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Empirical EDA Models to Fit and Project Time Series of Age-Specific Mortality Rates

by

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Abstract

This paper illustrates an application of Exploratory Data Analysis methods to inspect, fit and project a time series of age-specific mortality rates. The analysis centers on Norwegian age-specific mortality rates covering the period 1846–1988. The main emphasis lies on selecting a simple empirical model facilitating to extrapolate the time dimension in order to obtain mortality projections. As three dimensions may underlie the data —age, period and cohort factors—some attention is devoted to disentangle the relative magnitude of these three sources of variation. To circumvent the well known identification problem caused by the trivial relationship *period* = *age* + *cohort* the estimation method used is based on a weighted iterative procedure along stepwise robust estimation methods. The analysis shows that, other than the effects of the Spanish influenza in 1918 and the Second World War—which produced cohort traces in the form of selection and debilitation effects, but subsided by the sixties—no need is apparent to model cohort effects in the projection. A relatively simple age and period model is adequate to perform the projection.

1 Introduction

When carrying out population projections distinction is usually made whether the exercise is meant as a projection or a forecast, the latter implying an element of prediction while the former simply represents a numerical scenario derived from a particular set of assumptions. In the analysis undertaken here—restricted to one population component, mortality—the most simplistic projection perspective is subscribed, crude extrapolation. Although most of the emphasis is focused on how to facilitate producing a sound extrapolation, no claim is made on the likelihood that the projected rates would materialize on time. Extrapolation has been—and still is—a preferred method for mortality projection, even in models that incorporate covariates to guide the projection or models based on mortality *laws*, in a sense encompassing disease and attrition processes (Manton and Stallard, 1984 and 1988).

A crucial initial question is what to project. Keyfitz (1982) holds the view that effective mortality forecasting depends on a minimum parameter representation. That is, the simpler the (parametric) model assumed to represent mortality variation the easier and the more effective the task. Two extreme cases set limits to the problem: at one end every single age-specific rate is treated as an independent item subject to projection, at the other end projecting a single parameter or summary index—say, life expectancy at birth—assures the projection. No model bounds the age-specific mortality rates in the former, while a perfect one-parameter model is presumed to hold in the latter.

Considerable literature exists on assessing the minimum dimensionality of mortality variation. Keyfitz (1982) and Pollard (1987) give detailed reviews of mortality models with emphasis on their use for population projections. It is generally admitted that no less than eight parameters are required to model effectively the whole age range of mortality variation by mathematical formula.[1] Heligman and Pollard (1980), for instance, propose the following eight-parameter model of the probability of dying at age x:

$$q_x/(1-q_x) = A^{(x+B)^c} + D\exp\{-E[\ln(x/F)]^2\} + GH^x,$$

where the first term parametrizes early childhood mortality, the second term takes account of accident mortality in adolescence, and the last term models senescent mortality à la Gompertz.

Along the same rationale but modifying slightly the Heligman-Pollard formula, Rogers (1986) proposes the following eight-parameter model of the death rate:

$$\mu(x) = \mu_I(x) + \mu_A(x) + \mu_S(x)$$

[1] Since the seminal work of Gomperts in 1825 subsequent attempts to model mortality have been pursued by Makeham, Thiele, Perks and Barnett, among others. These models fit with varied degree of success the medium and older segments of observed mortality, but they have proved unsatisfactory to fit the whole age range.

where

$$\mu_I = \begin{cases} Q_0 & \text{for } x \simeq 0 \\ Q_1^{x^\gamma} & \text{for } x \geq 1 \end{cases}$$

$$\mu_A(x) = Q_A \exp\{-[(\ln x - \ln x_A)/\sigma]^2\} \text{for} a \geq 0$$

$$\mu_S(x) = Q_S \exp[x/x_S]/\{1 + Q_S \exp[x/x_S]\}.$$

The three terms represent the effects of infant and childhood (μ_I), early adult (μ_A) and senescent (μ_S) mortality risk factors embodied in these ages. The parameters Q_0 and Q_1 of the first term are approximately equal to the infant and child mortality rates, respectively, and γ is the speed of decline in the death rate throughout childhood. The parameters Q_A, x_A and σ of the second term are indicators of the level, location and spread of mid-life mortality, respectively. Finally, Q_S indicates the level of senescent mortality measured at age zero, and x_S is inversely related to the rate of increase of senescent mortality with age. Compared with the Gompertz term in the Heligman-Pollard formula, Roger's version uses a model of latent frail heterogeneity, which brings the model closer to the theoretical arguments in favor of explicitly modelling unobserved heterogeneity in hazard models (Manton, Stallard and Vaupel, 1986).

At any rate, to project eight parameters is hardly a major improvement over projecting the eighteen or so classical age groups of an abridged life table.[2] As an alternative, relational models, although empirically based, are considerably more parsimonious. Brass' logit-logit model, for instance, is a linear two-parameter system (Brass, 1971):

$$Y_x = \alpha + \beta Y_x^{(s)},$$

where

$$Y_x = \text{logit}(l_x) = \ln[l_x/(1 - l_x)]$$

and l_x is the probability survivor function of a life table. $Y_x^{(s)}$ is the logit transformation of a *standard* life table survivor function $l_x^{(s)}$. In order to project mortality (the survivor function in this case), one simply takes $Y_x^{(s)}$ as the logit of a current life table and projects α and β into future time, most likely as an extrapolation of observed past trends in these two parameters. Projected l_x values are obtained by inverting the logit. The parameter dimensionality of the projection is thus reduced to an age-specific standard $Y_x^{(s)}$ and two time series, α_t and β_t.

Unfortunately, the above level of simplicity is not good enough in many instances. Given a standard l_x, the two parameters α and β in Brass' system determine the level and the steepness of the l_x curve, but are ineffective to accommodate some sources of additional variation in the youngest and oldest ages of the life table age

[2] A clear advantage, however, would be that the parametrised projection would presumably hold the death rates 'disciplined' by the model. A risk often encountered while extrapolating individual age-specific rates is to run into unlikely or aberrant age schedules.

range. To overcome this limitation, Zaba (1979) expanded Brass' model to a four-parameter logit-logit system by making the standard itself a function of two additional parameters that reflect common patterns of deviation from the basic standard.

In an even more flexible model, Ewbank *et.al.* (1983) propose an alternative four-parameter model—called *adaptive*—in which a two-parameter transformation replaces the logit of the standard:

$$Y_x = \alpha + \beta T(l_x^{(s)}),$$

where

$$T(p;\kappa,\lambda) = \begin{cases} (2\kappa)^{-1}\{[p/(1-p)]^{\kappa} - 1\} & \text{for} p \geq 0.5 \\ (2\lambda)^{-1}\{1 - [(1-p)/p]^{\lambda}\} & \text{for} p < 0.5. \end{cases}$$

When κ and λ approach zero, $T(\cdot)$ becomes the logit transformation and the model collapses to Brass' logit-logit system. When either κ or β are zero the model becomes a three-parameter system. Thus, for a particular fit the number of parameters required (from two to four) remains an empirical question. Extensive fitting with the adaptive model shows that a wide range of mortality variation can be adequately fitted with two to three parameters and a suitable standard (Gómez de León, 1982).

In this contribution we substantiate the view that relational models of dimensionality three suffice to fit a time series of age-specific mortality rates when no significant cohort effects are present. In addition, we show that, of the three dimensions required, only one suffices to capture the time-trend effects when the fit is conducted with a model comprising additive and multiplicative terms. An illustrative mortality projection is shown based on extrapolating with these principles the time trend that results from fitting a series of Norwegian mortality rates.

The models used here are first and foremost *empirical*, in the sense that they do not stem from theoretical considerations like the arguments behind the mortality *law* components in Heligman-Pollard or Roger's models, or the linear logit link in Brass' relational model. The analytic perspective used is derived rather from Exploratory Data Analysis with its emphasis on disentangling structure in a data set from recursive model fitting, residual inspection, and model improvement. Section 2 describes the exploratory models used. Section 3 describes the estimation methods together with different diagnostic tools and criteria followed for model assessment. Section 4 describes salient traits of the data set: age-specific mortality rates by single year of age and single calendar years from 1846 to 1988, for each sex. Section 5 shows the results of alternative fits and the main features of the preferred model. Section 6 illustrates a mortality projection for Norway twenty years from now. Finally, a brief concluding Discussion closes the paper.

2 Exploratory Models for Two-Way Tables

A brief clarification of forecasting terminology seems in order before shifting attention to the models. The data series defines a two-way table y_{ij} of mortality rates (or their transformed values) with the usual row and column subscripts $i = 1,\ldots,I$ and $j = 1,\ldots,J$, where i index age and j index calendar time. Assume we wish to project a series of vectors $y_{.j}$ of age-specific mortality rates to $j = T = J + t$, filling the unobserved period $t \geq 1$. Clearly, the observations available to base the projection cover J years. Cohen (1986) calls $j = T$ the target of the projection, $j = J$ the launch date, and $j = 1$ the base date. These dates define three intervals, the *range* of the projection (from $j = 1$ to $j = T$), the *span* (from $j = 1$ to $j = J$) and the *gap* (from $j = J$ to $j = T$). The span is said to support the gap as extrapolation is based on the former.

A variety of models can serve to describe the underlying pattern in a two-way table y_{ij}; from the trivial constant model $y_{ij} = \tau$ to the singular value decomposition of y_{ij}. The models used here are of at most rank two. These comprise the following partial hierarchy:

Simple multiplicative model:

$$y_{ij} = h\,\alpha_i\,A_j + z_{ij} \tag{1}$$

where $\sum A_j^2 = 1$;

Simple Additive Model:

$$y_{ij} = \tau + \alpha_i + A_j + z_{ij} \tag{2}$$

where $\sum \alpha_i = \sum A_j = 0$;

Concurrent Model:

$$y_{ij} = \tau + \alpha_i + A_j + \kappa\,\alpha_i\,A_j + z_{ij} \tag{3}$$

where $\sum \alpha_i = \sum A_j = 0$;

Additive-plus-Multiplicative Model:

$$y_{ij} = \tau + \alpha_i + A_j + \beta_i\,B_j + z_{ij} \tag{4}$$

where $\sum \alpha_i = \sum A_j = 0$, $\sum B_j^2 = 1$;

Rows-linear Model:

$$y_{ij} = \tau + \alpha_i + \beta_i\,B_j + z_{ij} \tag{5}$$

where $\sum \alpha_i = 0$, $\sum B_j^2 = 1$;

Columns-linear Model:

$$y_{ij} = \tau + A_j + \beta_i B_j + z_{ij} \tag{6}$$

where $\sum A_j = 0, \sum B_j^2 = 1$;

Double Multiplicative Model:

$$y_{ij} = \alpha_i A_j + \beta_i B_j + z_{ij} \tag{7}$$

where $\sum A_j^2 = \sum B_j^2 = 1$, and $\sum A_j B_j = 0$.

The different models above specify different forms of structural relationship between the row effects (age effects) and the column effects (period effects) of a data matrix y_{ij}. Not all of them, however, are truly structurally independent, as some are just rewritten forms of others under different data transformations. The equivalence of the multiplicative and additive models (1) and (2) under the logarithmic transformation is quite obvious. Another simple power transformation—exploited below in Section 5—links the additive and concurrent models (2) and (3).

The *multiplicative model* (1) is so central to demographic analysis that it hardly needs any comment. It is the simplest proportional hazards model where the rates y_{ij} are the product of a 'standard' age-specific process factor α_i and a group-specific risk factor A_j. The *additive model* (2) is the classical no-interaction model in two-way analysis of variance. The single additive effects α_i and A_j represent deviations from the means of data variation in their respective dimensions i and j, with the matrix y_{ij} centered on τ, an overall measure of center.

