started to fall, even (slightly) at ages over 100. This has occurred not only in England and Wales (Thatcher, 1992) but also in most other industrialized countries (Kannisto, 1994; Kannisto *et al.*, 1994).

3.3.4. The parameter α

The force of mortality is dominated at low ages (30–35 years) by the parameter γ and at very high ages (90 years and over) by the parameter ϕ . In the middle range of ages, the parameter α is dominant. As an illustration, equation (2b) shows that $\alpha = z^2$ when $x = \phi/2$. In a modern example, where $\phi/2 \approx 50$ and γ is small, it then follows from model (3) that $\mu_{50} \approx \sqrt{\alpha}$. The force of mortality at 50 years of age therefore depends primarily on α (or vice versa).

There is an important relationship between α , β and ϕ . The definition of ϕ in equation (2b) can be rewritten as

Table 3. Expectation of life calculated from the logistic model (3)

Age (years)	Expectation of life at the following mortality levels:					
	Hungary 10th– 12th centuries	England 1640–1689	England and Wales			
			1841, males	1841, females	1980–1982, males	1980–1982, females
30	21.1	27.6	33.1	34.3	42.8	48.4
40	15.7	22.1	26.6	27.7	33.3	38.8
50	11.0	16.6	19.9	21.0	24.3	29.4
60	7.2	11.5	13.7	14.5	16.5	20.8
70	4.6	7.4	8.5	9.0	10.1	13.3
80	2.9	4.5	4.8	5.1	5.7	7.6
90	2.0	2.7	2.7	2.8	3.2	4.0
100	1.5	1.8	1.7	1.7	1.9	2.2
110	1.3	1.4	1.3	1.3	1.4	1.5

$$\beta = -\ln(\alpha)/\phi. \quad (14)$$

In the period when ϕ hardly changed but α was falling, we can see from equation (14) that β would necessarily rise. Thus there would be a negative correlation between α and β . The size of the falls in mortality in middle age could also explain the apparent slight increase in β over the centuries.

3.3.5. The expectation of life

The fitted parameters can be used to produce some new estimates of the past expectations of life at ages 30 years and over, given in Table 3. Their distinctive feature is that all the expectations are calculated on a comparable basis. It is also possible to extend the estimates to the very highest ages, and this has been done for theoretical interest. The results show that, whereas the improvements in the expectation of life at ages 30–50 years have been measured in decades, by age 80 they are measured only in years and by age 100 only in months.

Because Halley's life-table and the new life-table for England in 1640–1689 are almost indistinguishable, only the latter is shown separately. We have already noted that the estimates for the mediaeval period are less reliable than the others. As the forensic methods used by Acsadi and Nemeskeri (1970) are now believed to have underestimated ages over 60 years, the estimates of mediaeval expectations of life at ages 60 years and over are probably too low.

4. The highest attained age

4.1. Concept and methods

4.1.1. Distribution of the highest attained age

Consider a cohort of individuals, all born in the same year. Suppose that N members of this cohort survive to reach age x_0 . When all these N members have died, their N ages at death will have a highest value ω_N . This is the highest age which was attained by the members of this cohort.

It is important to note that ω_N is the highest age which was attained by the members of the cohort in the circumstances in which they actually lived. It is not the potential highest age

which might have been attained if the circumstances had been different. ω_N is an observable quantity and a proper subject for statistical study.

The observed value ω_N can be regarded as the extreme value of a sample of size N , drawn from the distribution of the ages at death. Thus ω_N will itself have a probability distribution, which can be derived from N and the mortality or hazard function $\mu(x)$. The theory of such extreme values goes back to Fisher and Tippett in the 1920s and it was specifically applied to human mortality by Gumbel (1937).

Let $s(x)$ be the expected number of members of the cohort who will survive to reach age x , starting from the initial value $s(x_0) = N$. It is a standard result that

$$s(x) = N \exp \left\{ - \int_{x_0}^x \mu(t) dt \right\}. \quad (15)$$

Now the probability that a randomly chosen member of the cohort will have a length of life which exceeds x is $s(x)/N$. Also ω_N will be less than x if all the N members of the cohort die before reaching age x and the probability of this is

$$\begin{aligned} \Pr(\omega_N < x) &= \{1 - s(x)/N\}^N \\ &\approx \exp\{-s(x)\} \quad \text{when } N \text{ is large.} \end{aligned} \quad (16)$$

The two equations (15) and (16) give the (almost) exact distribution function for ω_N .

By integrating equation (3) and substituting in equation (15) we obtain

$$s(x) = N \exp[\gamma(x_0 - x) + \beta^{-1} \ln\{1 + \alpha \exp(\beta x_0)\} - \beta^{-1} \ln\{1 + \alpha \exp(\beta x)\}]. \quad (17)$$

From equations (16) and (17) we can calculate the 'exact' probabilities for each completed year of age. This is the method which was used to construct Tables 4 and 5.

The probability distribution of ω_N has a mode, usually denoted by $\tilde{\omega}_N$. At least for models in which $s(x)$ and its first derivative tend to 0 as $x \rightarrow \infty$, and when N is large, it can be shown that $\tilde{\omega}_N$ is close to the value of x which satisfies

$$s(x) = 1. \quad (18)$$

This is not surprising, since at the highest age there is only one survivor.

We are here dealing with the classical problem considered by Gumbel, in which there is a known model for the distribution of ages at death. In more recent developments of extreme value theory, no assumption is made about a model. Instead, observed measurements of extreme values (such as floods) are fitted directly to theoretical distributions for extreme values. However, this modern approach cannot be used when, as for mortality in the historical periods, we do not have any proper reliable data for the extreme ages. In periods before the present century we have only isolated observations of very high ages, validated with great difficulty. There is therefore no alternative to using the classical approach. Later, we shall compare its predictions with some of the high ages which are known.

4.1.2. Sensitivity to errors

The value of ω_N depends on N , but it is not very sensitive to its exact value. In a typical situation, even doubling N will not increase ω_N by more than 1 or at most 2 years of age. (The reason for this is that at the highest attained age the probability of survival for 1 more year is generally about $\frac{1}{2}$. If two people reach age X , we expect that one of them will reach age $X + 1$. If four people reach age X , we expect that two will reach $X + 1$ and that one will reach $X + 2$.)

Thus doubling the size increases the expected highest age by 1 year.) In consequence, we do not need to have a highly accurate value for N to make predictions about ω_N .

Nor does the estimate of ω_N depend very critically on the choice of the age x_0 at which the calculation is started. In a test comparison it was found that it made hardly any difference whether the calculation was started at age $x_0 = 50$ or at age $x_0 = 90$ years. Nevertheless, in principle it is best to start from the highest age for which the size of the cohort is known, since this will minimize any errors which might result from errors in the mortality function $\mu(x)$.

4.1.3. *Methods for estimating sizes of cohorts*

In all except one of the historical cohorts to be considered later, proper estimates of the sizes of the cohorts at appropriate ages can be derived either from the censuses of population or from the population reconstitutions made by the Cambridge Group for the History of Population and Social Structure.

In the earliest cohort, before the censuses and parish registers, we can still make a rough estimate of the size of the cohort when it reached age 50 years. This happens to be possible because it appears to be an empirical fact that the number of people who were 50 years old at their last birthday is never very far from 1% of the total population, over an astonishingly wide range of circumstances. It was observed by Wilmoth (1995), analysing high mortality model life-tables with expectations of life at birth as low as 20–30 years, that the number of 50-year-old people was between 0.6% and 1.2% of the total population. At the other extreme, in a modern low mortality population, all the British censuses from 1931 to 1991 inclusive show that the number aged 50 years was between 1.0% and 1.5% of the total population, for both males and females. At the intermediate levels of mortality studied by the Cambridge Group, approximate estimates of the numbers aged 50 years are about 1.1% of the total population in 1651 and 1.0% in 1751. Thus as a rough rule of thumb, in the absence of better information, we can assume that the number aged 50 years is about 1% of the total population.

4.2. *Application to historical trends*

As an illustration of the methods described above, we shall now apply them to some historical cohorts in England and Wales. These are the cohorts born in five particular calendar years, chosen largely for interest and the availability of data. The results are shown in Table 4, which is based on data drawn from many sources.

We first describe how Table 4 was constructed. The top row shows the earliest historical cohort for which it was possible to make a rough estimate of size. (Its members were aged 50 years in 1086, the year of the *Domesday Book*, when the population of England was 1.1 million. Following Section 4.1.3, the size of the cohort at 50 years of age was taken as 1% of this figure.) For the cohorts born in 1600 and 1700, the sizes at ages 70 and 80 years were derived from the Cambridge Group estimates of the population in 5-year age groups. The size of the 1811 cohort at age 80 years was similarly derived from the 1891 census. The size of the 1881 cohort at age 90 years is given directly in the 1971 census.

