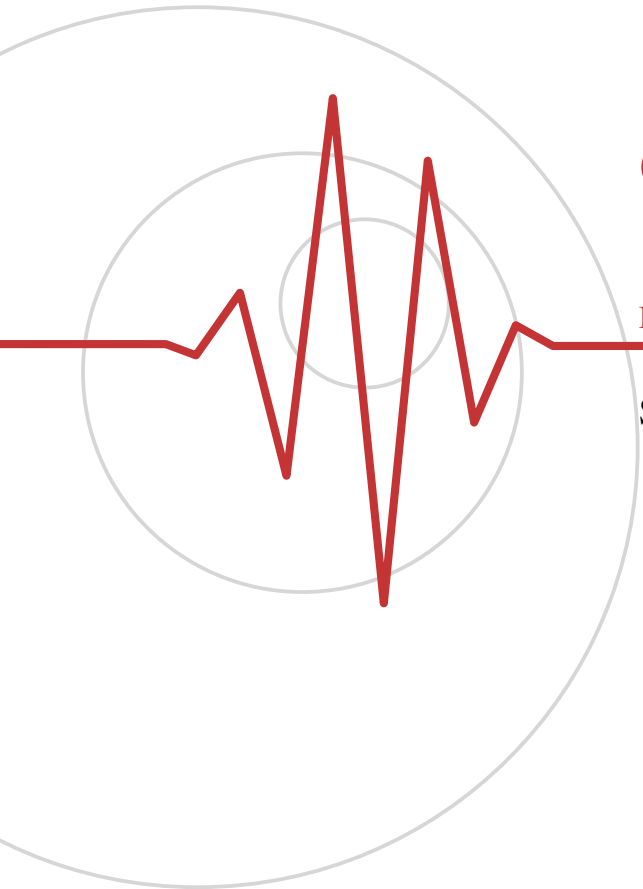


Perspectives on Mortality Forecasting

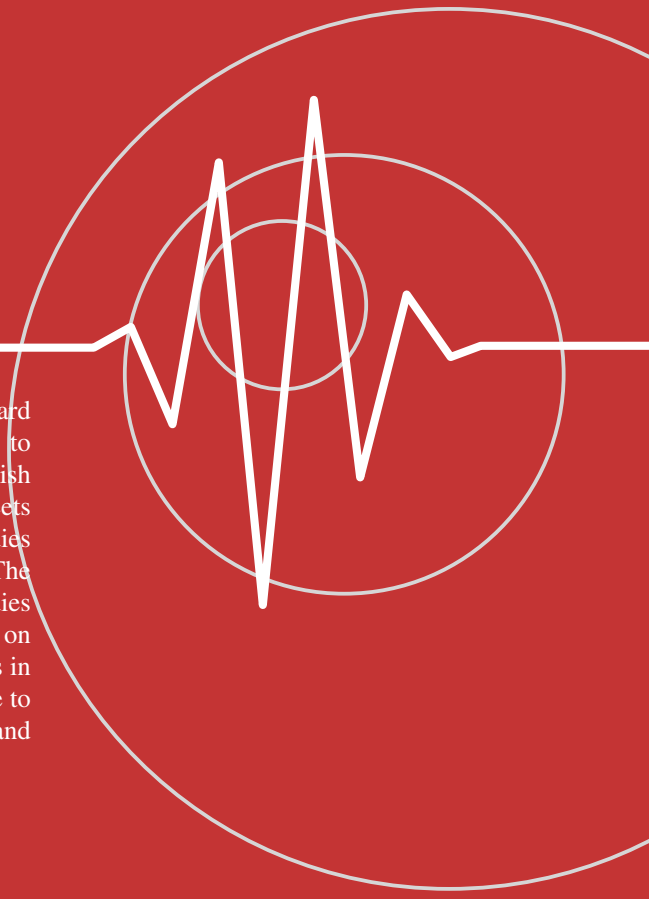
I. Current Practice

Social Insurance Studies | No. 1

Editors
Tommy Bengtsson
Nico Keilman

Swedish National Social Insurance Board

Series Editor
Edward Palmer



The Swedish National Social Insurance Board (RFV) has a long standing commitment to promote research and evaluation of Swedish social insurance and social policy. RFV meets this commitment by commissioning studies from scholars specializing in these areas. The purpose of the series Social Insurance Studies is to make studies and research focusing on important institutional and empirical issues in social insurance and social policy available to the international community of scholars and policy makers.