The *concurrent model* (3) states that the residuals from the additive model conform to the simple linear form $z_{ij} = \kappa \alpha_i A_j$ where κ is a single 'interaction term' (itself a function of the main effects α_i and A_j). Tukey (1949) proposed and labeled this model 'one degree of freedom for non-additivity'. The *additive-plus-multiplicative model* (4) seeks for multiplicative structure in the residuals z_{ij} from the simple additive model. It was first proposed by Mandel (1961) as a more general model than Tukey's concurrent model for non-additivity in two-way analysis of variance. It can be interpreted as the first principal component from additivity.

When additivity is not tenable and the extra 'one degree of freedom' of the concurrent model does not suffice, an alternative is to introduce a bilinear term for the rows or the columns, leading to the *rows-linear* (5) or *columns-linear* (6) models, respectively. Finally, the *double multiplicative model* (7) is equivalent to the two principal components decomposition of y_{ij}, except for the fact that the data matrix is not mean-corrected as usually required in component analysis. As an extension of model (1), the double multiplicative model represents a *biproportional* hazards model, where the underlying hazards α_i and β_i operate additively. A_j and B_j are the respective 'covariate' effects.

3 Estimation and Criteria for Model Selection

3.1 Fitting procedures

When in a matrix y_{ij} (as defined above) the interval widths of both age and period concord, a third dimension is discernable, that of cohort. Thus, except for refinements in the classification of data, any two-way table of demographic data congruously cross classified by age of the individuals and calendar time has the extra dimension of cohort or generation. This is indeed the case of the Norwegian mortality rates under analysis here, as will become clear in Section 4.

Logically, it is improper to treat age, period and cohort as three distinct dimensions as they are subject to the linear relationship $k = I - i + j$, where $k = 1, \ldots, K$ index cohort, and the other indices stand as before.[3] In some instances, however, the three factors are genuinely of import and one would like to incorporate their relative effects in a single model. To do so, one faces the well known indeterminacy problem posed by the redundancy embedded in the linear relationship cited above.

To circumvent this problem we take advantage of the fact that, when modelling age, period and cohort data with, say, only age and period factors, the effects of the variable left out from the model should—per force—appear as structured residuals along the diagonals of the residual matrix. If sufficiently prominent, these effects can in turn be estimated from the residuals. A convenient fitting strategy seems thus to resort to robust-resistant estimation methods, which precisely downgrade the weight of data non conforming to the structure stipulated by the model. We use, with minor adaptations, the procedures proposed by McNeil (1974) and further expounded and exemplified by McNeil and Tukey (1975), Breckenridge (1976 and 1983) and Orav (1977).[4]

Essentially, two basic estimation modules are necessary to fit the range of models (1) to (7), one to estimate linear (additive) components, and one to estimate bilinear (multiplicative) components. Joint linear and bilinear fits can be easily implemented by appropriately chaining linear and bilinear fits (Gabriel, 1978). For instance, the additive-plus-multiplicative model can be estimated in a stepwise manner by fitting first the linear terms $\tau + \alpha_i + A_j$ and then the bilinear term $\beta_i B_j$ to the corresponding residuals. Similarly, the rows-linear model can be estimated by fitting the bilinear term $\beta_i B_j$ from the residuals of the linear fit $y_{ij} = \tau + \alpha_i$.

[3] Moreover, age, period and cohort cannot be taken themselves as factors in a strict causal sense. They merely act as surrogate variables capturing underlying processes that exert their influence along these three dimensions. Of the three, age is perhaps the only factor exerting a more direct effect (both as biological ageing and accumulated injury), though tenable only at increasingly older ages.

[4] A similar perspective to inspect for age, period and cohort effects is used by Wilmoth, Vallin and Caselli (1989), but resorting to least squares as estimation procedure. Their analysis is pursued based on a model of the form $y_{ij} = \alpha_i + \beta_j + \sum_k^3 \gamma_{ki}\delta_{kj} + z_{ij}$, an extension of the additive-plus-multiplicative model (5).

The core of the fitting method is an extension to additive and multiplicative fits of M-estimates, where the weighting function is Tukey's *biweight* function (Mosteller and Tukey, 1977, Chapter 10):

$$w_i = \{1 - \min(1, u_i^2)\}^2, \tag{8}$$

with

$$u_i = (x_i - \theta)/cS = z_i/cS,$$

where θ is the parameter under fit of variate x_i, c is a resistance leverage constant (here set equal to 9), and S is the median absolute deviation:

$$S = \text{med}\{|z_i - \text{med}(z_i)|\}$$

Clearly, since θ is implicit in the weights resort must be made to iteration.

3.2 Diagnostics and assessment of fit

Model selection is based on two criteria: inspection of particular diagnostics, and assessment of goodness of fit. We pause briefly to describe each succinctly.

3.2.1 Diagnostics

Two models play a central position in screening the pertinence of different fits in the class (1)–(7), the concurrent model and the double multiplicative model. Tukey's concurrent model (3) essentially states that, under a suitable one-parameter transformation, the data conforms to an additive pattern. Thus, regressing the residuals z_{ij} of the additive model (2) on $\alpha_i A_j/\tau$ (called a *diagnostic plot*):

$$z_{ij} = k\,(\alpha_i A_j/\tau),$$

provides a basis for assessing the need for the extra parameter κ in (3). For instance, if the slope k above approaches one, model (3) becomes simply additive under the logarithmic transformation. In general, a power transformation $(y_{ij})^p$, with $p = 1 - k\tau$, can remove the non-additivity of data conforming to (3). Stated otherwise, a power transformation links models (2) and (3). For the Norwegian data under analysis below, both male and female diagnostic plots yield slopes k close to one, which points out the pertinence of the logarithm transformation to enhance additive structure.[5]

The double multiplicative model offers in turn a peculiarity. The two sets of row factors α_i and β_i can be taken as I points $\mathbf{g}_i' = (\alpha_i, \beta_i)$ and plotted in a two-dimensional coordinate system for each i. Similarly A_j and B_j can be viewed as

[5]One point worth making here is that the logarithm transformation also enhanced structure in models involving additive and multiplicative terms, like the rows-linear and double multiplicative models.

$\mathbf{h}'_j = (A_j, B_j)$ and plotted as J points in a plane. With a suitable choice of scale, both plots can be displayed jointly, thus simultaneously exhibiting relationships between rows *and* between columns in y_{ij}. Gabriel (1971) calls this a *biplot.* Of particular interest in our context is the fact that the biplot serves also as a graphical inspection tool to diagnose the pertinence of models (1)–(7) to fit a data matrix. Bradu and Gabriel (1978), Gabriel (1981), and Cox and Gabriel (1982) give formal arguments for this use and show illustrative examples. The following diagnostic rules apply:

- When the row and column points are jointly collinear and lie on one single line the data conforms to the *simple multiplicative model.*
- When both the row and column points are collinear and form lines at 90° to each other the indicated model is the *simple additive model.*
- When both the row and column points are collinear but the angle between their lines is not 90° the *concurrent model* is prescribed.
- When only the column points are collinear the *rows-linear model* is prescribed.
- Finally, when only the row points are collinear the *columns-linear model* is indicated.

In Section 5 we make practical use of the biplot diagnostic rules to assist in model selection.

3.2.2 Goodness-of-fit criteria

We now turn to goodness-of-fit which is usually the primordial criterion for assessment and selection among alternative models. In the context of resistant fitting, however, any conventional measure of goodness-of-fit may give the misleading impression of a poor fit as it picks up large residuals that may have insignificant weight in the fit. In view of this, two simultaneous approaches are taken to judge residuals: to examine a number of summary measures of size and dispersion of z_{ij}, and to inspect schematic plots of coded residuals.

In two-way analysis of variance a common measure of goodness-of-fit is the classical R^2, the fraction of the sum of squared variation explained by the fit. A related but more suitable measure in our context is:

$$P = \left(1 - \frac{\sum\sum |z_{ij}|}{\sum\sum |y_{ij} - \text{med}\{y_{ij}\}|}\right) \times 100,$$

the percent reduction in total absolute variation achieved by the fit. Although P is not as sensible as R^2 to outliers it still lacks resistance to stray values. We therefore propose to look also at $\sum |w_{ij} z_{ij}|$, $\text{med}\{z_{ij}\}$, and $\text{spread}(z_{ij})$, the latter defined as the difference between the middle values of each half of the ordered residuals.

To inspect for normality in the residuals we examine the *mid* and the *pseudo-standard deviation* estimates that result from successively splitting the ordered residuals exactly in half (Velleman and Hoaglin, 1981). For distributions approximately

normal, the mid and standard deviation estimates should be nearly constant at different depths of the data.

Finally, a complementary tool is to inspect visually the matrix of residuals, but converted into a coded table with codes replacing the cell figures by a character summarizing their relative position in the distribution of z_{ij}. This allows for quick inspection of residual structure. Three levels of codes are retained. These stand roughly for medium, large and extreme residuals, positive and negative.[6] All these criteria make the core of the analysis of Section 5. Before shifting attention to the results some general traits of the data are examined first.

4 A Time Series of Mortality Rates

The data under analysis consists of a series of Norwegian annual age-specific mortality rates for single year of age between 0 and 98, single calendar years between 1846 and 1988, and for each sex. The rates—designated $m_t(x)$—are defined as *prospective* rates, that is, cohort-period rates in the Lexis diagram. The corresponding probability is denoted $q_t(x)$.[7] The series up to 1980 was assembled and analyzed by Jens-Kristian Borgan (1983). Other than different methods and pursuing different analytic goals, a difference between the present analysis and Borgan's is that his is based on five-year cohorts or five-year periods (according to corresponding cohort or period perspectives) and with the rates smoothed by five-point moving averages. While inspecting for age, period and cohort effects we have preferred to base our analysis on the observed rates. The final retained model and the ensuing projection are based on smoothed rates (as described in Section 5).

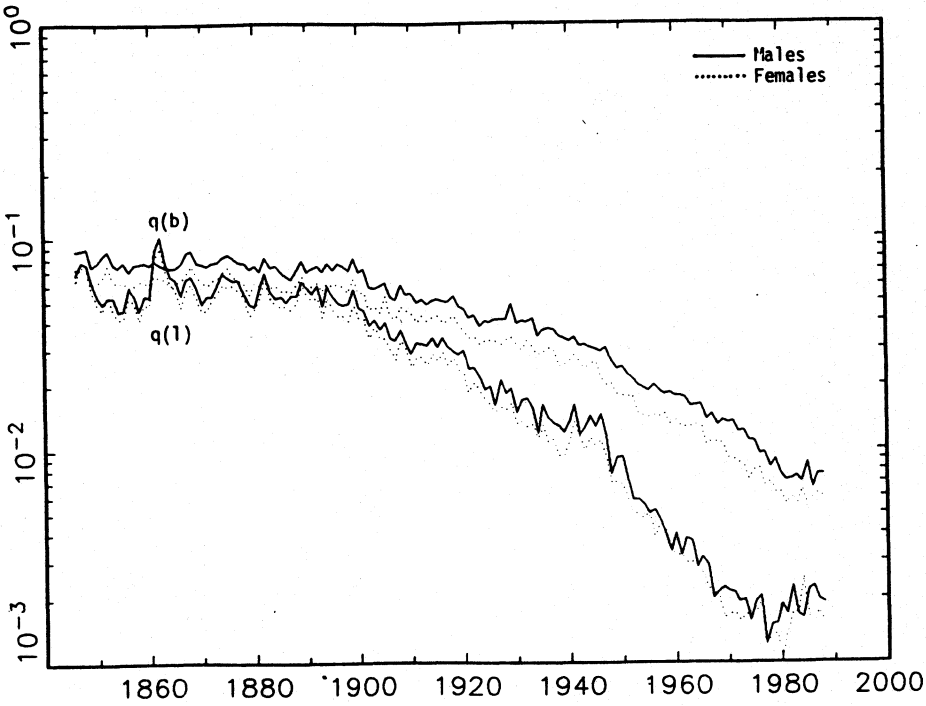
Figures 1 to 5 show the recorded time-series values of the probability of death $q(x)$ at selected ages 0, 1, 5, 10, 25, 35, 55 and 75 (plotted in the logarithmic scale). To a large extent the graphs speak for themselves and we do not plunge in details in our description.