The predicted distribution function for the highest attained age was then calculated by the exact method, equations (15)–(17), using the nearest relevant parameters in Table 2. It is here assumed that the logistic model, having been fitted to the age groups shown in Table 1, will continue to hold for just a few age groups further.

We now examine the results in Table 4. On the first row, the predicted distribution implies that the highest attained ages in the cohorts which became extinct in the 12th century were generally in the 90s. This prediction is strengthened, rather than weakened, by the fact that

Table 4. Predicted distribution of the highest attained age in particular historical cohorts in England and Wales, compared with some known high ages†

Year cohort born in	Estimated size at age shown	Predicted distribution			Known high ages
		Mode	Percentiles		
			1%	99%	
1036	11000 at 50 years	92	88	99	
1600	23000 at 70 years	103	101	110	At least 97 in 1701
1700	27000 at 70 years	104	101	110	100 around 1800
1811, males	14500 at 80 years	105	102	111	103–107 in 1911–1920
1811, females	19000 at 80 years	105	103	111	103–108 in 1911–1920
1881, males	7800 at 90 years	109	106	115	106–112 in 1985–1994
1881, females	24500 at 90 years	112	110	118	109–115 in 1985–1994

†Highest attained ages in completed years.

Acasadi and Nemeskeri's (1970) mediaeval life-table was found by forensic methods which are now believed to have underestimated high ages. There were certainly mediaeval people who were believed by their contemporaries to be over 90 years old but it is difficult to validate individual cases by modern methods. We must also remember that only one person out of each annual birth cohort reaches the highest attained age for that cohort, so such cases will have been exceedingly rare and almost impossible to identify and verify before the days of compulsory registrations of births and deaths.

The predicted distributions for the cohorts born in 1600 and 1700 imply that the highest attained ages in 1700 and 1800 were probably above 100 years of age. This is an interesting calculation. Hynes (1995) found several possible centenarians who died in England between 1700 and 1800, but the evidence was not conclusive. The observed value in Table 4 is conservative; it shows only the death at age 97 years of Sir John Holland (1603–1701), whose documentation is described as indisputable. There is a presumption that the attained highest age around 1800 was higher than this, perhaps at least 100 years by 1800 or soon after.

For the cohorts born in 1811 and 1881 we are on much firmer ground. The observed high ages are from the official death registrations from 1911 onwards, and their accuracy has been discussed by Thatcher (1992) and by Wilmoth and Lundström (1996). Ages of 110 years and over are included only when they have been verified by tracing the original birth registration. In each of the 10 calendar years 1911–1920 (and 1985–1994) there was a highest age at death, and the last column in Table 4 shows the range of these 10 highest ages. The predicted 1% and 99% percentiles are for the highest age in a single year, not for the range over 10 years.

4.3. Application to stationary populations

The method can be applied to a much wider range of population sizes than has been experienced in England and Wales. Purely for illustration, some results are shown in Table 5 for stationary populations. The calculations are easy in this case, because the cohort sizes and the mortality function are constant from year to year, and the age distribution is stable and consistent with them. For simplicity, Table 5 shows just the total population size. The cohort sizes at age 50 years were found by the 1% approximation in Section 4.1.3, assuming also that at age 50 years the numbers of men and women are equal.

The illustrative results in Table 5 show the mode of the distribution of the highest attained age in a randomly chosen year. It would, of course, be possible to calculate also the distri-

Table 5. Mode of the distribution of the highest attained age in a stationary population in a random year†

<i>Total population (males + females)</i>	<i>Ages (years) at the level of mortality in the following:</i>					
	<i>Hungary, 10th–12th centuries</i>	<i>England, 1640–1689</i>	<i>England and Wales</i>			
			<i>1841, males</i>	<i>1841, females</i>	<i>1980–1982, males</i>	<i>1980–1982, females</i>
10000	81	90	90	91	93	98
100000	87	96	96	97	99	103
1000000	92	100	100	101	103	107
10000000	96	104	104	105	107	111
100000000	99	108	107	108	110	114
1000000000	103	111	110	111	113	117

†Ages in completed years.

bution of the highest age which would be attained in a longer period, such as a decade or a century, but for the present purpose it is sufficient to consider a random year. It would also be possible to calculate the percentiles as well as the mode, but these all move together and the mode is sufficient to show the trends.

Table 5 shows that the transition from mediaeval to modern mortality increases the highest attained age by about 14–17 years. In addition, the difference between a population of size 10000 and a population of 1000 million can add a further 20–22 years to the highest attained age.

The boldest step in Table 5 is the extension of the mediaeval estimates to very large populations, implying ages which are far higher than those observed in the original data. However, some unexpected reassurance can be gleaned from Zhao (1995). He found reliable records of a few deaths at ages 96–102 years in the 13th–17th centuries in China, when the population was over 100 million. At least, the predicted ages in Table 5 are not totally without support.

5. Discussion

It is perhaps not surprising that a logistic model of the form (3) should be found to fit recent data on adult mortality, since it is very close to two other models which are already known to fit (namely Makeham's law at ages 30–70 years and Kannisto's logit model at ages 80 years and over). What is more surprising is that this model should fit, rather well, not only the recent data but also estimates of the force of mortality derived from historical life-tables for the 19th and 17th centuries, and some even earlier estimates for the mediaeval period. Moreover, the parameter which governs the relative rate at which the force of mortality increases with age (the so-called rate of aging) appears to have changed very little over many centuries, though it has shown a slight tendency to increase.

These findings clearly call for explanations. Section 2.4 describes some published theories which may perhaps help to explain why adult mortality should follow a logistic model, but these require critical examination. Perhaps further theories will be forthcoming, both on processes which can generate logistic models and also on the rate of aging.

The long running search for a law of mortality has recently been reviewed by Olshansky and Carnes (1997). In several approaches, deaths are analysed by cause and an attempt is

made to distinguish between intrinsic and extrinsic causes (or, in another terminology, between endogenous and exogenous causes). However, such methods cannot be applied to historical data and in any case the findings in the present paper do not seem to require them. It is the 'all-cause' mortality which is found to follow the logistic model.

The model, once fitted, can be used to make estimates of how the expectation of life at high ages, and the highest attained age, may reasonably be supposed to have evolved in England over the centuries. The results include the quite specific predictions that ages over 90 years were attained in the mediaeval period and that the age of 100 years was probably attained by the end of the 17th century.

We now turn from the past to the future. Although the method in Section 4 enables us to predict the probability distribution of the highest age which will be attained, given the mortality function, it does not produce a prediction for the mortality function itself. Thus the method does not answer one of the major controversial questions of the moment, about how much further mortality rates at high ages are likely to fall within the next few decades. The investigation, however, provides evidence which is relevant to another controversial question, about whether there is a fixed upper limit to the length of human life.

The view that there is a definite, fixed upper limit to the length of human life is very widely held, particularly by many biologists and gerontologists. However, it has not gone unchallenged. Gavrilov and Gavrilova (1991) commented that the idea of a fixed limit, unchanged over time, has become established by a process of 'mutual citation' rather than by convincing proof. It is therefore worthwhile to review the arguments.

If a definite fixed limit exists, call it X . Values of X are of three kinds. The first kind consists of values like $X = 110$ and $X = 115$ which have been seriously proposed or assumed, e.g. by Vincent (1951), Fries (1980) and Hayflick (1994), but which we now know, in 1998, *have already been exceeded*. We have the direct evidence of the case of Mme Jeanne Calment (born 1875, died 1997) who reached the age of 122 years. The documents authenticating her birth and life have been carefully verified by French scientists and the details have been published (Allard *et al.*, 1994).

The second kind of X is somewhere above 122 years of age, but still sufficiently close to have a visible effect on the shape of the distribution of ages at death. If X exists, then this distribution will come to an abrupt end at age X and μ_x at this age will be infinite. Even before age X , we would expect the observed values of μ_x to start to rise with age faster than the expected values given by the Gompertz model. In fact, the new data for the industrialized countries show that they are falling below the Gompertz model. Moreover, the age reached by Mme Calment was even higher than would be expected from the logistic model, let alone the Gompertz model (Thatcher *et al.*, 1998). There is no visible evidence here that the observed ages at death have been restricted by the effect of a fixed upper limit X .

The third kind of X is one that exists, but at so high an age that it has no visible effect on our observations. This is a hypothesis which can neither be proved nor disproved. It is also indistinguishable, statistically, from the hypothesis that there is no fixed limit.

Of course, the idea that there may be no definite limit to life is not new. The alternative to a fixed upper limit is not that immortality is possible but simply the more mundane hypothesis that there will continue to be a probability distribution for the highest age which will be attained in given circumstances, i.e. with a given mortality function and a given size of population, and which can be calculated by the method used in this paper. Since the population cannot rise indefinitely and the mortality function cannot fall indefinitely, there must be some high age which is unlikely ever to be exceeded, but it is not predetermined and it is not fixed and definite.