Perspectives on Mortality Forecasting

Published by: Riksförsäkringsverket (RFV), National Social Insurance Board, 2003

Series Editor: Edward Palmer

Issue Editors: Tommy Bengtsson and Nico Keilman

Social Insurance Studies can be ordered through the RFV homepage at www.rfv.se, by phone: +46 8 556 799 29 or by fax: +46 8 718 29 90, and cost SEK 120 excluding VAT and postage and packing.

National Social Insurance Board, RFV
Adolf Fredriks Kyrkogata 8
SE-103 51 STOCKHOLM, Sweden
Telephone: +46 8 786 90 00
E-mail: rfv.stockholm@rfv.sfa.se

Graphic Production: Kristina Malm
Cover Design: Jenny Övrebö
Printed by Elanders Novum AB, Göteborg, 2003

ISSN 1651-680x
ISBN 91-89303-24-5

Preface

Mortality projections are an essential input for projections of the financial development of pension schemes. Governments and insurance companies all over the world rely on good mortality projections for efficient administration of their pension commitments. Ideally, the expected value of the difference between outcomes and projections would be close to zero. In practice, during recent decades, demographers have continually underestimated improvements in life expectancy for persons 60 and older. The demographic models used in projecting mortality are usually based on statistical modeling of historical data. The question is, is it possible to bring the results of mortality modeling closer to the ideal, and if so, what do demographers need to do to achieve this result? This is the question that provided the impetus for forming the Stockholm Committee on Mortality Forecasting.

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

This is the first volume in a series presenting papers from workshops on mortality organized by the Stockholm Committee on Mortality Forecasting. The first paper, written by Edward Palmer, introduces the question of why we are interested in improving mortality projections. Nico Keilman covers types of models for projecting mortality and James Vaupel presents the SCOPE approach to forecasting life expectancy. Kaare Christensen discusses how to improve existing models from the perspective of an epidemiologist, and Tommy Bengtsson stresses the role of historical experience in getting forecasting right. Juha M. Alho, Helge Brunborg and Hans Lundström discuss current projection models in Finland, Norway and Sweden, respectively.

As editor of Social Insurance Studies, I am particularly pleased that the first issue in the series is on mortality forecasting because it brings together a broad range of disciplines. It is my hope that the published proceedings of the Stockholm Committee on Mortality Forecasting will contribute to a better understanding of the processes underlying increasing longevity.

Edward Palmer

Members of the Stockholm Committee on Mortality Forecasting

Professor Edward Palmer
Uppsala University and National Social Insurance Board, Sweden

Professor Tommy Bengtsson, Committee Secretary
Lund University, Sweden

Professor Juha M. Alho
University of Joensuu, Finland

Professor Kaare Christensen
University of Southern Denmark, Odense

Professor Nico Keilman
University of Oslo, Norway

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

Contents

Life Expectancy is Taking Center Place in Modern National Pension Schemes – A New Challenge for the Art of Projecting Mortality

Edward Palmer

Professor of Social Insurance Economics at Uppsala University, Sweden, and Chief of the Research Division, Swedish National Social Insurance Board

Introduction

Mortality scenarios are a standard tool in projecting costs of defined benefits (DB) pay-as-you-go pension systems. Government actuaries and statisticians have used mortality projections to estimate the costs of national pay-as-you go defined benefit pension systems since their conception, although the development of computer technology in the 1970s and 1980s was a prerequisite for more sophisticated analysis. With the maturation of computer technology, the possibilities to model and examine various assumptions are nowadays more-or-less unbounded. We have become used to official publications from, for example, the Office of the Government Actuary in the US or, in Sweden, the National Social Insurance Board, that provide a picture of future contributions and payments given various demographic and economic scenarios. These reports provide a picture of the financial development of national pension systems.