Mortality in the youngest age segments $q(0)$ and $q(1)$ (Figure 1) show no appreciable decline during the nineteen century; only after 1900 a decrease is apparent, particularly noticeable for $q(1)$. The peaks of $q(1)$ in 1848 and 1862 mark epidemic periods of diphteria and scarlatina, and the two plateaux around 1915 and 1943 attest temporary slow downs in the decline. The slight mortality increases after 1980 in both $q(0)$ and $q(1)$ are attributed partly to complications after birth of 'high-risk' pregnancies that come to term only as the result of increasingly intensive specialized

[6] The ranked codes are: $\emptyset$, =,−, +, ×, *. Unfortunately, the size of the coded tables overflow the regular paper size and, for this reason, they are not presented except for Figure 9 retained as the sole illustration shown.

[7] In Lexis diagram terminology $m_t(x)$ designates $m_x^{(t)} \simeq [L_{x-1}^{(t)} - L_x^{(t+1)}]/l_x$ which can be viewed as mortality centered at exact age one. This convention is retained for all ages except for $m_t(0)$, which corresponds to exposure from birth to the end of year t. The conventional formula $q_x = 2m_x/(2+m_x)$ is used to convert rates into probabilities for $x \geq 1$. $q_0 = 1 - L_0/l_0 = 2m_0/(4+m_x)$.

Figure 1: Probabilities of Death $q(0)$ and $q(5)$ for Males and Females, 1846–1988



medical measures during the late stage of gestation and at delivery. Mortality at ages five and ten (Figure 2) follow a similar but more gradual decline. The 1848 and 1862 diphtheria periods are also evident at these two ages, plus two eye-catching spikes protruding at 1918-19 and 1943, the marks of the influenza pandemic—the 'Spanish flu'—and the Second World War occupation.

Mortality at age 25 (Figure 3) shows a peculiar increase from 1846 to 1895 (slightly more salient for men), followed by a decline perturbed quite overwhelmingly by the Spanish flu and the War. The 1918 peak is equally prominent for the two sexes while mortality during the War is overwhelmingly higher for males[8] than for females. Also noteworthy is that the so prominent mortality gap between the sexes prior to 1918 almost vanishes during the decline, but it reappears and increases after 1950 as the male decline slows down and arrests earlier than the female decline.

Mortality at age 35 (Figure 4) remains approximately constant until 1890, followed by a tenuous decline until the 1918 epidemic. Very conspicuous at this age is the absence all along the 19th century—and well until 1910—of the characteristic male mortality disadvantage. Indeed, for some relatively sustained periods, female mortality surpasses male mortality. This feature plainly reflects the high levels of maternal mortality prevailing during the 19th century.

At ages 55 and 75 (Figure 5) several features compel notice. Most remarkable is the absence of traces of the 1918 and 1940 mortality crises. Clearly, whatever the

[8]The beginning of the War in 1940 shows a clear mark in this age group.

Figure 2: Probabilities of Death $q(5)$ and $q(10)$ for Males and Females, 1846–1988

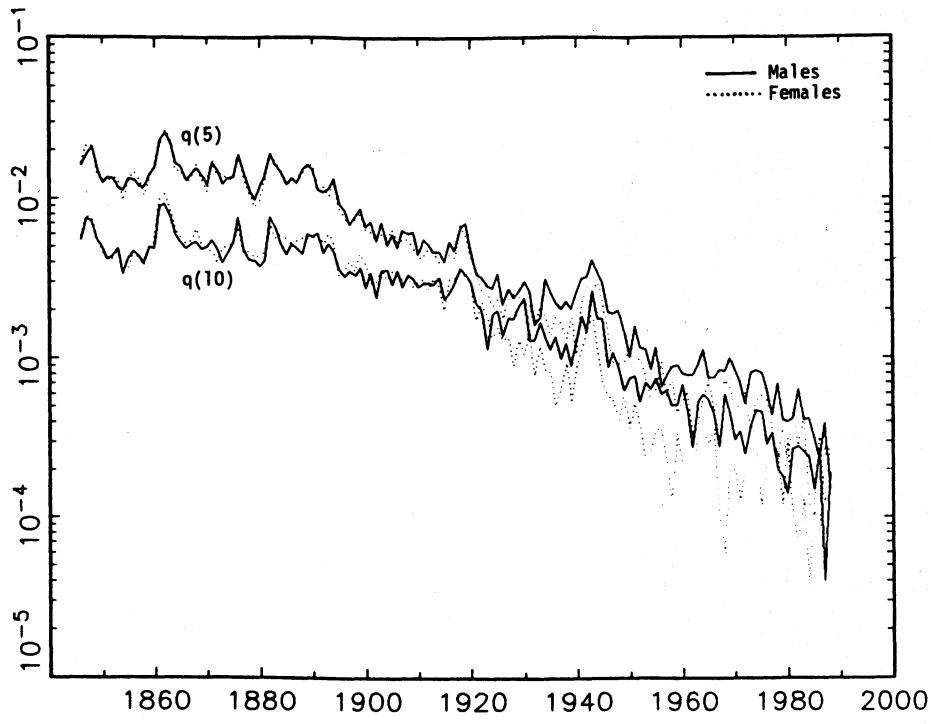


Figure 3: Probability of Death $q(25)$ for Males and Females, 1846–1988

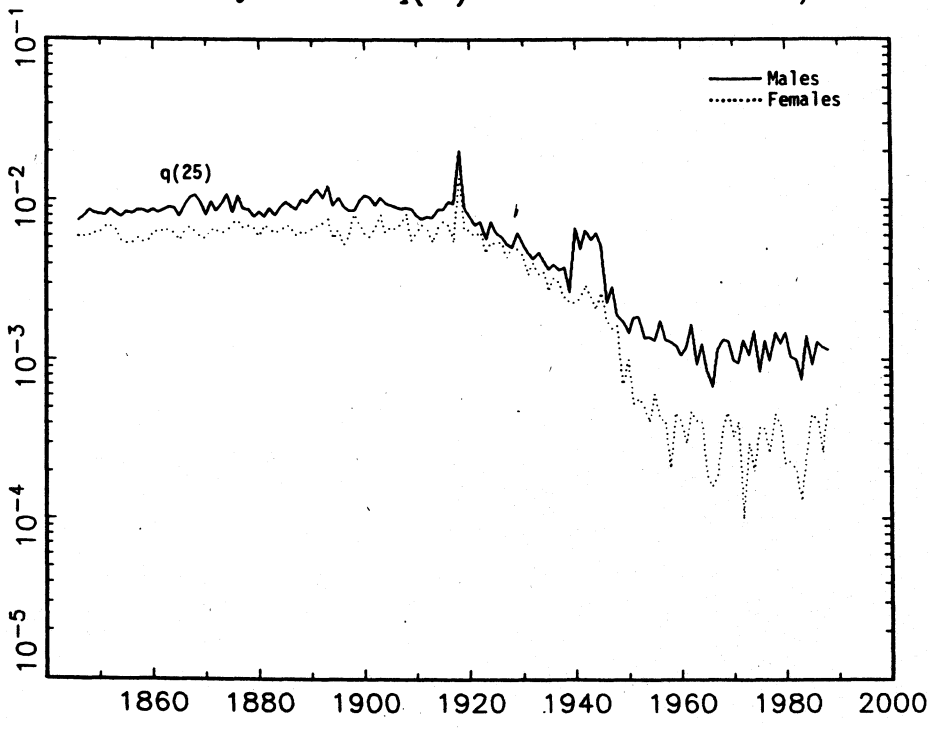


Figure 4: Probability of Death $q(35)$ for Males and Females, 1846–1988

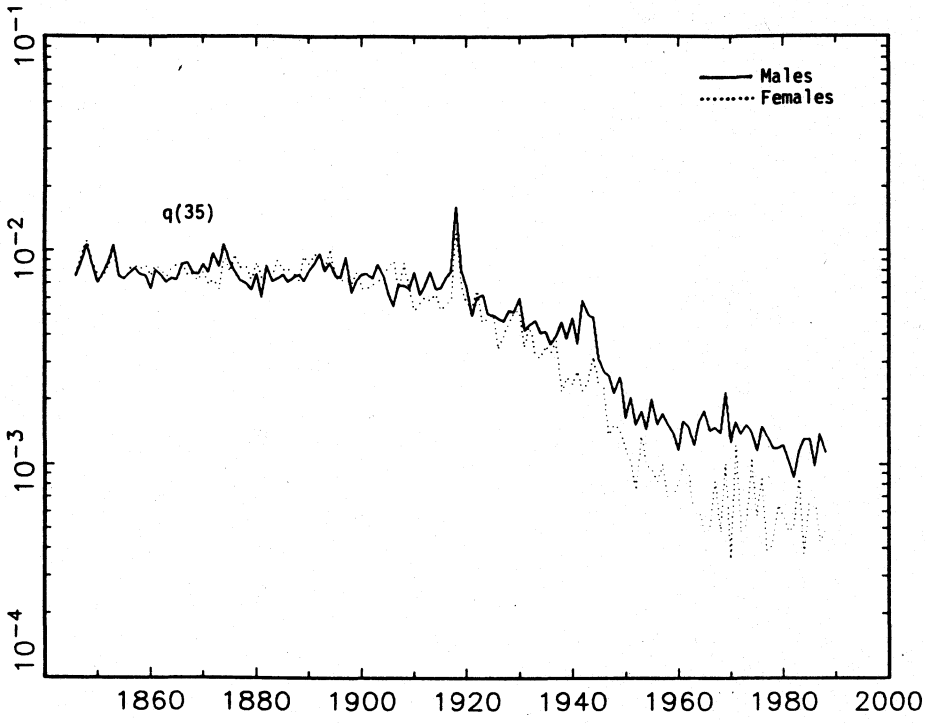


Figure 5: Probabilities of Death $q(55)$ and $q(75)$ for Males and Females, 1846–1988