It is perhaps worth observing that even if there is no strict limit the length of human life will still be governed by an interaction between biology and circumstances. It is biology which determines the probability that people of a given age will die in given circumstances. Seen from this point of view, Fig. 2 illustrates how these circumstances have changed over the centuries.

Acknowledgements

This work would not have been possible without the meetings and workshops on old age mortality arranged by Peter Laslett and James Vaupel, and the new data assembled by Väinö Kannisto. I am very grateful to all three, for many reasons, and to the referees for their critical and constructive comments. I am much indebted to Trevor Sweeting for fitting the logistic model (3) to the data from the historical life-tables and for producing Figs 2–9. I am indebted also to Kirill Andreev for calculating the standard errors in Table 2. The paper was written as part of the project on maximal length of life of the Cambridge Group for the History of Population and Social Structure.

Appendix A: Formulae and approximations for the force of mortality

If $\mu(x)$ is the force of mortality at age x , then the probability that a randomly chosen individual who has just reached age x will die before reaching age $x + dx$ is $\mu(x) dx$. The force of mortality is also known as the hazard rate and as the instantaneous death-rate.

Let $s(x_0)$ be the number of members of a given cohort who reach age x_0 , and let $s(x)$ be the expected number who will survive to reach age x , where $x > x_0$. Then

$$\frac{s(x)}{s(x_0)} = \exp \left\{ \int_{x_0}^x -\mu(x) dx \right\} \tag{19}$$

$$\mu(x) = -\frac{1}{s} \frac{ds}{dx}. \tag{20}$$

Now let $\bar{\mu}(x_0, x)$ be the average value of $\mu(x)$ between ages x_0 and x . Then

$$\bar{\mu}(x_0, x) = \frac{\ln\{s(x_0)/s(x)\}}{x - x_0}. \tag{21}$$

This is the formula which was used to derive the ‘observed’ values in Table 1 from the published life-tables, identifying $s(x)$ with l_x in life-table notation.

The force of mortality at age $x + \frac{1}{2}$ is very close to the average force of mortality between ages x and $x + 1$. Thus from equation (21) we have

$$\mu(x + \frac{1}{2}) \approx \ln\{s(x)/s(x + 1)\} \tag{22}$$

$$\approx -\ln(1 - q_x) \tag{23}$$

where q_x in life-table notation is the probability of dying within 12 months of reaching age x . When q_x is known exactly, model (23) gives a very close approximation for the force of mortality. Even in extreme cases, when the force of mortality increases exponentially by 10% with each year of age, the numerical error in expression (23) is only 0.04%.

In a large population, $\mu(x)$ is also close to the age-specific death-rate found by taking the annual number of deaths between ages $x - \frac{1}{2}$ and $x + \frac{1}{2}$, and dividing this by the average population between these ages. Hence $\mu(x + \frac{1}{2})$ is close to the age-specific death-rate based on deaths between exact age x and exact age $x + 1$, which is m_x in actuarial notation. Hence

$$\mu(x + \frac{1}{2}) \approx m_x. \tag{24}$$

This approximation is given by Pollard (1973). In the same extreme case as before, the numerical error in expression (24) does not exceed 0.8%.

The approximations in this appendix apply to adult mortality but not to infants, because the pattern of deaths is very non-uniform in the first year of life.

Throughout the paper, age is measured in years. Thus $\mu(x)$ and $\bar{\mu}(x_0, x)$ measure the force of mortality in units of deaths per person-year.

Appendix B: Methods of fitting the model

The modern data described in Section 1.3 show the estimated numbers at risk as well as the deaths. If N_x people reach age x during a given period, of whom D_x die within 12 months, then we can regard D_x as the outcome of N_x binomial trials, each with probability q_x as given by the model. The likelihood of the observed data set is the product of all the relevant binomials. The parameters of the model can then be estimated by maximizing the likelihood. This method can be applied whenever we know N_x and D_x , whether the population is stationary or non-stationary.

The historical life-tables present a different problem, because they do not show the absolute numbers at risk. The life-tables, on their own, simply show the behaviour of a hypothetical stationary population which has probabilities of dying q_x , as estimated in various ways by the compilers of the life-tables. From the life-table we can derive the implied relative frequency f_i of observed deaths in the i th age group. We also have the probability p_i that a death will fall in this age group, according to the model. Now consider a hypothetical cohort of arbitrary size N drawn from this hypothetical population. We assume that the ages at death in the cohort will follow the life-table, so that the observed number of deaths in the i th age group will be Nf_i . The probability that the model values p_i will produce this observed result is then given by the appropriate term in a multinomial distribution. The resulting log-likelihood for the observed result is $\sum Nf_i \ln(p_i)$. The parameters which maximize this log-likelihood are the same for all N . These 'best estimates' of the parameters, calculated in this way by Trevor Sweeting and given in Table 2, are all that we need to construct Tables 3–5.

Because the best estimates from the life-tables do not depend on N , and are not based on known numbers at risk, they have no associated standard errors. However, the earlier historical life-tables are based on rather limited numbers of observations (of deaths in Breslau etc.). If we had been fitting the parameters to the original data, instead of to the life-tables, the estimates of the parameters might have had substantial standard errors. Unfortunately, we cannot find this out directly, because in many cases the original data are not available in the form required. It has, however, been possible to make estimates of N^* , the total number of deaths at ages 30 years and over which were included in the data on which the life-tables were based. These are shown in the second column of Table 2.

Now consider a cohort which starts with size N^* at age 30 years and has subsequent deaths which follow the values of q_x given either by the life-table or by the fitted model. We can calculate the standard errors for parameters fitted to such a cohort. Of course, this is not how the life-tables were actually constructed, but at least the method gives an order of magnitude for those errors which are unavoidable, owing to stochastic variations in the ages at death in a limited number of observations. Standard errors estimated in this way are shown in Table 2.

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Discussion on the paper by Thatcher

M. Murphy (*London School of Economics and Political Science*)

This paper is at the interface of demography, statistics and biology that is producing a wealth of new insights (Wachter and Finch, 1997). As examples, Mr Thatcher's paper helps to demolish the idea that there is a fixed human 'lifespan' (or biologically determined maximum lifetime), and he shows that mortality at the oldest ages has been improving steadily in developed countries, a topic of debate in earlier decades. This paper is an example of statistics as a core part of the scientific process, rather than a technique that is useful for other disciplines. Statisticians rightly concentrate on the estimation of population aggregates, such as means, and give little attention to extreme values. However, in the case of human longevity, both public interest and scientific insight come from the analysis of one individual out of the 6 billion on this planet.

This paper shows that we can be confident that the number of older people will increase substantially in decades to come, a trend that is sometimes referred to rather unfortunately as the 'demographic time-bomb'. I would like to concentrate on some of the implications for both the elderly and the overall populations arising from this paper's analysis, starting with forecasts of the elderly population. Current British official projections (Government Actuary's Department, 1998) suggest that the population aged 60 years and over will increase by 50% between 1996 and 2066, and there will be aging within the elderly population, so that of those aged 60 years and over the proportion who are aged 85 and over will increase from 9% to 14%. The projected number of centenarians increases 14-fold, from 6000 to 102000, even though this paper estimates that the first centenarian in England and Wales may not have been seen until the 18th century. Population projections, however, are subject to considerable uncertainty: in the USA, in 1994 there were 3.7 million people aged 85 years and over; by 2050 this is expected to increase to 18.2 million in the central variant projection, but with a range from 9.6 million to 31.1 million between the lowest and highest scenarios (US Bureau of the Census, 1996). Even these changes are modest compared with what may occur in developing countries.

In the past, official forecasts in all countries have consistently underestimated improvements in mortality at older ages (Olshansky, 1988; Murphy, 1995). The parameterization for old age mortality in this paper provides a potentially valuable starting point for improved forecasts and assessment of forecasting error.

The prolonging of life is welcome, but a key issue is how many of these additional years of life are likely to be spent in the healthy rather than the unhealthy states (Robine and Ritchie, 1991). Establishing trends in the health status of older people is difficult (Grundy, 1997). In the 1970s and 1980s, it appeared that the additional years of life were mainly being spent in an unhealthy state, although often not of a very serious nature. More recently, there is evidence that the health status of older people has been improving in countries such as Canada, France and the USA. If US disability rates had remained at 1982 levels, there would have been an additional 1.2 million disabled people in the USA in 1994 (Manton *et al.*, 1997). Elucidating the interaction between trends in disability and mortality would appear to be a priority for future work since the numbers and health status of elderly people will have major implications for health care and long-term financing. Germany has introduced compulsory long-term care insurance, and in Britain expenditure on health and personal social services per person aged 85 years and over was £3995 compared with £263 per person aged 18–64 years in 1989–1990 (Central Health Monitoring Unit, 1992).

While people live longer, the average age at entry to the labour force has been increasing and people leave it at earlier ages. In 1971 15% of men aged 60–64 years were economically inactive, compared with 57% in 1996 (Office for National Statistics, 1998), a trend reflecting preferences by workers and/or employers, rather than government compulsion. Although the role of aging for problems in funding of pensions has been exaggerated, it continues to be important (Johnson, 1997).