Financial projections illuminating the financial status of pension systems are indisputably important, not the least because pay-as-you-go systems transfer considerable resources from workers to pensioners. As life expectancy increases, and assuming current retirement patterns, 25–30 per cent of the total population in the OECD will be pensioners within the coming three decades. There is now considerable debate in countries all around the world about how to cope with the aging population. The World Bank's publication Averting the Old Age Crisis (1994) constituted a milestone in the debate. The World Bank recommended that its client countries adopt multi-pillar schemes, with a funded "second pillar" playing an important role.

The Swedish pension reform, first published as a proposal in Swedish in 1992, and then legislated by parliament as a framework law in early 1994, introduced the notional defined contribution scheme to the international political arena. Since the mid-1990s, there has been a large-scale conversion of national pension schemes from defined benefit (DB) to defined contribution (DC) – both notional defined contribution (NDC) and financial defined contribution (FDC). As countries convert to DC schemes, life expectancy projections are used to *calculate individual pensions* in national systems.

The purpose of this paper is to summarize the dramatic change that has occurred in the design of national pension schemes in the past ten years and to discuss the need for accurate mortality projections in this context.

Basic Pension Economics – the Role of Mortality

The financial costs of a pay-as-you-go pension system are determined by a few key parameters. In the aggregate these are the size of an average benefit relative to the average wage, the number of workers and the number of pensioners. The ratio of the latter two is the system dependency ratio. The system dependency ratio can also be computed as the number of years people work related to the number of years they are retired with a benefit. These are two sides of the same coin. The policy parameters of a country are the size of a pension and the level of the contribution rate. The demographic and economic determinants of pensions are largely exogenous to policy, although these can respond to system design.

These ideas follow from the two fundamental equations of pension economics, which provide the point of departure for an overview of the links between mortality and pay-as-you-go pension systems. Where bars denote averages, and p and w depict pensions and wages, these equations are:

$$Contribution\ \ rate = \frac{\overline{p}}{\overline{w}} * \frac{pensioners}{contributors}$$

or for the average participant in the scheme (*see e.g. Palmer* 1999)

$$Contribution\ \ rate = \frac{\overline{p}}{\overline{w}} * \frac{years\ in\ retirement}{years\ of\ work} \quad .$$

The first equation tells us that pension costs, measured in terms of the contribution rate on wages, is determined by the average benefit times the number of recipients, and the contribution base, expressed in the first equation as the product of the average wage and the number of contributors. If pension payments grow faster than contributions, the contribution rate needed to finance them increases, and vice versa. The second equation expresses the same relationship for the average scheme participant in terms of years worked and years of retirement. It tells us that more years of retirement in relation to years of work will result in higher pension costs.

Mortality affects both the numerator and the denominator of the first equation. Declining mortality among working age persons increases the size of the working age population and the (potential) labor force. Decreasing mortality of persons above the pension age increases the population of retirees. In other words, the age distribution of mortality improvements matters.

In terms of the second equation, increasing mortality from the age of retirement implies that people will have to work more years – at the same wage – to receive a benefit of a given level and with a fixed contribution rate. Conversely, if the pay-as-you-go system is designed so that a benefit is defined, for example, in terms of the number of covered years of work and contributions required to qualify for a benefit of a fixed amount, then declining mortality, *i.e.* more years in retirement, increases the contribution rate that the younger generation of workers has to pay in order to sustain the fixed level benefit over more years.