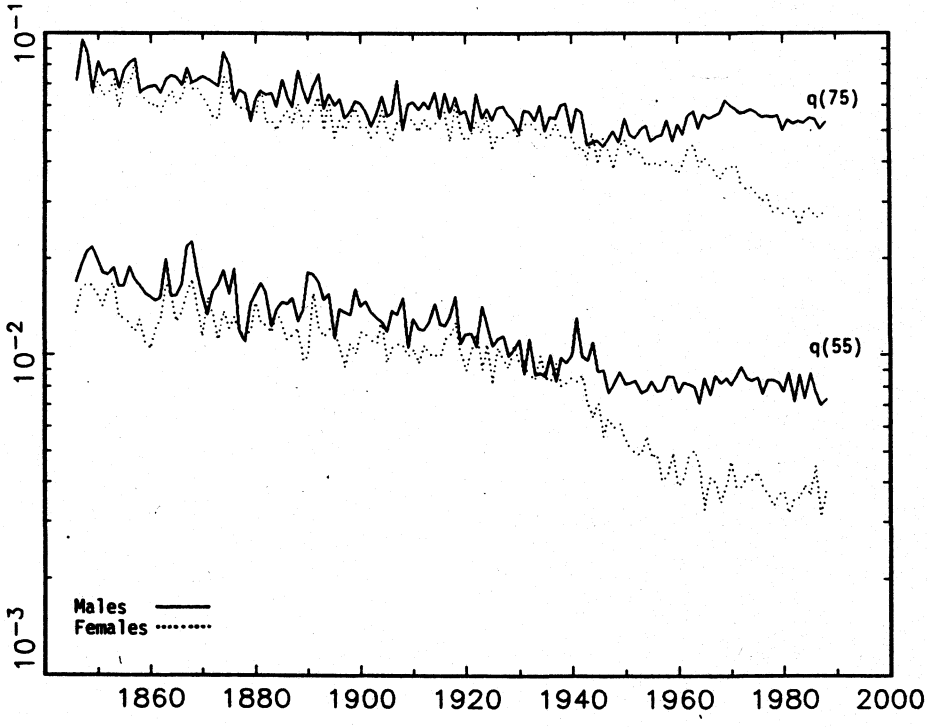
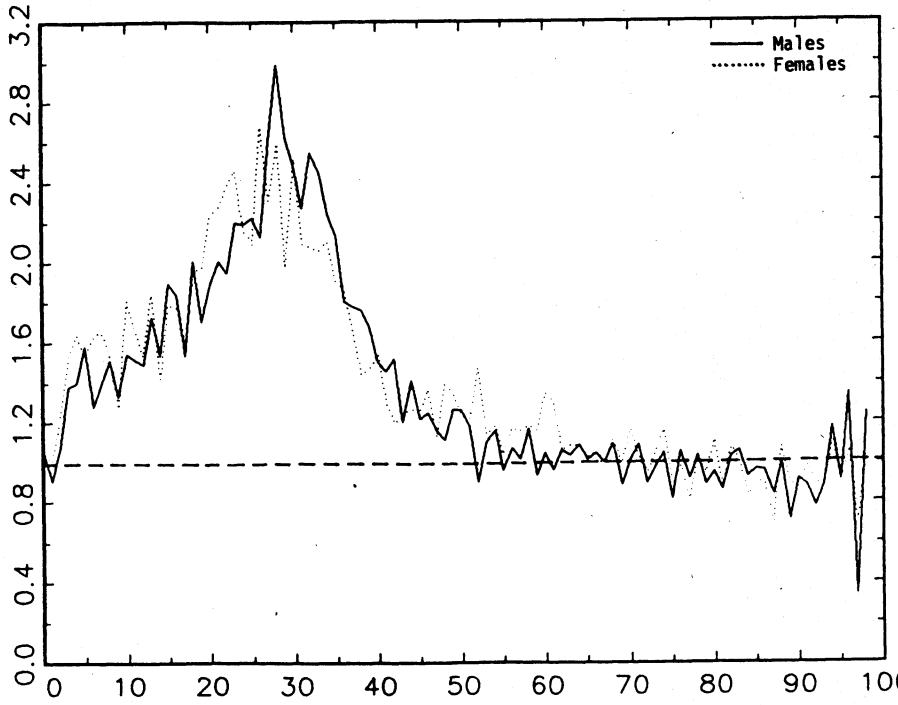


Figure 6: Age and Sex Profile of Excess Crisis Mortality: 1818

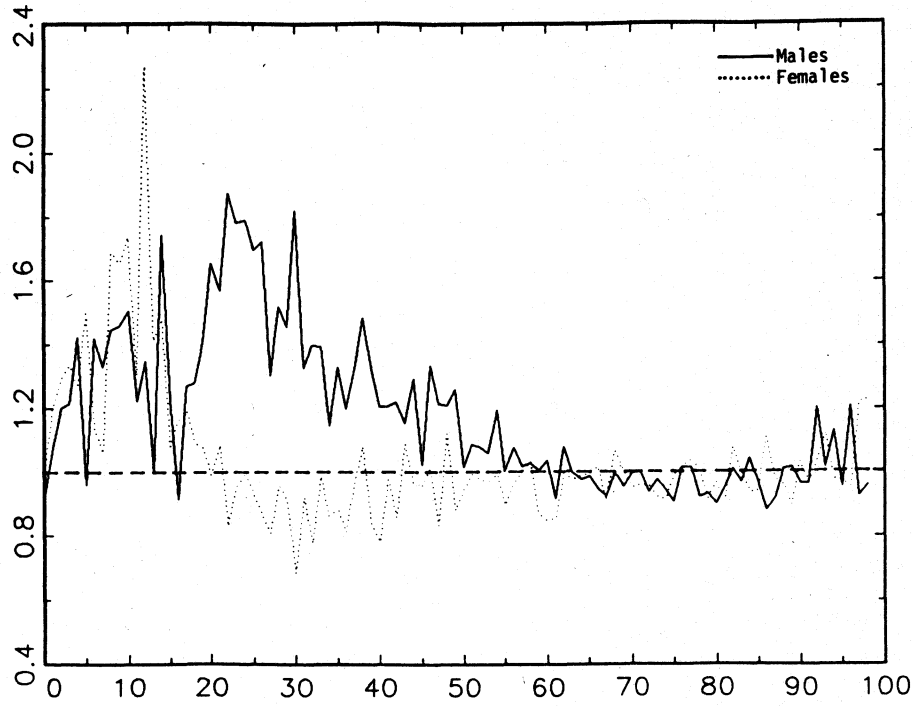


etiology of the Spanish flu and the Second World War, the onslaught of these nocuous events hit selectively by age, sparing persons of 55 years and over. Related to this is the nearly linear trend of the graphs until 1940, to the point of almost effacing the logistic shape common in the other figures. At younger ages, the Spanish flu and the Second World War 'hastened' the mortality declines already in course before 1918 and during 1920–1940. In contrast, at ages largely spared by these events, the pace of the decline is rather gradual and steady. Finally, after a trend in which the relative disadvantage of males seems constant or slightly narrowing the sex gap, the male decline abruptly arrests after the War. During the fifties and sixties even mortality increases occurred for males at ages higher than 55. Only after 1970 a tenuous reversal of this tendency is noticeable.

In sum, it is clear from Figures 1 to 5 that the 'mortality transition' in Norway is the result of a number of complex processes affecting the decline in a marked differential way by age and sex. In general, all the age-specific probabilities decrease, but they do so in quite discrepant and some times unsystematic manners. Particularly salient is the disrupting effect of the two major 'shocks' noticed, the pandemic flu of 1918 and the Second World War. To appreciate the age and sex selectivity of these two onslaughts, Figures 6 and 7 show ratios of 'crisis' versus 'normal' probabilities of death $q(x)$.[9] It is apparent that the flu epidemic hit predominantly young and young adults (ages 15 to 40) with no trace of sex differential effects, while the Second World War hit predominantly young adult males (aged 20 to 30) and children in

[9]The 'normal' probabilities of death are the average q_x values for the years 1914–1917 in Figure 6, and 1937–1939 in Figure 7. The corresponding 'crisis' years are 1818 and 1940–1942.

Figure 7: Age and Sex Profile of Excess Crisis Mortality: 1940–1942



school ages. In both cases, the impacts are restricted to the population under age 50.

The magnitude of these two 'crises' and their marked age and sex differentials lead us to suspect that some cohort traces—in the form of selection or debilitation effects—may be of no negligible importance in a thorough account of mortality by age and period. A simultaneous inspection of age, period and cohort effects is, thence, called for.

5 Model Selection and Major Results

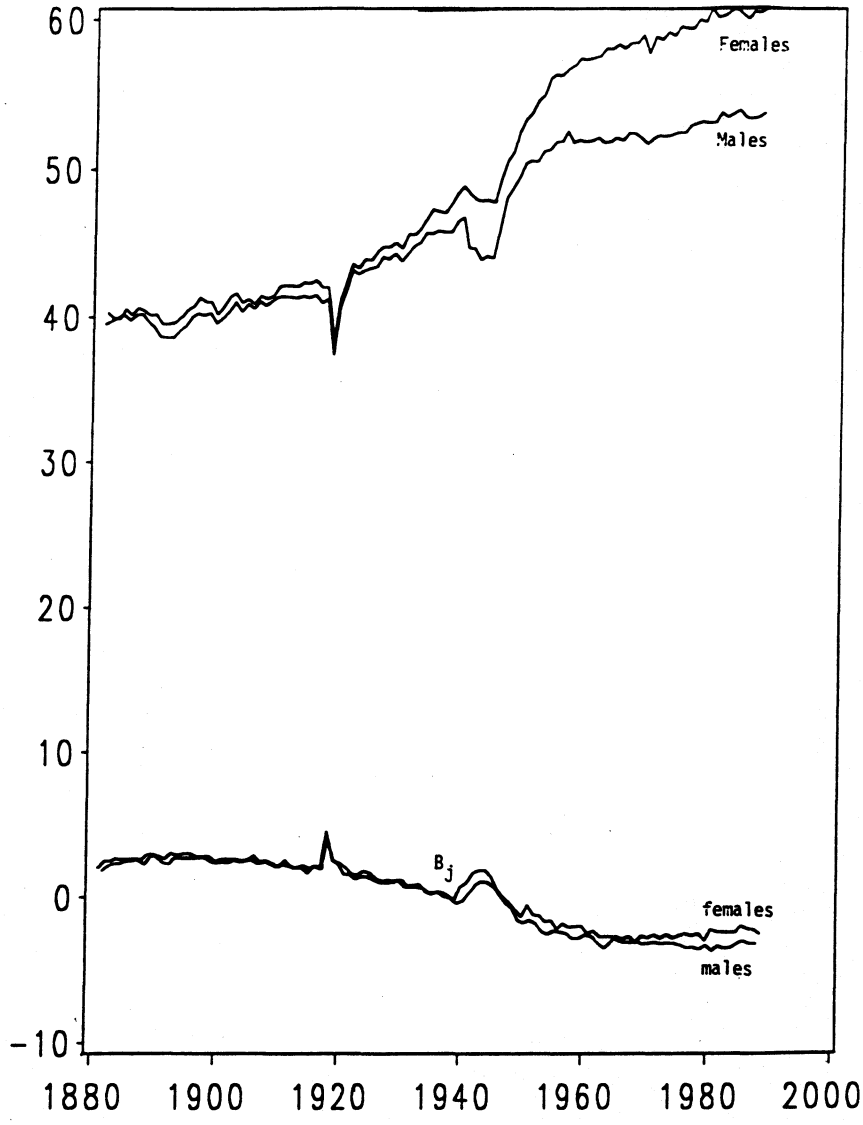
To facilitate our review of the findings we decided, for reasons that should become clear shortly, to split the material into three: results based on the span 1881–1988, results based on the middle span 1918–1956, and results based on the more recent span 1965–1988. We draw only briefly upon the results of the first since, comparatively, the latter two yield more relevant information for the purposes of this exercise.

5.1 The span 1881–1988

Figure 8 shows the time trend vectors A_j and B_j that result from fitting the double multiplicative model $y_{ij} = \alpha_i A_j + \beta_i B_j$ to the respective male and female matrices of age-specific mortality rates over the period 1881–1988.[10] The fit is rather satisfactory

[10]In line with their diagnostic properties, the additive and double multiplicative models were fitted first in our analytic strategy. Results of the additive model are omitted, however, as they only point

Figure 8: Male and Female Time Trend Parameters A_j and B_j of the Double Multiplicative Model, 1881–1988.



for each sex: the goodness-of-fit statistic P is 92.5 percent for males and 91.6 percent for females, the residuals are fairly Gaussian in both cases, and no trace is apparent of a grossly ill-specified model. We concentrate first on A_j, which summarizes the time trends referred to above.