Informal care of older people is usually undertaken by close family members and recent trends in developed countries with declines in marriage and increases in breakdowns of partnerships and voluntary childlessness are likely to reduce the pool of this key caring group in the long term, although not in the short term (Wolf, 1995; Wachter, 1997).

I am very pleased to propose the vote of thanks to this stimulating and fascinating paper by Mr Thatcher.

J. W. Vaupel (*Max Planck Institute for Demographic Research, Rostock*)

Roger Thatcher has presented a thoughtful, thought-provoking analysis of mortality at older ages.

His contribution is intelligent, judicious and stimulating, in the best tradition of English statistical thinking.

As Thatcher emphasizes, ‘the population *is* heterogeneous’. Frailty models provide a convenient way of roughly accounting for unobserved differences in susceptibility to death among individuals of the same age. Thatcher’s results, as well as research by Lee and Carter (1992) and Lee and Tuljapurkar (1994), and some earlier findings about the evolution of Swedish mortality (Vaupel *et al.*, 1979) suggest to me that the following frailty model may provide some useful insights into the dynamics of mortality over age and time:

$$\mu(x, y, z) = z \mu_0(x) \exp\{-r(x) \phi(y)\} + c \exp\{-\psi(y)\}, \quad (25)$$

where $\mu(x, y, z)$ is the force of mortality at age x and time y for individuals with fixed frailty z in some cohort, $\mu_0(x)$ is the base-line or standard force of mortality at age x , c is a positive constant, $\phi(y)$ and $\psi(y)$ are functions that determine how mortality is affected by altered conditions and $r(x)$ is a function that determines the relative effect of altered conditions on mortality at different ages. If z follows a gamma distribution with mean 1 at the starting age of observation x_0 , then

$$\bar{\mu}(x, y) = \frac{\mu_0(x) \exp\{-r(x) \phi(y)\}}{1 + \sigma^2(y-x) \int_{x_0}^x \mu_0(t) \exp\{-r(t) \phi(y-x+t)\} dt} + c \exp\{-\psi(y)\}, \quad (26)$$

where $\bar{\mu}(x, y)$ is the force of mortality at age x and time y and $\sigma^2(y-x)$ is the variance at age x_0 of the gamma distribution for the surviving cohort born at time $y-x$. This formula can also be written as

$$\bar{\mu}(x, y) = \mu_0(x) \exp\{-r(x) \phi(y)\} \bar{s}(x_0, x, y-x)^{\sigma^2(y-x)} + c \exp\{-\psi(y)\}, \quad (27)$$

where $\bar{s}(x_0, x, y-x)$ is the proportion of the cohort born at time $y-x$ that survived from age x_0 to age x . Other distributions for frailty z could be used, but the gamma distribution is convenient because it leads to the simple formulae (26) and (27).

‘Biology’, ‘circumstances’ and ‘heterogeneity’, three key factors in Thatcher’s analysis, are distinguished in formulae (25) and (26). Biology (in specified base-line circumstances) is captured by $\mu_0(x)$ and c , the effect of changing circumstances by $r(x) \phi(y)$ and $\psi(y)$, and heterogeneity by $\sigma^2(y-x)$. To identify the model some arbitrary restrictions must be imposed, such as setting $r(0)$ equal to 1 and $\phi(0)$ and $\psi(0)$ equal to 0. This model is surely oversimplistic and hence wrong, but it might lead to some insights about how biology, circumstances and heterogeneity have interacted to determine mortality over age and time. Thatcher’s logistic formula (3) is a special case of equation (26).

Models like equation (26) can be fitted to mortality surfaces over age and time. Such surfaces can be constructed from reliable data for the past century or longer for Denmark, England and Wales, Finland, France, Italy, the Netherlands, Norway and Sweden (Vaupel *et al.*, 1998). Thatcher’s results demonstrate that research on estimating models of mortality surfaces can lead to important findings. If data are available for 100 years of age and 150 years of time, then 15000 death-rates can be analysed. Values of $\phi(y)$, $\psi(y)$ and $\sigma^2(y-x)$ can be estimated for every year and $r(x)$ and $\mu_0(x)$ for every age. The mortality surface can then be decomposed, in a flexible way, into components pertaining to biology, circumstances and heterogeneity. Alternatively, functional forms might be specified for $\phi(y)$, $\psi(y)$, $\sigma^2(y-x)$, $r(x)$ and $\mu_0(x)$. The mortality surface can be analysed from birth or, following Thatcher, for adult ages above 30 years, where the Gompertz model might be a reasonable choice for $\mu_0(x)$. To capture general trends over age and time roughly the following six-parameter version of equation (27) might prove useful, at least at ages above 30—or perhaps 50—years and for restricted stretches of time:

$$\bar{\mu}(x, y) = a \exp\{bx - \phi y\} \bar{s}(x_0, x, y-x)^{\sigma^2} + c \exp(-\psi y). \quad (28)$$

Thatcher’s paper, in sum, is both interesting in itself and likely to encourage some interesting additional research. Consequently, I would like to second the vote of thanks and to congratulate the author on his stimulating insights.

The vote of thanks was passed by acclamation.

Väinö Kannisto (*Lisbon*)

One of the revelations of Mr Thatcher’s excellent paper is that mortality does not rise as high as has generally been thought. Projected to age 120 years, the probability of dying within 12 months would still be in the neighbourhood of 0.6. I made a search in the Odense database to see whether there are exceptions to this pattern. In 147 old age life-tables from 31 countries since 1950 there were about 2000 q -values at ages 100 years and over. Among them, q_x exceeded 0.6 by more than 1 standard error in only three instances, and in none by more than 1.5 standard errors.

I think that this fits neatly into the pattern described by Mr Thatcher in accordance with the logistic curve. There is no proof that the underlying probability of dying ever exceeded 0.6 in this material.

The same may also apply to high mortality countries where any given death-rate may be found at an age up to 10 years or even 15 years younger than in the West, leaving few survivors to very high ages. In contrast, the subsequent ascent of these high rates with advancing age is slower as is well illustrated in Fig. 2 of Mr Thatcher’s paper.

Sidney Rosenbaum (*Radlett*)

Over 40 years ago I was struck by a review by Kendall (1955) of Gumbel (1954) and I acquired a copy of the book. Although Gumbel’s main field of application was floods, he had an interesting example of the oldest ages in Switzerland which was closely modelled by the so-called double-exponential distribution whose cumulative density function is $\exp\{-\exp(-y)\}$. His estimate for the oldest age was based on the mode of the distribution of these ages, plus an add-on factor which was a function of their standard deviation (SD). Thus he argued that a large SD could more than compensate for a low mode, using the analogy of a stream with a large variation in its flows that could result in greater floods than a bigger, more placid river. Hence a country with a lower expectation of life might record a higher age at death. We shall explore this in the following indicative calculations. I have some difficulty with Gumbel’s notation and propose to use the mean rather than the mode, which avoids having to calculate the latter.

If x denotes the oldest age in each of N years, the standardized add-on is

$$\frac{\max - \bar{x}}{S_x} = \frac{\ln(N) - \bar{y}_N}{\sigma_N} \rightarrow \frac{\ln(N) - \gamma}{\pi/\sqrt{6}} \quad \text{for large } N$$

where \max is the maximum age in a period of N years and γ is Euler’s constant: for $N = 100$, as in the following example, the limiting value is quite accurate.

To start with England and Wales, suppose that the average of the oldest ages over the past century was 108 years, with an SD of 2. Then \max is found to be 114. This is for a population at mid-century of 40 million. For Uroland, with 10 times the population, i.e. 400 million, we must add a further $(S_x/\sigma_N) \ln(10)$, with $\max = 118$.

Suppose that the fabulous country of Chindostan has a population of 4000 million with a mean extreme age, over the same period, of only 98 years but with twice the SD of 4. Then in comparison with England and Wales the effect of the increase in SD alone yields $\max = 111$, with the addition for the size of the country of 15, totalling 126. Evidently a further increase in SD would result in even greater ages.

The effect of a population reduction can be similarly calculated; for instance, to get down to the number of Fellows of the Society we must subtract $(S_x/\sigma_N) \ln(10^4)$, say (however, the Society has existed for 164 years so the subtraction must be reduced slightly); I calculate that the maximum age would have been slightly under 100 years. To my knowledge, we have had one centenarian, Lt Col William Butler, who died in 1969 at 101 years of age.

John Charlton (*Office for National Statistics, London*)

Professor Murphy has eloquently said much of what I was intending to say, but it is perhaps still interesting to look at the figures that I have which illustrate some of the points.

The trends shown by Mr Thatcher are the result of both growth in the adult population in the preceding age groups ages and improvements in mortality at higher ages—there is a multiplicative effect (Fig. 10). If we turn to looking at mortality by birth cohort (Fig. 11) we see widening differences between successive birth cohorts, as mortality levels have decreased.