This is a static picture, however. Assume now that the labor force grows at the rate λ and productivity at the rate g, and that the average wage grows at the rate of growth of productivity. Then the first equation tells us a number of things. First, if benefits are inflation-indexed, which is a common form of indexation for pay-as-you-go systems, then positive values of productivity and labor force growth will counteract negative effects on costs of increasing longevity after retirement. During the past two decades some countries have taken advantage of this mechanism to reduce costs, by replacing wage indexation of benefits with price indexation.[1]

[1] The UK did this in the early 1980s. This has also been one of the mechanisms used by transition country governments during the 1990s to cut back on burdensome pension costs – where the immediate problem was a dependency ratio around 1.5 workers per pensioner, due to very generous pension ages inherited from the old communist regimes.

The first equation also tells us that a country can afford *wage* indexation as long as the labor force grows faster than the number of pensioners. With wage indexation, the real-valued (inflation adjusted) pension grows with the rate of increase in productivity, and productivity growth is shared between workers and pensioners.[2] Many countries have seen this as a desirable redistributional aspect of a pay-as-you-go pension system. Whether or not this level of ambition can be attained depends on the connection between the construction of the pension system and the demography behind changes in the work force and the number of pensioners.

Assume now net migration of zero and a birth rate that reproduces the population. Maintaining a fixed ratio between the average pension and the average wage through wage indexation of benefits over time requires that the positive effect on the work force of declining mortality surpass the increase in pension payments resulting from decreased mortality among the retired population.

Of course, the effect on the working population of decreasing mortality will have to be associated with sufficiently healthy years of life to make a difference. In addition, the economy must be able to employ the extra labor created in this way.[3] Employability is usually viewed as a short-run problem, however, as the growth of the working age population and labor supply are usually regarded as the long-run determinants of a country's economic growth, together with the rate of growth of productivity.

The second equation provides technical insight into how a pay-as-you-go system can be designed to adjust to changes in the life expectancy of pensioners. Given that people continue to work with the average wage, increasing life expectancy after retirement can be dealt with by increasing the minimum benefit age at the rate of change in the life expectancy of retirees. This also requires that people work and contribute during the additional time prior

---

[2] Denoting the rate of inflation by p, the *nominal* wage rate grows at (1+p) (1+g), as does the *nominal* wage base. For a fixed ratio of pensioners to contributors (the first equation) it is possible to index benefits with this factor, and maintain a constant contribution rate, since the numerator increases at the same rate as the denominator.

[3] In addition, the years added to the older population need to be (relatively) healthy years in order not to create other social costs, for example, increasing costs for health and home care of the elderly. In a broader model of the social "costs" of caring for the elderly, *e.g.*, health and home care, could be added to the numerator of the first equation by adding the cost per capita.

to a retirement age[4] that is sufficient to maintain a fixed ratio of years of retirement to years of work. Alternatively, the pay-as-you-go scheme can be constructed as a notional defined contribution scheme as discussed below.

NDC and FDC Schemes – and Life Expectancy

A notional defined contribution – NDC – scheme is the pay-as-you-go equivalent of the financial defined contribution – FDC – scheme. The difference is in the rate of return, which in the NDC scheme is based on economic growth, whereas it is a financial rate of return in the FDC scheme. In the neoclassical Golden Rule, these are equivalent in the long run. However, many current writers make the claim that FDC schemes should always be expected to yield a higher return (*e.g.*, Feldstein and Samwick 1997 and 1998). This claim is based on the observation that the financial rate of return has surpassed the rate of economic growth over the past half century in the US. Data for Sweden (*e.g.*, Frennberg and Hansson 1992) yield a similar result, at least up to the fall of the stock market in 2001–2002.