The upward slopes indicate fairly parallel decreasing mortality for the two sexes until 1918. After the Spanish flu and up to 1940, the female gradient is only slightly higher than the male gradient. Then, after the War, it becomes much steeper, notably between 1945 and 1955. Thereafter, an almost linear trend follows for each sex, with females gaining ground more rapidly than males.[11] The crisis years of 1918 and 1940-44 (visible here as notches) are highly prominent. In Figure 8, the flu and the war appear—more clearly so than in Figures 1 to 5—as events that catapult the decline after their respective impacts. This evokes immediately the plausibility of selection mechanisms, most certainly combined with gains in sociomedical and sanitary progress.

Now, the vector B_j (first differences from A_j after controlling for $\alpha_i A_j$), besides being almost indistinguishable for each sex until 1965, clearly is of considerably lower magnitude than A_j. In addition, it shows a slope not substantially different from zero. All these features stand out suggestively as a hint to the possibility of collapsing the time trend 'sufficient statistics' of the model into only one vector—a slightly modified A_j—by forcing B_j to become zero. The result will be a most desirable one: to simplify to the minimum the task of extrapolation by having merely to project a single parameter. This point is returned to in subsection 5.3. Before that, we turn briefly to inspect the residuals of the double multiplicative model.

Figure 9 shows a segment of the table of coded residuals of model (7) fitted to the male rates over the span 1881–1988. The selected segment goes from 1915 to 1960 and shows what seems an instance of cohort effects: a corridor of positive residuals for a group of about ten cohorts, aged 12 to 20 years in 1918 and 34 to 42 in 1940. The trace is noticeable only after 1918 and runs—though gradually fading out—until shortly after the end of the War. They seem to be the product of the influenza epidemic. In a less striking manner but still eye-catching enough, a cloud of negative residuals stretch along the cohorts under 5 years of age in 1918 and those born shortly after.[12] In view of this evidence one is left with the riddle whether the Spanish flu produced selection effects for the cohorts hit at young ages while it produced debilitation effects on the cohorts aged, say, 10 to 20 years in 1918. To belabor this question exceeds the purpose of this paper, but we take brief pause to

---

out the pertinence of the logarithm transformation to enhance structure in the data. Thence, except otherwise stated, in all references to y_{ij} the transformation $y_{ij} = \ln m_t(x)$ is assumed, holding $x = i$ and $t = j$.

[11] Further down in Section 5.3 we delve somewhat more deeply into the finer details of the trends in recent years.

[12] These two features are also noticeable in female cohorts, notably the cloud of positive longitudinal residuals between 1918 and 1940.

Figure 9: Coded Table of Residuals, Double Multiplicative Model, Males, 1881-1988.

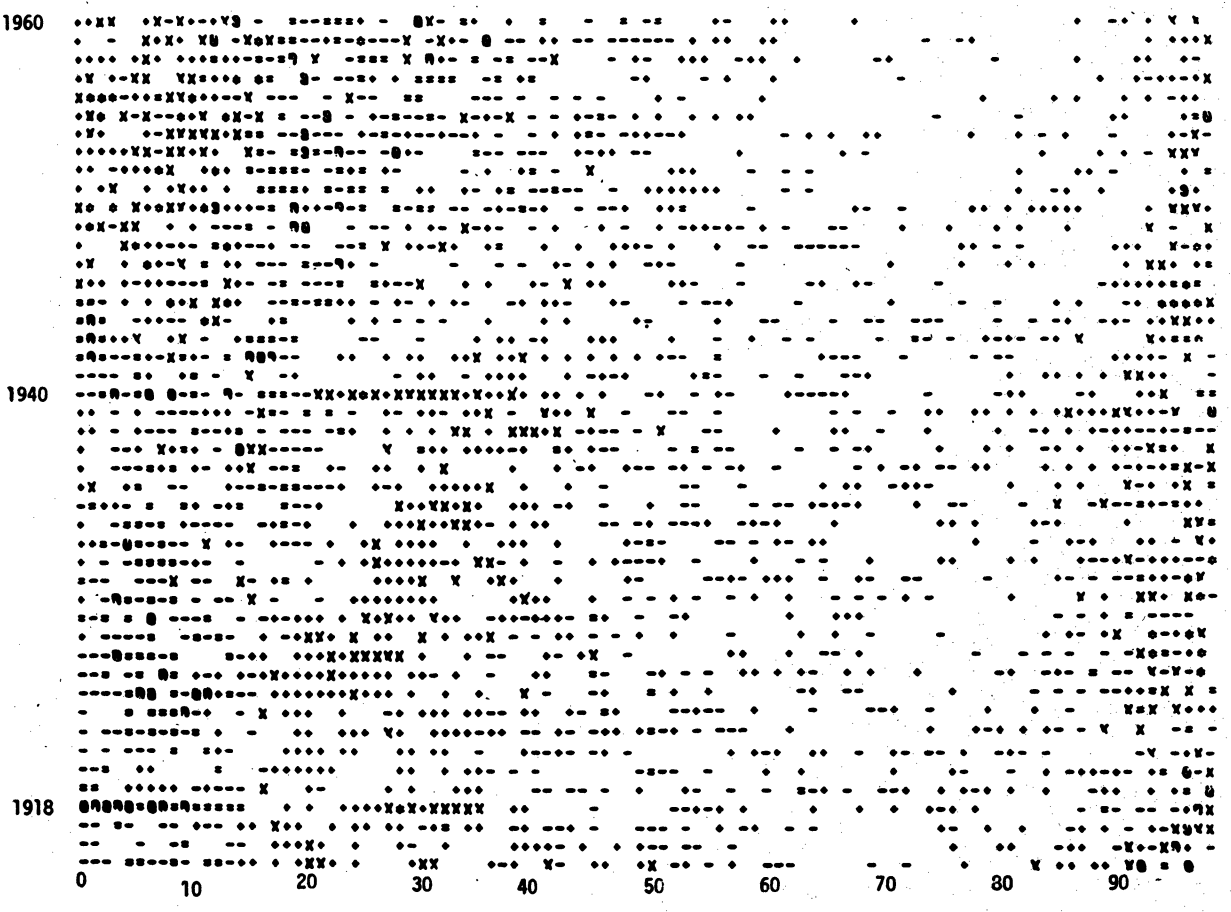
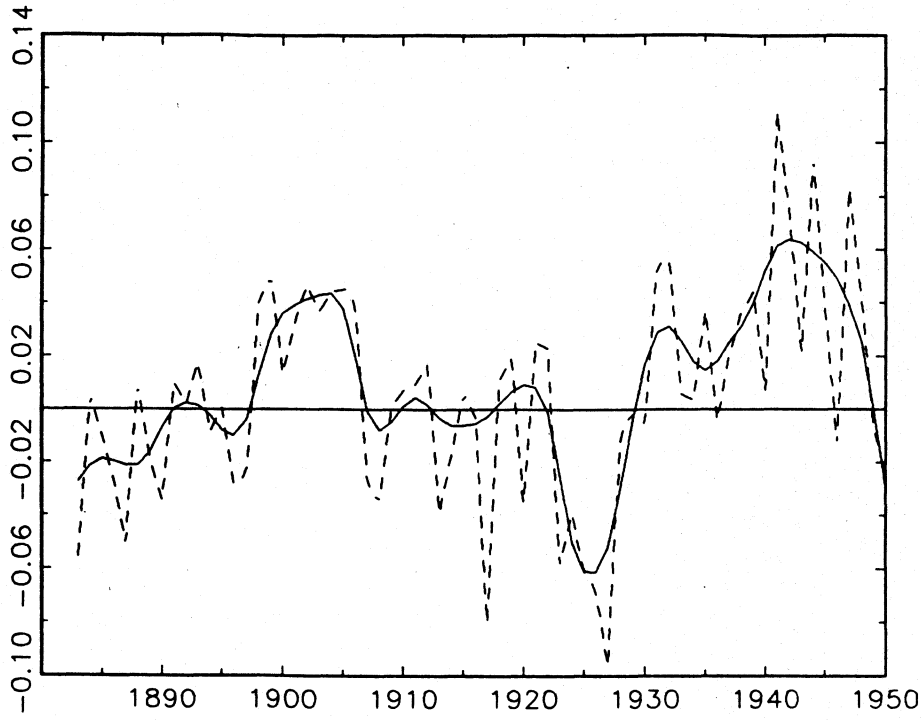


Figure 10: Cohort Effects from the Double Multiplicative Model, Males, Selected Cohorts Born 1883–1950.



examine somewhat closer the cohort traces.

5.2 The span 1918–1956

Figure 10 shows the cohort effects resulting from the residuals of the double multiplicative model[13] when fitted to the male rates in the span 1917–1956, restricted to ages up to 60 years. In all 100 cohorts cross the rectangle, but some are observed only at a few years. The graph shows a selection running from the 1883 cohort (observed 26 years between ages 35 to 60) to the 1950 cohort (observed 7 years from birth). Only the central cohorts in the graph—those born between 1896 and 1918—are observed for the same number and range of ages (from birth to age 60).

It seems apparent indeed that some groups of cohorts have high or low *relative* mortality among the cohorts shown. We notice immediately the group of cohorts singled out before in Figure 9. It comprises the generations 1898–1906, the adolescent cohorts in 1918.[14] According to Figure 10, the conjecture of the Spanish flu

[13]These are location M-estimates of the diagonal vectors of the matrix of residuals z_{ij}. They measure higher or lower mortality between cohorts in *relative* terms, as they are constrained to sum zero.

[14]One is tempted to call these *debilitation* effects from the Spanish flu but, without a clear understanding of the nature of the mechanisms involved, it is adventurous to attribute them directly to any particular form of determinacy. A late wave of emigration that peaked in 1923 further complicates the issue, as most probably migration introduced additional selection, in itself dependent on the effects of the Spanish flu. Curiously enough, Wilmoth *et.al.* (1989) identify excess mortality in France for practically the same male cohorts (1894–1904) but they attribute this phenomenon to long term

introducing selection effects in the young cohorts alive in 1918 seems not tenable. In turn, the low mortality of the cohorts 1923–1927 is somewhat puzzling. A detailed inspection of these residuals along age confirms that the cohort effect is driven preeminently by low mortality at ages under five. Thus, a direction to look at for possible explanations can be to study parental changes after the flu that may have resulted in less frail birth cohorts. To account for the cohort effects certainly requires further scrutiny. Without supplemental information it is hard to depart here from mere speculation.

Now, perhaps not too much should be read into the results of Figure 10. The excess mortality of the cohorts born under the War, for instance, is based on cohorts observed only during a few young ages, at which, in fact, higher relative mortality is apparent. This cohort trait disappears when the cohort is observed for a longer period. Similarly, the high mortality of the adolescent cohorts in 1918 is the result of high mortality between 1918 and 1940, but, afterwards, this relative excess dwindles down without any noticeable compensation to balance the sign.[15] At any rate, drawing upon the arguments above and our discussion of Figure 10, one conclusion seems to stand sufficiently firm for the purpose of mortality projection: by 1988—the launch date of the projection—the cohort effects alluded to before have by then disappeared. It seems safe, therefore, to completely dispense with modelling cohort effects when carrying out the projection.

5.3 The span 1965–1988

In Figure 8, starting about 1965, a nearly linear trend was noticed in the time parameters A_j and B_j for both sexes. Based on this finding, we concentrate the rest of our analysis on the span 1965–1988. The trend after 1965 appears to be the stabilization of gradual mortality changes after the war. It seems thus a convenient choice to base the projection on this recent span. At the same time, drawing from the results of subsection 5.2 above (in the sense that cohort effects are indiscernible after the fifties) the rest of our analysis is based on two major modifications of the procedures used so far: first, the data were smoothed to remove random variation in the age-specific rates[16]; second, the resistance leverage constant c in equation (8) was set to $c = 100$, to bring the parameter estimates close to least squares estimates. Not having to concern about cohort effects these changes seem a sensible way to proceed.