If the lines in the graph are projected forwards (though we have no certainty that improving trends will continue) then life expectancy would be rather longer than previously expected. Perhaps it is worth modelling this graph to consider different scenarios. Even with more conservative assumptions about

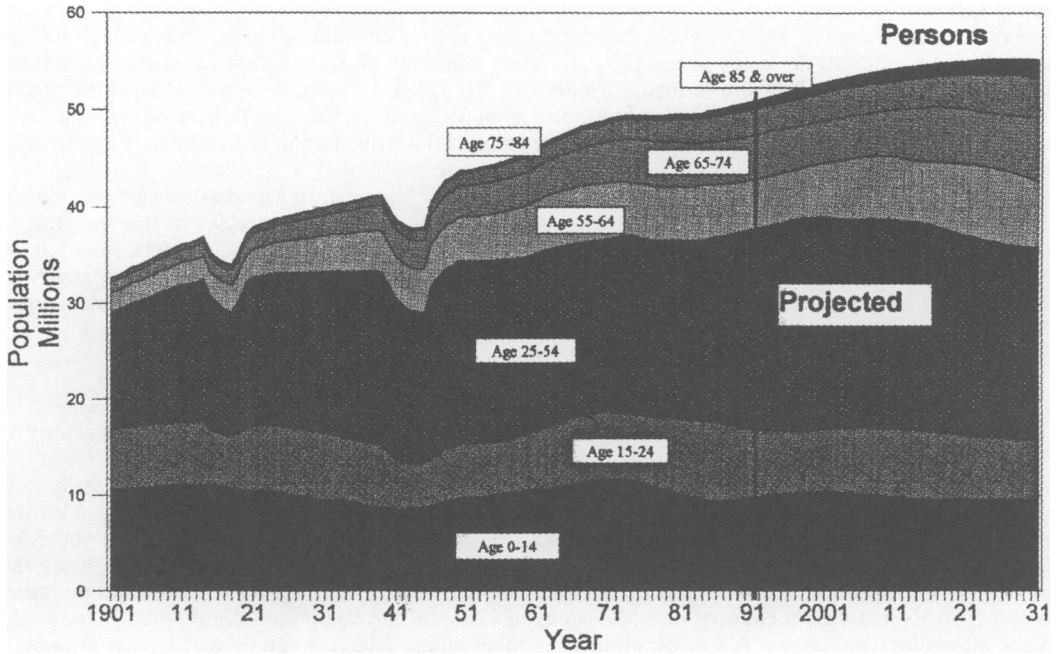


Fig. 10. Population of England and Wales, 1901–2031

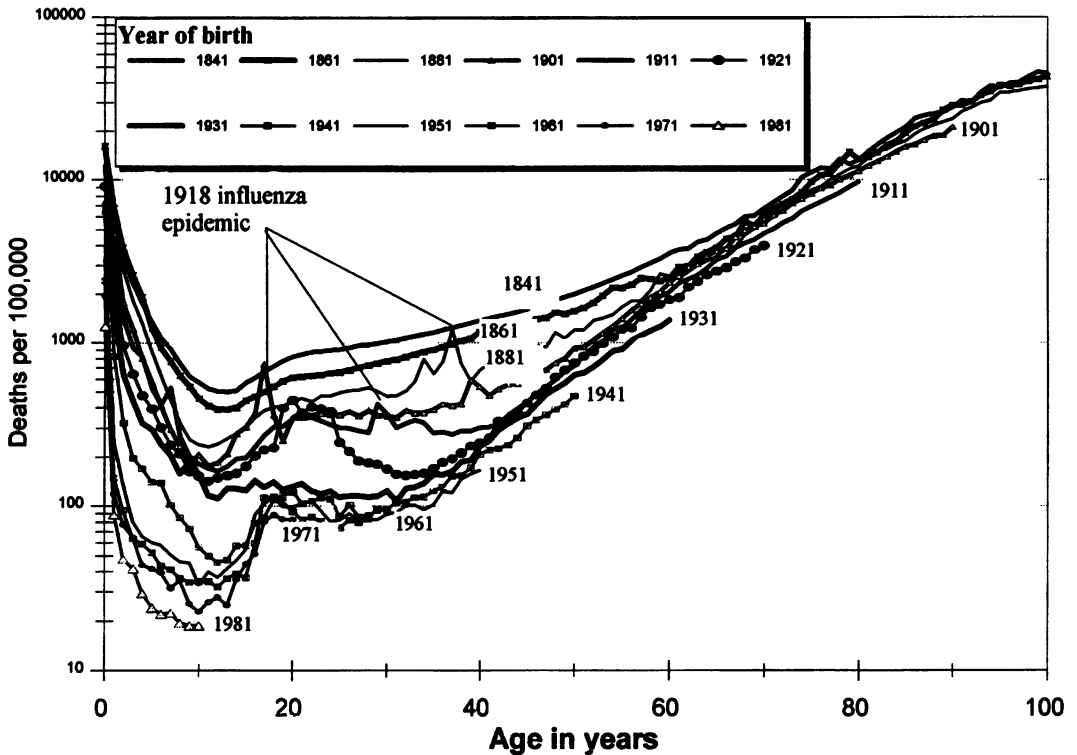


Fig. 11. Mortality by age and year of birth, male cohorts 1841–1981

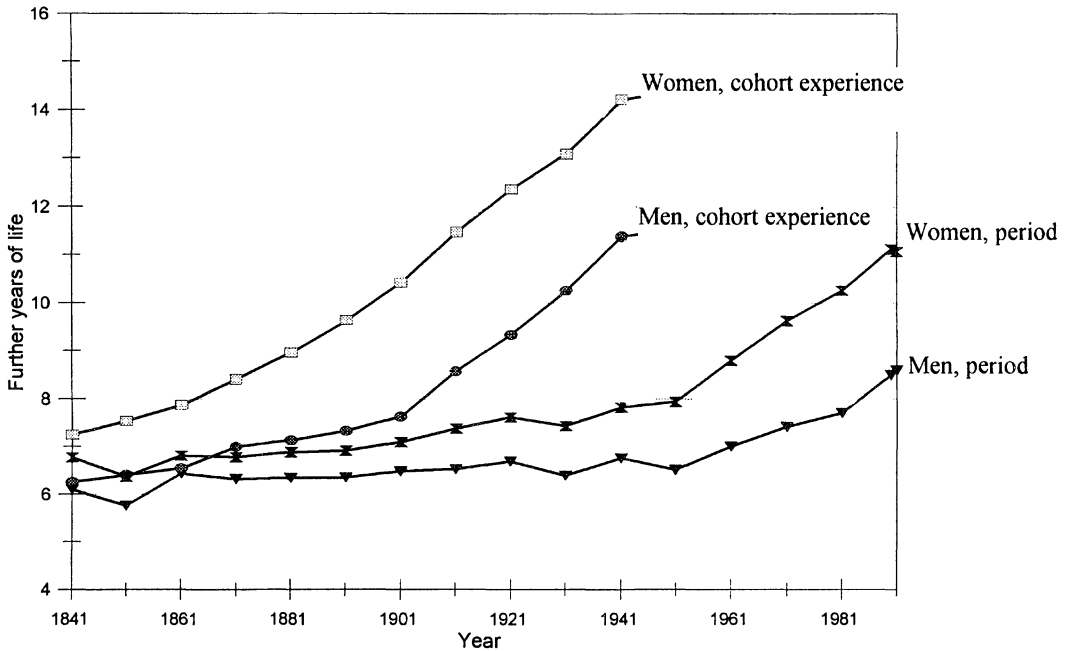


Fig. 12. Average life expectancy at age 75 years: cohort life expectancy based on Government Actuary's Department mortality projections for recent years