How does an NDC scheme work in practice? In the NDC public scheme, just as in any public or privately managed FDC scheme, wage earners pay contributions based on a fixed contribution rate. The value of these are accredited their accounts – this is the defined-contribution feature of the system. Contributions are paid on earnings as long as people work, and if people combine work with a pension then they continue to pay contributions on earned income and increase their pension capital accordingly. The previous year's account value is indexed annually with a nominal per capita wage index in Sweden,[5] where the system was conceived in 1992–1994. The wage sum is

---

[4] In a DB scheme where the right to a full benefit is based on a certain number of years, *e.g.*, 40, it might be necessary to increase this number to assure that the period of working and contributing also become longer as the minimum pension age is increased.

[5] In principle, a system must follow the development of contributions, i.e. the wage sum on which contributions are based, in order to maintain financial equilibrium. This means that if the per capita wage is used to index notional capital and benefits, then the system must be equipped with a brake that keeps it in financial equilibrium when labor force growth (λ) is negative. In the Swedish system a financial balance is kept that relates estimated system assets to liabilities. Ceteris paribus, if liabilities exceed assets because labor force growth is negative, both benefits and notional capital will be indexed downwards to bring the system back into equilibrium. (See Palmer 2000 and 2002 and Settergren 2001.)

used for indexation in Latvia and GDP in Italy, both of which were legislated in 1995.

The NDC annuity is calculated by dividing the value on the account at the chosen age of retirement with a factor based on unisex life expectancy at the age of retirement. In addition to this, in Sweden a real rate of return of 1.6 per cent and in Italy 1.5 per cent is calculated into the annuity. This form of front-loading is an alternative to possible wage indexation (from a lower initial level) over the lifetime. Annuities are also indexed to annual changes in prices in both countries. Annual indexation in the Swedish scheme also includes an adjustment for digressions of actual real growth from 1.6 per cent, and if estimated system liabilities exceed assets a balancing adjustment to bring the system back into financial equilibrium. This keeps the aggregate contribution rate in line with the individual contribution rate of 16 per cent.[6]

Góra and Palmer (2002) have recently made the claim that the main difference between NDC and FDC is in the nature of the "fund," which in the NDC case can be seen as a fund of bonds bearing the rate of return of the wage sum (tax base) in the economy, where it is a needless exercise to sell the bonds on the market fund, since this simply creates transaction costs. These authors also note that both NDC and FDC funds are illiquid until retirement, and that from retirement both are paid out as yearly annuities. Both FDC and NDC have the advantage that they eliminate negative externalities by creating a direct link between contributions and benefits. On the other hand, an FDC scheme is associated with the positive externality of creating financial funds. If these are placed in non-government debt instruments they contribute to financing private investment and – if they do not offset private saving – provide additional financing for economic growth. The major difference between NDC and FDC is, thus, that NDC does not create this opportunity. On the other hand, an FDC scheme that invests solely in government bonds can be viewed as a cost-inefficient NDC scheme, since the transaction costs of marketing the debt do not create offsetting revenues.

Proponents of NDC claim that it represents a paradigm shift in social security thinking (*e.g.*, Palmer 2000). By creating a direct link between contributions

---

[6] In principle, the Italian system should achieve a long-term equilibrium around the weighted contribution rates of the employed and self-employed if contributions and accrual factors are brought more in line. In practice, the absence of a mechanism to offset chronic divergence from the imputed return of 1.5 per cent may lead to financial difficulties. Palmer (1999) examines the stability conditions of the NDC PAYGO system, as does Valdés-Prieto (2001).

and the annuity and by basing the size of the annuity on life expectancy at retirement, NDC systems reduce the impact on system costs of individual behavioral choice and of unexpected changes in longevity. In comparison, in the DB framework the burden of the risk is unclear. It may fall on future generations of workers or on present workers before they retire.

NDC pension schemes are subject to the same "political risks" as DB pay-as-you-go – "political" management of public funds causing low rates of return, special interest lobbying, etc. For example, in Italy, the rate credited into the notional account is actually higher than the payroll tax earmarked for pensions, while in Poland it is lower, which is synonomous to taxing accounts. The NDC provides a framework for monitoring the costs of these interventions, as Fox and Palmer (1999) have argued in discussing the Latvian NDC scheme.