Tables 1 and 2 show some goodness-of-fit measures from fitting the double multiplicative model to the respective series of male and female age-specific mortality rates

effects of the Second World War instead of the flu.

[15] In addition, the overall magnitude of the cohort effects—the abscissa values in Figure 10—is of no prominent importance when compared to the age and period effects. We show in short values of α_i, β_i, A_j and B_j that allow comparison.

[16] The procedure 4253EH(twice) proposed by Velleman and Hoaglin (1981, Chapter 6) was used. It smooths by repeated running medians of 2, 3, 5, 3; smooths the end points and finally smooths by *hanning*.

Table 1: Goodness-of-Fit Measures, Double Multiplicative Model, Males, 1965–1988

```
% Reduction in Var   96.847
LS-Goodness of fit   99.979
Letter-Value Display
   Lower     Upper     Mid      Gaussian shape
  -0.033     0.036     0.001     0.051
  -0.069     0.073     0.002     0.062
  -0.112     0.110    -0.001     0.072
  -0.159     0.144    -0.007     0.081
  -0.207     0.181    -0.013     0.090
  -0.282     0.231    -0.025     0.106
  -0.370     0.290    -0.040     0.124
  -0.432     0.364    -0.034     0.138
  -0.487     0.396    -0.046     0.143
  -0.517     0.424    -0.046     0.143
  -0.566     0.461    -0.052     0.147

  Median     0.002
  Spread     1.027
  SADMed  3697.190
  Sum|z|   116.591
Sum|w.z|   116.080
```

during the period 1965–1988. In both cases, the proportion of variance explained by the model indicates a very good fit: $P_m = 96.8$ percent and $P_f = 95.4$ percent, for males and females, respectively.[17] The residuals are fairly concentrated and show practically Gaussian shape. The resulting parameter estimates α_i, β_i, A_j and B_j are displayed jointly as biplots in Figures 11 and 12.

The most prominent feature in the graphs is the striking collinearity of the column markers, which unmistakably suggest the pertinence of the *rows-linear* model

$$y_{ij} = \tau + \alpha_i + \beta_i B_j + z_{ij},$$

according to the biplot diagnostic rules. This is confirmed in addition by Figure 13 where the time trend parameters A_j and B_j clearly suggest that one of the parameters, A_j, can be reduced to a constant. Before shifting to the rows-linear model we examine briefly some of the results from the double multiplicative model.

A number of issues attract attention in Figure 13. Particularly salient is the fact that the male and female trend parameters share approximately the same slopes for both A_j and B_j. The gap difference in the A's captures, to some extent at least, the different mortality 'levels' between the sexes. More puzzling appears the fact that the male and female B's run virtually on top of each other when we know that gains in life expectancy at birth have been higher for females than for males

[17] The accompanying least squares goodness-of-fit measures R^2 indicate a virtually perfect fit. These high values are not unusual when fitting data characterised by a very high degree of structure, like the mortality rates under analysis here. Hobcraft and Gilks (1984) obtain comparable results from fitting a time series of Swedish mortality rates.

Table 2: Goodness-of-Fit Measures, Double Multiplicative Model, Females, 1965-1988

```
% Reduction in Var  95.426
LS-Goodness of fit  99.962
Letter-Value Display
   Lower     Upper     Mid     Gaussian shape
  -0.051     0.056     0.003    0.079
  -0.114     0.112    -0.001    0.099
  -0.184     0.170    -0.007    0.116
  -0.246     0.219    -0.014    0.125
  -0.303     0.308     0.002    0.142
  -0.349     0.388     0.020    0.152
  -0.510     0.445    -0.033    0.180
  -0.547     0.473    -0.037    0.177
  -0.639     0.555    -0.042    0.193
  -0.705     0.594    -0.055    0.197
  -0.713     0.614    -0.050    0.190

  Median     0.003
  Spread     1.327
  SADMed  3933.779
  Sum|z|   179.917
Sum|w.z|   179.277
```

Figure 11: Biplot Display of the Row (α_i, β_i) and Column (A_j, B_j) Parameters of the Double Multiplicative Model, Males, 1965–1988

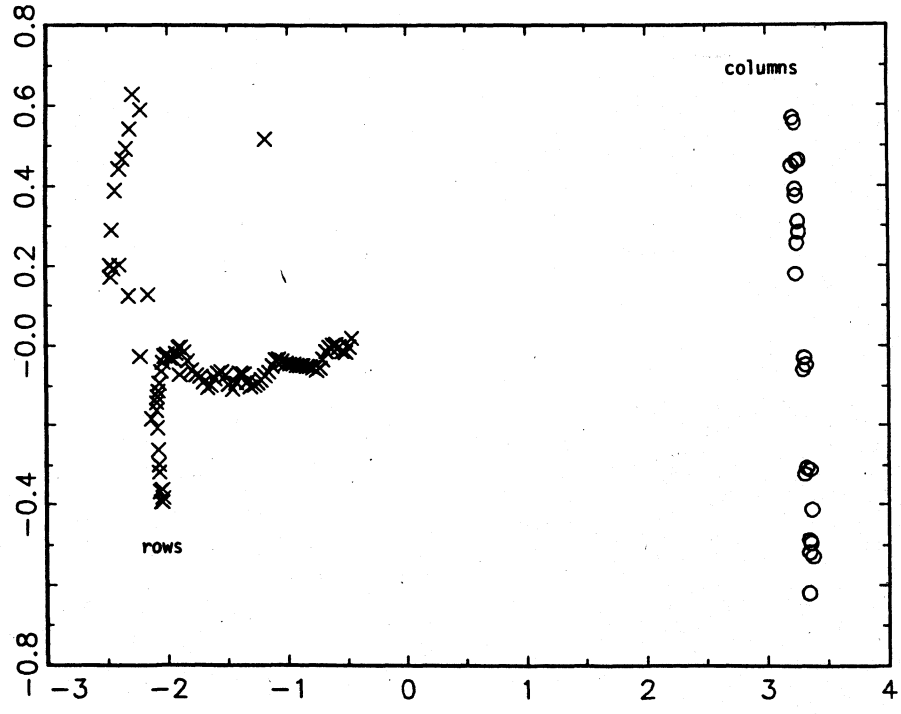
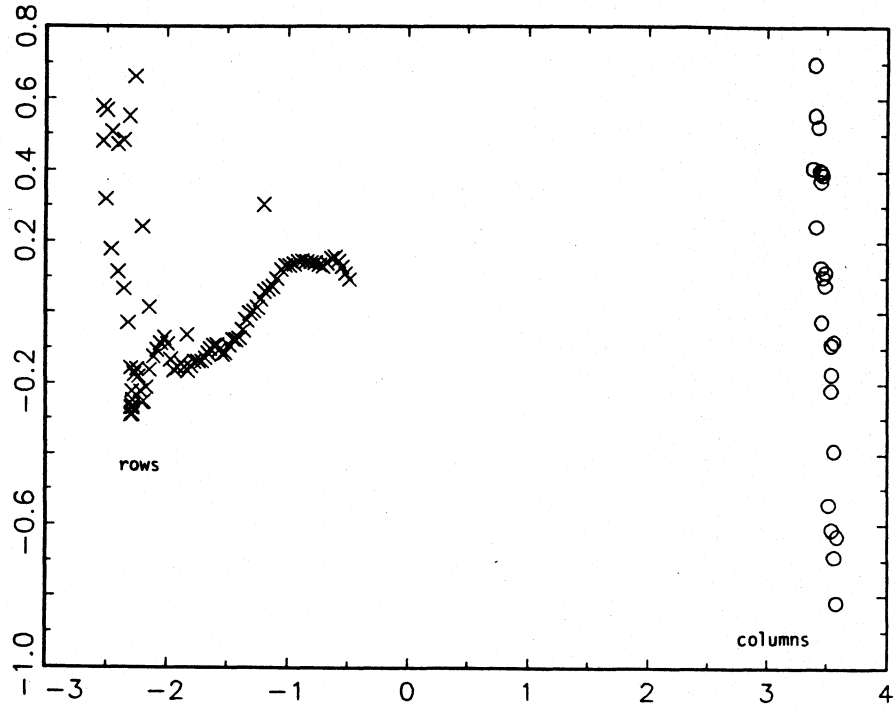


Figure 12: Biplot Display of the Row (α_i, β_i) and Column (A_j, B_j) Parameters of the Double Multiplicative Model, Females, 1965–1988



during the period. The answer lies in looking at B_j in conjunction with β_i, as we demonstrate in short. But here it is convenient to inspect the extent of linearity in B_j—the parameter to extrapolate when carrying out the projection—benefiting from the higher resolution of Figure 13 compared to Figure 8. For males, the B_j values seem to follow a fairly linear trend over the whole span except after 1986. For females, a slightly kinked line seems to divide the span into two segments, one from 1965 to 1976, and the other from 1976 to 1988, though the second is hard to determine as marked faltering obliterate the trend. At any rate, looking at the two lines together it is clearly possible to discern a common trend, which seems roughly captured by a straight line.

Figure 14 shows the corresponding age parameters α_i and β_i of the fit. The α's do not need any comment: they represent the underlying age-specific hazard rates for each sex. They conform to the expected profile of differential mortality by age and sex. The two curves are somewhat brought together by the fact that the A's standardize for overall sex differentials in mortality, as indicated before. The β's represent a pattern of first differences from the α's, that is, they constitute a schedule of age-specific mortality variation across the period. Further down we elaborate in more detail the interpretation of α_i. One point worth making at this stage, however, is that the slight male and female differences in α_i noticeable in Figure 14 is what accounts for the fact that the respective trend parameters B_j in Figure 13 show no marked difference for each sex. In other terms, the β's take into account age- and sex-specific differences that bring the sex trends close to each other.

Figure 13: Trend Parameters A_j and B_j of the Double Multiplicative Model, Males and Females, 1965–1988

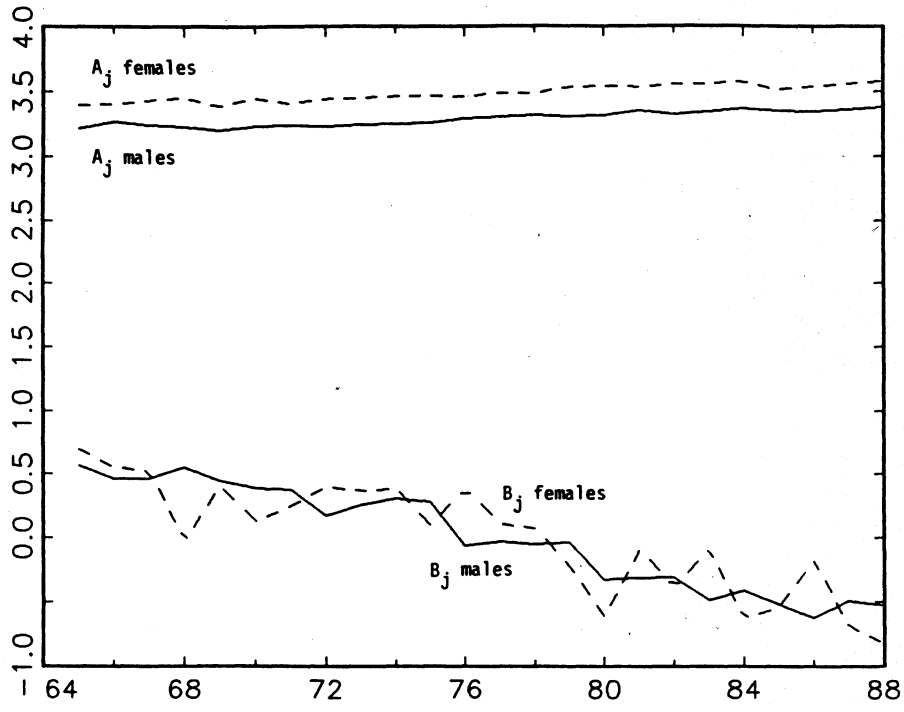


Figure 14: Age Parameters α_i and β_i of the Double Multiplicative Model, Males and Females, 1965–1988