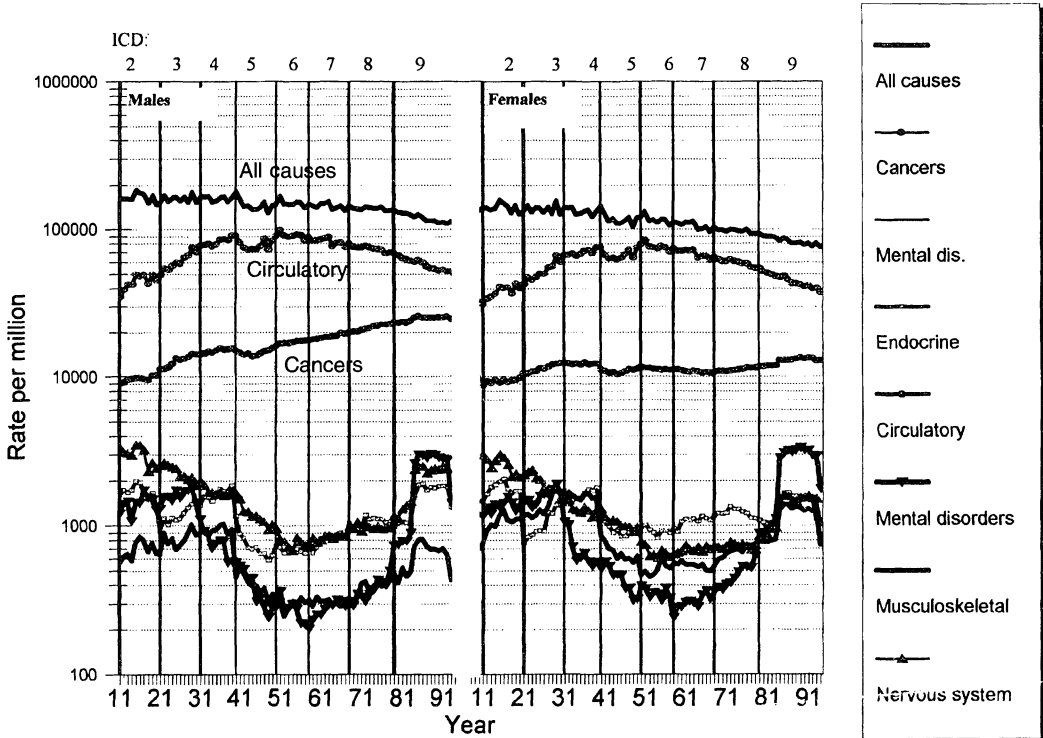


Fig. 13. Mortality trends, 1911-1994: ages 75 years and over, by selected causes

Table 6. Parameters of the fitted logistic models†

<i>Dynasties and years of birth</i>	<i>Deaths at ages 30 years and above</i>	$10^4\alpha$	β	γ	ϕ
Tang dynasty and earlier, 805 BC–978 AD	510	18.7 (18.1)	0.057 (0.012)	0.022 (0.010)	111 (7)
Song dynasty, 979–1278 AD	433	7.7 (2.3)	0.069 (0.004)	0.000 (0.000)	103 (3)
Yuan and Ming dynasties, 1279–1643 AD	337	2.9 (2.7)	0.080 (0.012)	0.010 (0.005)	102 (4)
Qing dynasty, 1644–1860 AD	235	7.2 (3.0)	0.072 (0.007)	0.000 (0.000)	101 (4)

†Standard errors are given in parentheses.

improvements in cohort mortality we can see in Fig. 12 that at age 75 years the improvements in cohort life expectancy are much greater than those calculated on the basis of period life-tables.

The improvement observed in overall mortality among those aged 75 years and over has not been paralleled in *all* the underlying causes of mortality, however—for example death-rates for cancers, musculoskeletal disorders, mental disorders and diseases of the nervous system (including Alzheimer’s and Parkinson’s diseases) and some other degenerative diseases are rising (Fig. 13). This rise in mortality may also reflect an increase in morbidity among those over 75 years old among whom the centenarians still represent only a small proportion (about 0.1%). Trends in the burden of morbidity and disability are of considerable importance to the Health and Social Services, especially if the numbers are growing so fast, but these are currently not very well understood.

David Bartholomew (*Stoke Ash*)

The logistic model is distinguished by the fact that, at very high ages, it levels off to a horizontal asymptote. This implies that, as age increases, age-independent causes of death are becoming relatively more important than age-dependent (i.e. biological) factors. We know that deaths among the very old are often precipitated by falls or other kinds of accident and, given the frailty of all within that age group, we might expect such ‘accidental’ death to be largely independent of age. If this observation is correct, we would predict a levelling-off of the force of mortality and only the logistic model captures that feature.

Q. Tan and J. W. Vaupel (*Max Planck Institute for Demographic Research, Rostock*)

Thatcher presents a logistic model (his formula (3)) and then fits it to several sets of data, with parameter estimates shown in his Table 2. We were curious whether his model would fit data that we have compiled on historical patterns of mortality in China. Our data consist of the age at death and year of death of famous Chinese men who died between 805 BC and 1900 AD (Liao *et al.*, 1990). We omitted a small number of cases for which we suspected that the age at death might have been misreported. We assessed the age at which each man had become sufficiently famous to warrant his inclusion in the book. We then used maximum likelihood methods to fit Thatcher’s model to these conditional survival data. The resulting parameter estimates are shown in Table 6. A visual inspection of graphs such as those presented in Thatcher’s Figs 3–9 indicated that the fit was reasonable, although the observed values did vary owing to the limited size of our samples. The parameter estimates are interesting because they are quite similar to those of Thatcher. This can be taken as additional evidence that simple models might provide serviceable approximations to surfaces of mortality over age and time.

Shiro Horiuchi (*Rockefeller University, New York*)

Thatcher has shown that

- the logistic model fits the historical series of adult mortality in England and Wales very well,
- trends in the logistic parameters are interpretable biomedically and
- the estimated logistic models are consistent with records of the highest age attained.

These findings are important, and I would like to give a few suggestions for future work in this line of research.

Firstly, to justify the use of the logistic model, it is essential to compare the extent to which the logistic and other mathematical models fit data. (I presume that the author is conducting this type of analysis as well.) However, in the conventional semilogarithmic plot of death-rates by age, many different models often appear to fit data fairly well. Thus I recommend that the life-table aging rate LAR (the age-specific rate of relative mortality increase with age) be used for model evaluation. Since the implied LAR pattern differs markedly between the mortality models, the observed variations with age of LAR indicate unambiguously which model really fits data well. In our analysis of international female mortality data, the logistic equation was the only model that fitted the observed LAR patterns reasonably well (Horiuchi and Coale, 1990).

Secondly, this study focuses on period mortality only. It seems interesting to investigate relationships between period and cohort mortality patterns in relation to the logistic model. Should the logistic model fit period schedules or cohort schedules better? Probably it fits both very well, but levels and trends of the logistic parameters are likely to differ significantly between periods and cohorts. For example, if the mortality level is declining, the β s for cohorts should be lower than the β s for periods. Then a question arises: which series of β s reflect the underlying genetically determined pattern of aging, and which series of β s are statistical artefacts?

Finally, it is wisely assumed in this paper that the κ -parameter in equation (1) is fixed to be 1. The parameter seems to be determined mainly by death-rates at age 85 years and over, which are lacking in the three early life-tables. I would like to stress that this assumption should be limited to data in which oldest old death-rates are (partly or totally) lacking or unreliable. The κ -value is the maximum senescent mortality, and its stability should always be checked with the latest data.

Bernard Jeune (*Odense University*)

In his excellent and stimulating paper Roger Thatcher compares Halley's well-known life-table (Breslau, 1687–1691) with Wrigley's new life-table (England, 1640–1689) and finds remarkably similar results. As, however, Buffon (1835) emphasized, mortality figures for this period differ considerably from study to study. Recently Bourdelais (1993) has mentioned several new studies that show variations in adult mortality in the 1600s and the 1700s based on different geographical areas and social classes. Thatcher's remarkably similar results could therefore be nothing more than a coincidence.

The historical trends of the highest attained age are discussed in Jeune and Vaupel (1995). Regrettably, we failed to consider an important study by Charbonneau and Desjardins (1990). Among 210000 burials registered in Quebec before 1800, 178 listed an age at death of 100 years or more. After age validation, only one case could not be refuted. On average the alleged centenarians were in fact 88 years old. However, among the 20000 people born in Quebec before 1700 Desjardins (1998) reports a dozen people aged 95 years and older (the highest confirmed age was 99 years). I therefore agree with Thatcher that people in the 1600s could have reached the age of 90 years. But it is still an open question whether the age of 100 years was reached before 1800. We still lack valid documentation for even one case of a centenarian from that time.

According to some studies reported by Bourdelais (1993) the probability of reaching the age of 60 years in the 1700s for young adults was probably not higher than the probability of living to 80 years old today.

In Table 5 Thatcher shows that the mode of the highest attained age could have been 100 years in a stationary population of 1 million people in the 1600s. This estimation is based on a fitted logistic model of the force of mortality in England from 1640 to 1689, where the highest age among the observed cases was below 85 years (Table 1). Surprisingly, the mode of distribution of the highest attained age in a stationary population of 1 million in England in 1841 is no higher than this, although the force of mortality is clearly lower for all ages—at least up to 85 years—and an observed age of 95 years was not reached. This fact begs for an explanation.

Richard L. Smith (*University of North Carolina, Chapel Hill*)

My comments are concerned with the application of extreme value theory in Section 4.

The author distinguishes between 'classical' extreme value theory, in which the underlying distribution is assumed known, and more modern statistical approaches. He only considers the classical approach, but the way in which this is applied is rather trivial.

Instead, it seems to me that the problem is well suited to modern statistical methods based on exceedances over high thresholds; see for example Davison and Smith (1990). The advantage of these methods, in comparison with those used by the author, is that they allow attention to be focused specifically on the highest ages, so that there is less bias due to a parametric model fitting very well over

the bulk of the data but not in the most extreme portion. The author apparently rejects this approach because of the absence of 'proper reliable data' for the most extreme ages, but this may be based on a misconception of the methodology. What matters is that we have a reasonable quantity of data over the threshold selected. If a few of the very extreme observations are suspect, it might be possible to treat them as censored observations or in some other way to downweight their influence. The presence of such observations does not in itself make the methods invalid.