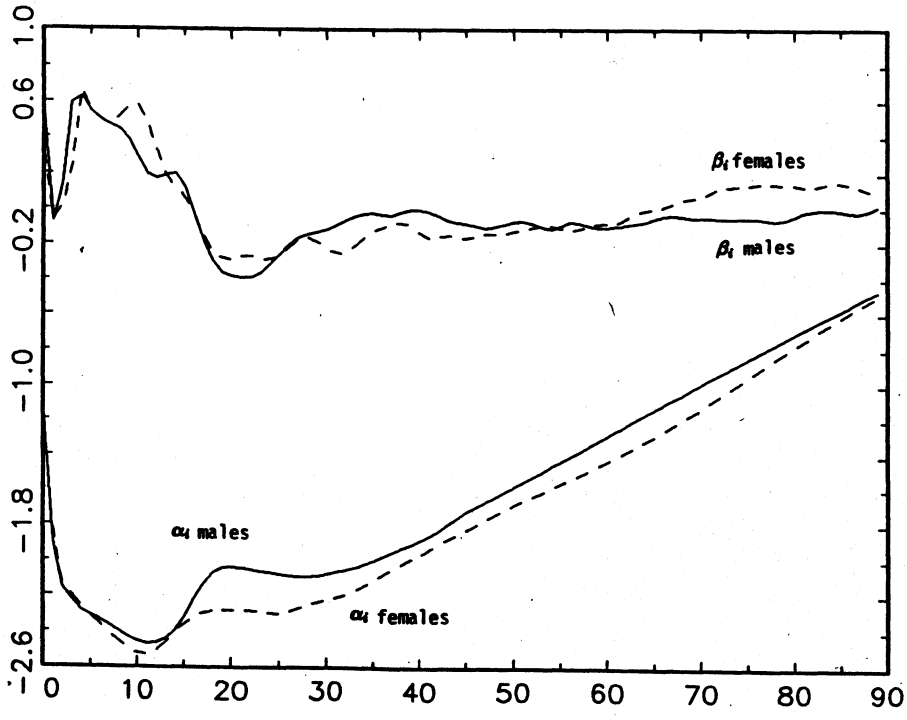


Table 3: Goodness-of-Fit Measures, Rows-linear Model, Males, 1965–1988

```
% Redn in Var  96.745
Letter-Value Display
   Lower     Upper     Mid      Gaussian shape
  -0.037     0.032   -0.002      0.052
  -0.074     0.073   -0.000      0.064
  -0.116     0.117    0.001      0.076
  -0.161     0.151   -0.005      0.084
  -0.222     0.188   -0.017      0.095
  -0.281     0.244   -0.019      0.109
  -0.379     0.287   -0.046      0.125
  -0.422     0.348   -0.037      0.134
  -0.460     0.386   -0.037      0.137
  -0.537     0.422   -0.058      0.145
  -0.564     0.476   -0.044      0.149

  Median    -0.003
  Spread     1.040
  SADMed  3697.190
  Sum|z|   120.346
Sum|w.z|   119.818
```

We now turn to the model diagnosed by the biplot: the *rows-linear* model. Tables 3 and 4 show the respective male and female measures of goodness-of-fit. Compared with the corresponding values for the double multiplicative model, the rows-linear fit is hardly an inferior one, despite the fact that the degrees of freedom went up by 23. The proportion of variance explained by the fit is virtually the same ($P_m = 96.7$ and $P_f = 95.3$) as well as the spread and the distribution of the residuals. This comes as no surprise after the structure revealed by the biplot. Thus, Tables 3 and 4 corroborate that the rows-linear model captures sufficiently well, with one single term, B_j, the age and period mortality variation displayed by the data. The rest of our analysis exploits the results of this 'preferred' model.

Let us concentrate first on the time trend B_j. Figure 15 shows the corresponding estimated values for males and females, which, with minor differences, seem like a replica of Figure 13. Here it seems somewhat clearer that a straight line captures the trend for males from 1968 to 1988. For females the situation is less clear as it seems that a new pace in the trend sets in starting about 1980. At any rate, the two trends run fairly parallel to each other. A resistant regression through the span 1965–1988 yields a common slope equal to $b = -0.03$.

The corresponding male and female α_i and β_i parameters are shown in Figure 16. The α's hardly need any comment: they reflect the average (across time) male and female age-specific mortality rates adjusted from their respective overall means, $\tau_m = -5.232$ and $\tau_f = -5.909$. Similarly to the double multiplicative results, the rows-linear α_i schedules conform to the characteristic outline of mortality differentials

Table 4: Goodness-of-Fit Measures, Rows-linear Model, Females, 1965-1988

```
% Redn in Var  95.312
Letter-Value Display
   Lower     Upper     Mid      Gaussian shape
 -0.059     0.051   -0.004      0.081
 -0.112     0.113    0.001      0.098
 -0.187     0.171   -0.008      0.117
 -0.259     0.233   -0.013      0.132
 -0.312     0.321    0.005      0.147
 -0.408     0.383   -0.013      0.164
 -0.516     0.449   -0.033      0.181
 -0.620     0.494   -0.063      0.193
 -0.723     0.508   -0.107      0.199
 -0.763     0.543   -0.110      0.198
 -0.794     0.588   -0.103      0.198

  Median    -0.005
  Spread     1.382
  SADMed  3933.779
  Sum|z|   184.429
Sum|w.z|   183.692
```

Figure 15: Trend Parameter B_j of the Rows-linear Model, Males and Females, 1965–1988

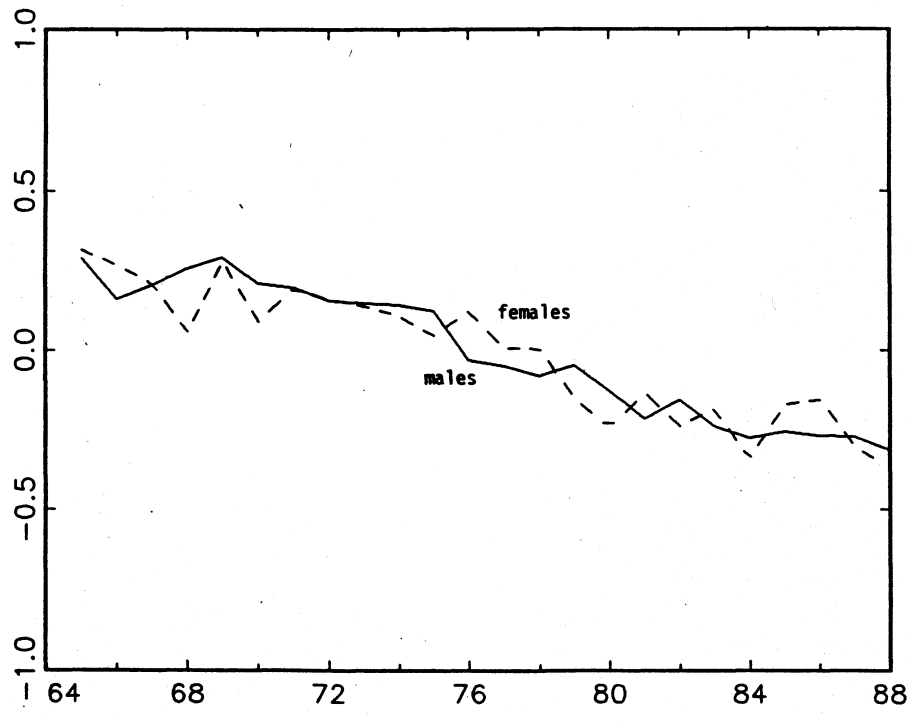
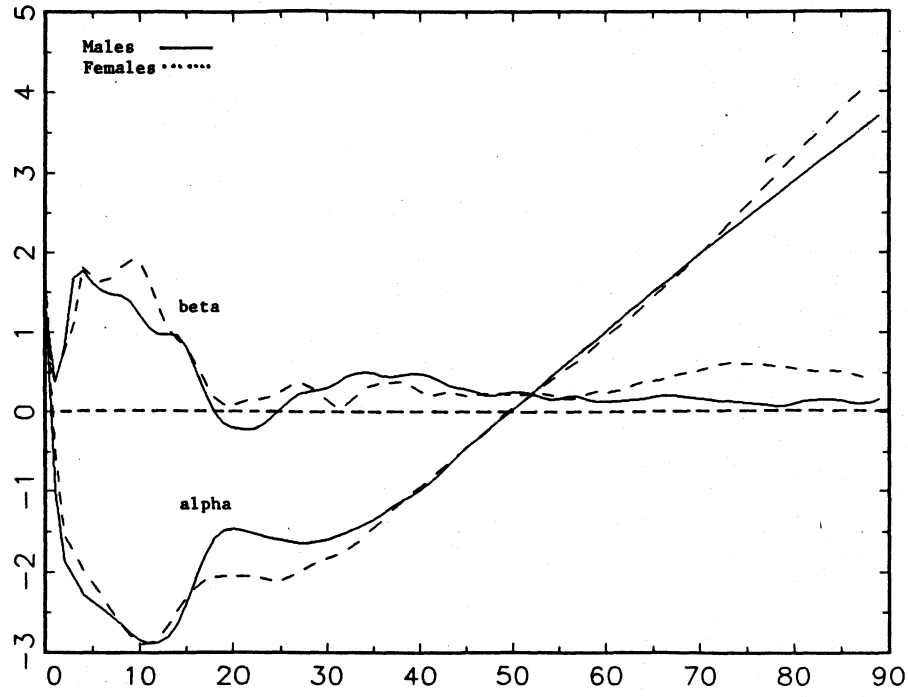


Figure 16: Age Parameters α_i and β_i of the Rows-linear Model, Males and Females, 1965–1988



by age and sex.[18] In contrast, the β's show a somewhat haphazard behavior around a pattern that, at first sight, does not lend itself easily to interpretation. To clarify their meaning it is useful to rewrite the rows-linear model as a special case of the additive-plus-multiplicative model:

$$y_{ij} = \tau + \alpha_i + B_j + (\beta_i - 1)B_j + z_{ij}$$

where the $(\beta_i - 1)$ stand as regression coefficients of the rows residuals $z_{i\cdot}$ from the additive model onto B_j. It becomes clear, then, that the β's represent a pattern of first order deviations from the *standard* α_i, where the deviations are quantified as slopes $(\beta_i - 1)$ vis-à-vis B_j. The negative β values for males at ages 18–24, for instance, indicate relative mortality deterioration in that age segment (attributed to increased fatal motor vehicle accidents). The β's in Figure 16 delineate thus sex differentials in the age-specific pattern of mortality change during the span 1965–1988.

6 An Illustrative Mortality Projection

In what follows we show a tentative projection of age- and sex-specific mortality rates for Norway to the year 2010. We consider the results of our exercise illustrative only, as eventually more judicious refinements are necessary to claim a realistic forecast.

[18] Compared to Figure 14, the male and female α_i in Figure 16 are 'brought together' as the rows-linear model adjust the α's from their respective means, τ_m and τ_f.

The gist of the projection rests on extrapolating the male and female trend values B_j, holding constant the age standard α_i and the schedule of deviations β_i. Several more or less complicated projection alternatives are open. We use here perhaps the simplest: a (robust) linear regression extrapolation based on the whole span 1965–1988. In Section 5 we deemed the B_j trends linear enough while eye-inspecting Figure 15. This is largely confirmed by the regression, where only a few points receive slightly reduced weights. The projection simplifies then to extrapolate into the gap the linear trend of the B_j. As noted before, despite the fact that the models were estimated separately for males and females, the secular trend for each sex seems well represented by a common slope.

Before looking at the results, a minor adjustment needs to be described. In order to free the projection from the deviating male mortality around age 20—as it is unlikely that this injurious element will remain unchecked—, the corresponding β_i values were adjusted to muffle out any mortality deterioration.[19] Other than the mere extrapolation of the estimated patterns, this is the only adjustment that seemed pertinent. Figures 17 and 18 show the projected rates—five years apart and for males and females, respectively—reported in a special fashion: as proportional differences from the 1980 rates.[20] On the whole, the resulting graphs merely spell out perfunctorily what expected. For males, mortality rates decline ostensibly at ages between 3 to 17, and between 30 to 50 years.[21] Females show declines just as large as males in the school ages (if not slightly larger), and more marked declines than males at ages over 55. Centered at age 31, a curious 'hindrance' thwarts somewhat the female decline in the young adult ages. In the absence of any satisfactory explanation for this fact, it is taken as a peculiarity of the data.

Figure 19 shows the projection in terms of the more familiar life expectancy at birth. Here again the pattern is just the mere confirmation of what expected: a gradual progression of the observed trend in the past. The *target* e_0 is 74.62 for males and 81.82 for females. These represent respective life expectancy increases of 1.6 and 2.2 years from the *launch* year 1988. Clearly, the projection could have been extended beyond the year 2010 along the same principles, or modifying the slope or the shape of the B_j trends, as judged pertinent. In this illustration, rather than to focus on the plausibility of particular mortality projections, we point out the merits of the proposed method.

[19]The adjustment consist of smoothing the β's around age 20 in order to impede negative values to occur. Altogether, the adjustment is rather minor.

[20]The baseline rates are the 1980 rates obtained from the linear fit to the B_j and the α_i and β_i parameters. In all, 30 years of mortality are reconstructed, but only the last 20 represent projected mortality proper.

[21]Male mortality around ages 18 to 25 is checked from deterioration, as explained above.

Figure 17: Reconstructed and Projected Mortality Rates 1985–2010 as Proportion of the 1980 Rates, Males

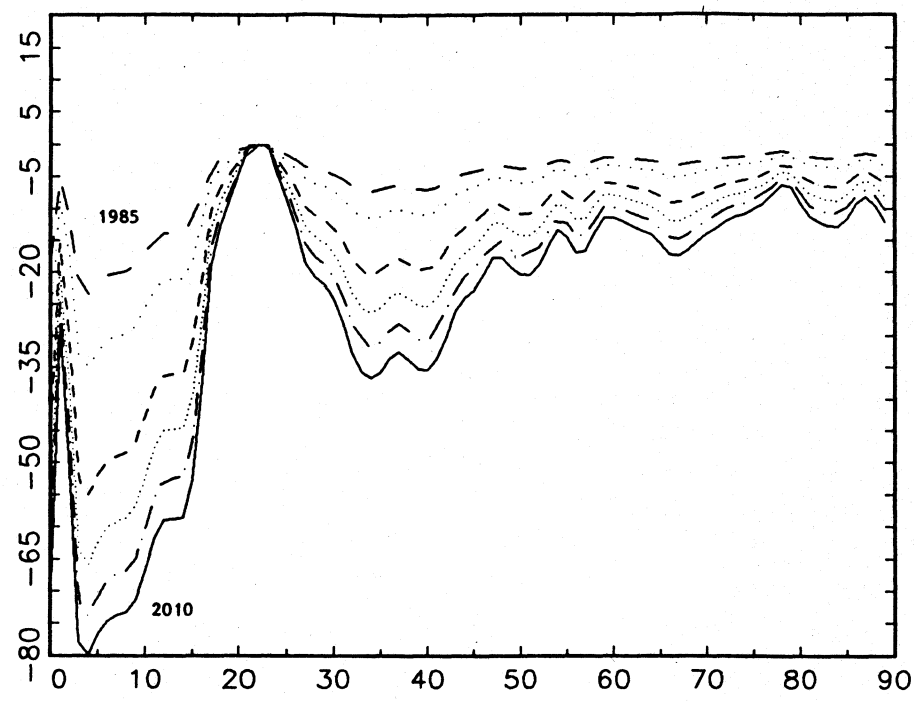


Figure 18: Reconstructed and Projected Mortality Rates 1985–2010 as Proportion of the 1980 Rates, Females

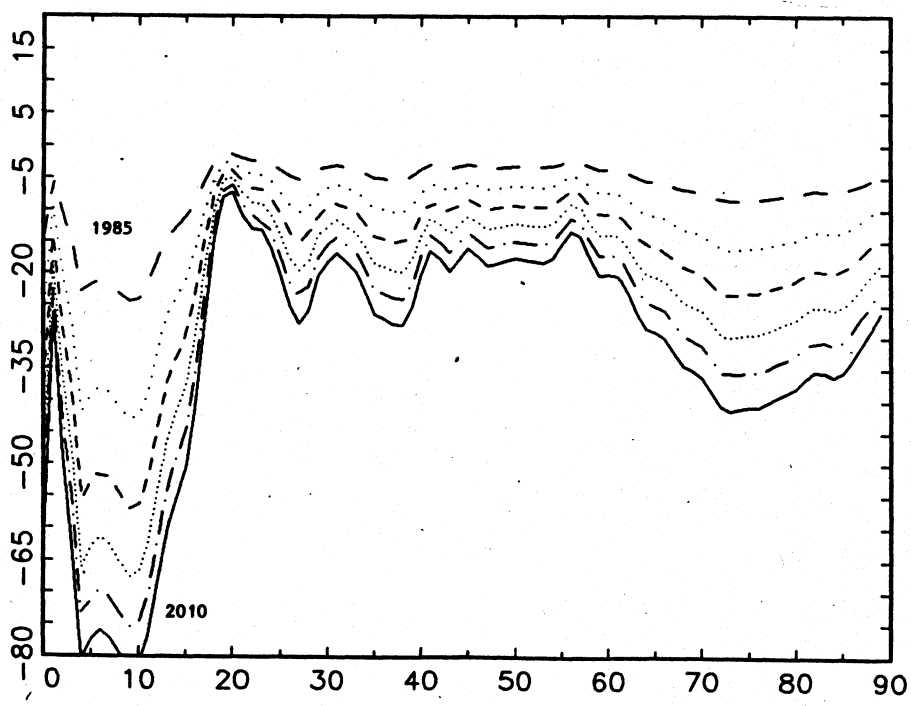
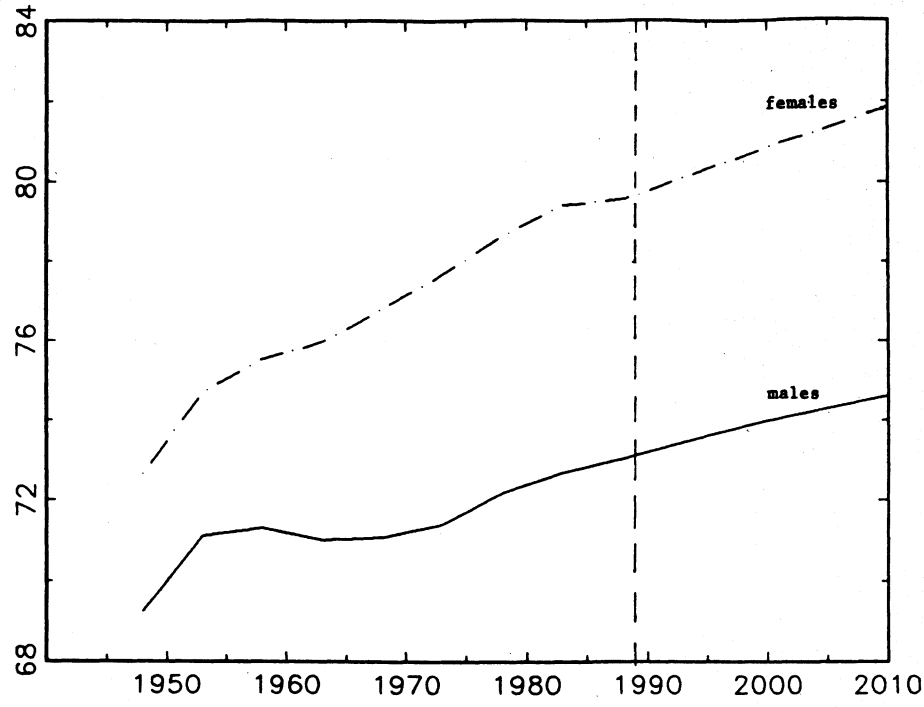


Figure 19: Recorded and Projected Life Expectancy at Birth, Males and Females, 1950–2010



7 Discussion

To conclude, we recapitulate succinctly the most salient result in this exercise. We give convincing evidence that a relational model of the form

$$\ln \mu(x) = \tau + \alpha(x) + B \cdot \beta(x),$$

accomplishes adequately the task of fitting a large time series of age-specific mortality rates. The age standards $\alpha(x)$ and $\beta(x)$ represent, respectively, a baseline hazard function and a pattern of common deviations from the basic standard. The constant τ is an overall scaling factor (that can be subsumed in the α's) and B is a parameter indicating a certain degree of deviation from the baseline hazard. The model is empirical in the sense model life tables are. One of its advantages is that it facilitates mortality projection by extrapolation of the sole parameter B.

The proposed model, though justified and derived along Exploratory Data Analysis considerations, is closely akin to relational models of extensive use in demography. Coale's marital fertility model is a good example (Coale, 1971). In Coale's model age-specific marital fertility is expressed as:

$$\ln f(a) = \ln M \cdot n(a) + m \cdot v(a),$$

where $n(a)$ is a 'natural' marital fertility standard, $v(a)$ is a schedule of common departures from the standard (interpreted to reflect the age pattern of conscious behavior to control fertility), M is a scale factor, and m is a parameter reflecting the

extent of fertility control (as a factor of $v(a)$). The structural form of Coale's model is virtually that of the relational model above.

Similarly, Brass (1977) and Zaba (1979) constitute examples of mortality models based on a standard and a pattern of deviations from the standard. These models, however, require four parameters—plus the standard—for every fit. In comparison, the relational model used here is essentially a one parameter model—plus two standards.[22]

Clearly a number of issues require further comment in our exercise. Other than the extrapolation of the time trend itself, perhaps the strongest assumption in the projection is whether the standards α_i and β_i serve to project the age trajectories into the future. We feel confident that, as the span 1965–1988 reflects the steady and gradual change that characterize recent mortality improvements in Norway, to base these schedules on this period is as reasonable a choice can be. However, how far ahead into the future one can reasonable 'stretch' α_i and β_i (holding them constant while B_j 'drives' the projection), remains still an empirical question.

Also, a more refined method could have been used to project the trend parameter B_j. Keeping things simple, logistic extrapolation seems a good alternative. This choice, however, bears essentially on the 'philosophy' of the projection. That is, on whether one holds the view that mortality reductions should slow down gradually in the future, or they may continue to decrease steadily at about the observed pace in the span. Observing the fact that, in general, mortality reductions have been systematically underestimated by population projections in the past, no major blunder seems to lurk in the latter view, particularly if the target lies in the not so distant future (see Vallin, 1989).

[22]It is worth observing that the dimensionality of the rows-linear fit (two age standards plus a trend vector) is the same as Brass' logit-logit model (one age standard plus two trend vectors). Brass' original model is somewhat inflexible, however, and the rows-linear model proves superior for the same number of parameters.

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