I have two other comments related to extreme value theory.

- (a) Determining the distribution of the maximum of a sample of size n , where n is some large but finite number, and when the underlying parameters of the model are unknown, can be regarded as a problem of predictive inference. Similar issues have arisen in the discussion of athletics records (Robinson and Tawn, 1995; Smith, 1997). Bayesian methods have a role to play here.
- (b) Does environmental variation play a role? We often hear accounts of how people in certain parts of the world live to exceptional ages because of unique life style or genetic factors. This may suggest some kind of random effects analysis, instead of treating the population members as independent.

In making these comments, I do not seek to be critical of the author's work. The data collection which he and his colleagues have performed is truly impressive; I see my own comments as indicative of the potential for further research. I hope that the data will be made generally available, to allow interested statisticians the opportunity to pursue these fascinating questions.

Nicholas T. Longford (*De Montfort University, Leicester*)

I think that this paper brings out very nicely how crucial is the model specification in estimating quantities about which the data contain little information. A freak event (record longevity) will almost always happen, especially if we search for it sufficiently hard. This makes all models for single extreme events very vulnerable. Although appreciating the historical importance of the 'highest attained age' as an estimand, I would propose as an alternative a high percentile of the ages at death, such as 99.9%, because its estimation is likely to be much more precise, and yet it is a similar characteristic of the studied population.

The following contributions were received in writing after the meeting.

Russell Ecob (*Medical Research Council Medical Sociology Unit, Glasgow*)

May I congratulate the author on his formidable undertaking in collating and modelling mortality, particularly in older age groups over both time and place? These models have potential use to aid in the explanation and understanding of differences in mortality by social groups, regions and countries, both contemporarily and historically (incidentally, I wonder how representative of national populations are the life-tables used in Hungary and England over the period 1640–1689?). Currently there is some concern (Drever and Whitehead, 1997) over the differences in mortality between social groups in both Britain and other European countries, differences in life expectancy at birth for males in the UK during 1977–1981 showing differences between social classes I and II and IV and V of 4.5 years and showing some signs of an increase since then, and differences in life expectancy for males between extreme regions in the UK in 1987 amounting to 3.2 years (Illsley *et al.*, 1990).

Differences between regions also vary according to age, a decrease in the between-regional variance particularly in age groups below 35 years over the period 1931–1981 resulting in larger between regional variance over the period 1971–1981 in the 35–64 years age group (though with more recent increases at ages 25–34 years to similar values) (Ecob *et al.*, 1997). Interestingly there is a relative lack of between-regional variation in the age groups 75 years and over.

What would the author suggest as the most useful method of extending such models to accommodate these differences? In discussion of the 'fixed frailty' model, he suggests that individual frailty α_i is fixed from the age of 30 years onwards. However, the above findings suggest that such models may be usefully fitted to data disaggregated by social-economic factors and possibly by region, allowing in addition random variation at individual and higher levels in other parameters, possibly γ .

Finally, could such extensions to the model account for the apparent lack of fit of the model in particular age groups (particularly the underestimation of mortality in recent periods for females in England and Wales between ages 45–60 years, shown in set G in Table 1)?

T. B. L. Kirkwood (*University of Manchester*)

Thatcher provides an analysis of mortality patterns which will be of undoubted value to those interested in the biology of aging. As he correctly states, most gerontologists now believe that aging results primarily from the gradual accumulation of random damage to cells and tissues in the body, which builds up at a rate that is determined, on average, by the evolutionary optimization of investments in somatic maintenance and repair (Kirkwood, 1996). There is an important difference between this view and the earlier idea that aging is programmed by some kind of biological clock. If such a clock existed, the concept of a fixed definite limit would make more sense. However, there are strong evolutionary arguments against programmed aging. Aging through the accumulation of damage is a process that one would expect to be susceptible to modification, if circumstances change so that the body is subject to fewer intrinsic and extrinsic stresses. The long-term trends described by Thatcher are consistent with this interpretation and it is interesting to ask what biological mechanisms may be responsible for the long-term stability of the parameter β .

The slowing of the mortality rate increase at the highest ages, as shown by the better fit of the logistic model, has attracted much biological interest. There is good reason to regard the model by Mueller and Rose (1996), which suggests that a mortality plateau is intrinsic to the predictions of evolutionary theory, as flawed (Pletcher and Curtsinger, 1998; Kirkwood, 1998). Current explanations for apparent mortality plateaus include genetic heterogeneity, non-genetic heterogeneity due to the stochastic nature of the aging process and modifications of behaviour (human centenarians tend, for example, to receive greater support than people in their 80s and 90s, whereas old fruit-flies crawl instead of flying). All these factors may contribute.

Thatcher's analysis of the distribution of the highest attained age is illuminating and confirms that a species's maximum lifespan is a statistic that should be used with caution, a point which I and others have argued previously (see, for example, Kirkwood (1985)). Comparative biologists are coming to appreciate that the parameters of the gamma–Makeham or logistic model provide a better basis for analysing species or population differences (but this requires larger samples than are often available). This paper reinforces this important message.

Anthony W. Ledford and Michael E. Robinson (*University of Surrey, Guildford*)

The standard practice in extreme value statistical modelling is to base inference on just the extreme observations, and to model these by using a family of theoretically justified tail models. This approach is adopted because the non-extreme and extreme observations may behave differently; thus models which are appropriate for the main part of the data may be inappropriate when extrapolated into the extremes and vice versa. As extreme ages are of primary interest here, extreme value methods have a useful role to play.

We focus on one such method: the peaks-over-threshold approach of Pickands (1975) and Davison and Smith (1990). Let the random variable X denote age at death and u some high age. Then, under standard assumptions, the behaviour of X above u may be described by the generalized Pareto distribution (GPD) given by

$$\Pr(X > u + x | X > u) = (1 + \xi x / \sigma)_+^{1/\xi} \quad \text{for } x > 0,$$

where $s_+ = \max(s, 0)$, $\sigma > 0$ is a scale parameter and $\xi \in \mathbb{R}$ is a shape parameter. The parameter ξ is of particular significance: if $\xi \geq 0$ then the upper tail of X extends to infinity, but if $\xi < 0$ then X has a finite upper tail. To illustrate the above, we followed the approach suggested in Appendix B and fitted the GPD to the males and females data for 1980–1982 (see Table 1) for several values of u . For both sexes, our results suggest that there is a finite upper end point for the age at death. However, owing to the limited nature of the available data, this finding is not conclusive.

Modern extreme value statistical methods may be useful here in other ways. For example, the similarities between the force-of-mortality curves in Fig. 2 suggest that a joint analysis may be appropriate, with parameters that vary with time and depend on sex. Such an analysis would make more efficient use of the available data and would quantify how the extremes of age at death vary between the sexes and how they have changed through history. However, to address such issues adequately, data recorded at a finer resolution or at an individual level may be required. Although the author remarks that *reliable* historical extreme age data are not available, a careful use of censoring may allow less reliable data to be incorporated appropriately in such methods. We are pleased that the full data sets are to be made available to researchers, and we commend the author for his efforts.

John R. Wilmoth (*University of California, Berkeley*)

Roger Thatcher presents a concise and well-reasoned account of our present knowledge about the shape of the age pattern of mortality in adult humans and about the highest ages attained throughout history. I agree with almost everything that Thatcher says in this paper, but I would like to make two specific comments regarding statements made in the discussion.

First, Thatcher asserts: 'The results include the quite specific predictions that ages over 90 years were attained in the mediaeval period and that the age of 100 years was probably attained by the end of the 17th century'. Here, I think it is appropriate to underline that this conclusion, which emerges from Table 4 of the paper, applies to England and Wales only, not to the entire world. For example, the modal maximum age at death for the single cohort born in England and Wales in 1036 would have been only 92 years. However, estimates of the size of the world's population during the 11th century were around 300 million, compared with 1.1 million for England and Wales in 1086. Referring to Table 5, we see that the modal maximum age at death based on mediaeval life-tables in a population of 300 million would have been around 100 years already in this period. Furthermore, Thatcher notes that these life-tables are now thought to underestimate the number of survivors to older ages. Overall, I find Thatcher's empirical results to be entirely consistent with my previous conclusion that human centenarians should have occurred regularly once the world's population exceeded about 100 million people, which is thought to have occurred around 2500 BC. Of course, this conclusion requires the rather heroic assumption that early human mortality was not significantly higher than during the mediaeval period, but the extant evidence from various high mortality populations suggests that such a presumption is not entirely unreasonable.

Second, Thatcher concludes: 'Since the population cannot rise indefinitely and the mortality function cannot fall indefinitely, there must be some high age which is unlikely ever to be exceeded, but it is not predetermined and it is not fixed and definite'. After such a careful discussion of the probabilistic nature of limits to the human lifespan, I found this statement to be puzzling. I would say that the evidence for finite limits to the earth's 'carrying capacity' for human life or for an insuperable lower bound on age-specific mortality rates has no firmer scientific foundation than the claim that the maximum length of life is fixed.

Anatoli Yashin (*Max Planck Institute for Demographic Research, Rostock*)

I congratulate Mr Thatcher on his very interesting and stimulating paper. I have just two brief comments. First, I would like to mention that the logistic mortality curve of the LeBras model of changing frailty is identical with the mortality curve obtained in the 'gamma-Makeham' fixed frailty model (Yashin *et al.*, 1994). This observation shows that survival data alone are not enough to distinguish between the two fundamentally different mechanisms of aging and survival. It seems that data from longitudinal studies of aging, which require more sophisticated models (Yashin and Manton, 1997), are more appropriate for this purpose. Second, there is some question about the explanatory power of the gamma-Makeham model in describing the levelling-off of mortality at late ages due to the fact that univariate frailty models are non-identifiable. Since there is no biological background for the choice of the Gompertz-Makeham underlying hazard, other functions can be used for modelling $\mu_0(x)$ as well. In particular, the logistic $\mu_0(x)$ can be used. Such a choice, however, may exclude heterogeneity in mortality from the causes of the levelling-off of the mortality rate. Fortunately, the bivariate correlated gamma-frailty model (which is identifiable) can be used for the evaluation of both the heterogeneity distribution and an underlying hazard from survival data on related individuals. The analysis of twin data shows that the estimates of $\mu_0(x)$ may increase faster than the Gompertz curve (Yashin and Iachine, 1997).

The **author** replied later, in writing, as follows.

I am grateful to both the proposer and the seconder of the vote of thanks for their kind words. Both have adopted a very wide perspective, but they have not raised any specific points on the paper which call for reply. One may note an interesting difference of approach between mine and Vaupel's. Given a very large volume of data, my natural instinct was to try to find a simple model which would fit. Vaupel, seeing the simple model, has generalized it. Such action and reaction are the stuff of progress and I shall be glad if my paper encourages further research.

Of the specific issues which were raised in the discussion, the most important is whether the force of mortality tends to a finite limit as age increases. The paper argues that both the modern and the

historical data are consistent with the logistic model, which has this property. This implies the corollary discussed in Section 5, that, if a fixed upper limit to life exists, it must be at so high a level that it has no visible effect on our observations.

In any debate about whether there is a finite limit to life, the views of biologists must be crucial. I am therefore extremely glad to see the authoritative contribution by Kirkwood. He lists three current explanations for the levelling-off of mortality at high ages, namely genetic heterogeneity, non-genetic heterogeneity due to the stochastic nature of the aging process and modifications in behaviour. Bartholomew adds a further reason, the proneness to accidents at very high ages.

In the discussion, two additional pieces of factual evidence were presented which further support the conclusion. Charlton showed very clearly in Fig. 11 how age-specific death-rates (and hence the force of mortality) have levelled off at high ages in the cohorts born in England and Wales since 1841. Kannisto reported an important new finding, that the observed upper limit for q_x has not significantly exceeded 0.6 in any of the 31 countries which are now included in the new database on old age mortality.

On the other side of the argument, Ledford and Robinson apply one of the modern methods of extreme value theory, the 'peaks-over-thresholds' approach. This is based on the fact that under standard assumptions the behaviour of the ages at death above a sufficiently high threshold u can be described by the generalized Pareto distribution (GPD). On fitting the GPD to the data for 1980–1982 in Table 1, they find that their results support (though not conclusively) a finite end point for the age at death. However, this finding needs to be examined critically, because we know that the same data can also be fitted by the logistic model, which points to exactly the opposite conclusion. There is a logical contradiction here. It arises, I think, because the thresholds used by Ledford and Robinson are not sufficiently high for the GPD approximation to be valid. If μ_u denotes the force of mortality at the threshold age u , and if mortality truly follows the logistic model (3), then at all ages $x > u$ we shall have $\mu_u < \mu_x < 1 + \gamma$. If u is sufficiently large for μ_u to be close to $1 + \gamma$, then μ_x will be almost constant when $x > u$, whence the distribution of ages at death will be almost exponential and so will be close to the GPD with $\xi = 0$. Thus the extreme value theory will work perfectly, but only when u is sufficiently high for μ_u to be close to $1 + \gamma$. Unfortunately, the force of mortality does not reach this level until beyond the range of ages which we can observe.

In Thatcher *et al.* (1998), my colleagues and I applied a battery of tests which would have detected any serious departures from the logistic model below age 120 years, but we did not find them. Of course, the model may break down somewhere above this age, but it would still need a threshold above age 120 years to detect this. Although I can see the theoretical attractions of the peaks-over-thresholds approach, as advocated by Smith, I think that he will find it difficult to apply this method to the data on human mortality, because the required threshold is so high. The methods which I used in the paper may look old fashioned, but at least they can be applied to the data which are available in this particular problem.

I now turn to the comments on the historical highest ages. I am delighted that Wilmoth finds that my calculations are generally consistent with his own. He is quite right to stress that my Tables 1–4 are confined to England and Wales. He and I are in good agreement from the Middle Ages to the present, and it is a very minor difference that we seem to take different views about the indefinite future! In my case, I simply thought it unlikely that mortality rates will fall as far as 0, when our evolutionary past dictates that the human body will deteriorate after the age of reproduction.

I also very much welcome the informed comments of Jeune. Of course, I accept that the close agreement between Halley's and Wrigley's life-tables may be partly coincidental, but it is a happy coincidence. The only clear difference between Jeune and me is really rather slight, about whether there were any centenarians before the year 1800. Both Wilmoth's and my statistical calculations suggest that there were probably a few, though very rare indeed. Also, Desjardins has now identified a person born in Quebec in 1648 who definitely reached 99 years of age. The population of England in 1648 was much larger than the population of Quebec, so it is reasonable to suppose that there may have been a centenarian in England before 1800, let alone somewhere in the rest of the world. However, I accept that this remains an open question until a definite case has been identified. The last point raised by Jeune is based on a misunderstanding. The 'observed' data in Table 1 are truncated, for the reason stated in the paragraph below Table 1. This does not mean that there were no observed deaths above age 95 years in 1841.

I was also very pleased to see the comments of Horiuchi. His excellent summary of my paper is far more concise than my own and I was glad to receive his endorsement of my decision to assume the fixed value $\kappa = 1$ for the historical periods, in the absence of more reliable data. Material that is relevant to some of his questions will be found in Thatcher *et al.* (1998). For example, the coefficients β are indeed

found to be lower for cohorts than for periods, as they are bound to be when mortality is falling. Both, however, can be affected to some extent by changes in circumstances, so neither is a completely pure measure of an underlying pattern.

Rosenbaum, in his inimitable and entertaining way, has applied extreme value theory to the ages of Fellows in our own Society. His example is instructive and also strikes home!

Tan and Vaupel have produced some extraordinary and completely unexpected data on ages in early China. Perhaps one should not take the results for the Tang dynasty too literally, but the other results show mediaeval levels of mortality in the middle range of ages and modern levels at very high ages. This is similar to data from Roman tombstones (see Section 3.1). There is food for thought here. It is good to know that these data exist and that the model can fit them.

I am glad that Charlton showed us his figures and I have already commented on his valuable Fig. 11. Fig. 12 must be interpreted with care because there are time lags. For example, the people who were 75 years of age in 1991 (with the expectation of life represented by the last points on the period curves) were members of the cohort born in 1916, not the cohort born in 1941.

Ecob asks about applying the model to study the long-standing differentials in mortality between regions and between social classes. This is an important question and we need to proceed in stages, the first priority being to discover whether the model will be useful for this purpose. My suggestion would be to start very simply indeed, by taking regional data for a modern (post-1950) period. For these we would expect γ to be negligible, so that use can be made of equation (6). If plots of logit (μ_x) against x show that the lines are reasonably straight at ages over 30 years, for individual regions (or social classes), then the model will be useful and we can start to consider the implications of the levels and slopes of the lines. On Ecob's final question, although the relative error for these groups is in one case 13.8%, the corresponding absolute error in the force of mortality is only 0.0007 and it is not clear whether this is a matter for concern.

I am grateful to Yashin for his further references and to Longford for his comment, though he will find that high percentiles are not easy to measure in historical cohorts.

Several contributors were keen to have more data. The position is that the full historical life-tables can be found in the references cited in Section 3.1. The pooled modern data for 13 industrialized countries have now also been published, in Thatcher *et al.* (1998). Thus there is already plenty of material for those who wish to make a start. The full disaggregated data for up to 31 individual countries are expected to be available shortly, on application.

I would like to thank all the contributors to the discussion, and particularly those who travelled from overseas to attend the meeting.

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