An NDC scheme with demographic reserves and indexation of notional capital and benefits that follows the growth of the contribution base, and with an annuity based on life expectancy projections that on average do not deviate from the outcome for birth cohorts, yields approximate long-run financial stability (Palmer 1999). If as in Sweden indexation follows the per capita wage rate and life expectancy is based on current outcomes – rather than a cohort projection – then there is a built in risk that the assets of the system will fall below the liabilities. Financial balance is secured in Sweden through a balance mechanism, based on current estimates of system assets and liabilities.

Briefly, the Swedish balance mechanism works as follows. If assets, measured as the estimated future stream of revenues from contributions and the current value of the system's buffer fund(s), are less than liabilities, *i.e.*, claims on future payments of pensions of pensioners and the notional account values of workers, the balance index falls below unity. In this case, both notional account values and benefits are adjusted to bring the system back into balance.

The Swedish NDC scheme was started with a large buffer fund inherited from the old system – these funds will help to cover the demographic pressure associated with the large birth cohorts of the 1940s. Various scenarios using relatively extreme demographic and economic assumptions show that it is likely that if the balance index has to be used in the future, the negative effect on future benefits during the whole retirement period of a pensioner is not likely to be more than 10 per cent in total, given some of the worse scenarios (Settergren et al. 2000).

Most importantly for the present topic, both FDC and NDC schemes work more efficiently with good expectancy projections. Poor projections give rise to a need for adjustment, and in general, do not provide the information needed by scheme participants to plan their "economic lives" given information about the development of their own cohort's life expectancy and the expected value of their own stream of pension benefits.

It's More Important than Ever to Project Life Expectancy Accurately

Public NDC and FDC schemes have been introduced in a number of countries since the mid-1990s.[7] In addition, also since 1995, public FDC schemes have been introduced in a large number of Latin American countries, and have become popular in especially the transition countries of Central and Eastern Europe.[8] These countries join, then, some of the forerunners of funded schemes, among them, Chile, Australia, Denmark, the Netherlands, Switzerland and the UK. The newer countries in the league differ from many of the forerunners, however, in their explicit DC construction. For example, the mandated employer schemes in Australia and the "opting out" schemes in the UK have been largely financial defined benefit (FDB) schemes, although there is a recent tendency for these also to convert to FDC. The difference between an FDB and an FDC scheme is small, however, since financial solidarity requires that life expectancy projections be on target in both.

In principle, there are two approaches that can be applied in estimating the life expectancy factor to be used in the calculation of annuities for NDC, and FDC, schemes. One is to base the projection on the current period tables – perhaps with some form of smoothed moving average. This is the approach applied by Sweden.[9] The major alternative is to produce a cohort projection.

---

[7] In addition to Sweden, Italy, Latvia and Poland, which have already been mentioned, versions of NDC schemes have been introduced in Kyrgyzstan, and in Russia.

[8] See Fox and Palmer 2001 for a discussion of the driving forces behind this movement.

[9] Although the life expectancy factor is continuously updated in Sweden, since people continue to live longer after receiving their pension, the procedure used to calculate it will underestimate the actual outcome. This is counterbalanced either through other, positive factors contributing to the financial balance, or through triggering the balance index (see above).

This is the approach applied by Latvia. The Swedish projection is revised yearly with new information on mortality, as is the Latvian projection.

The revision process differs in Latvia, however. Latvia bases changes in the projection of life expectancy on a professional judgment,[10] with a demographic analysis as the point of departure. In the revision process, it is asked whether new information provides sufficient evidence to revise the existing long-term projection.[11] One could view this approach as a sort of error-correction mechanism. In principle, the methods available for projecting life expectancy for FDC schemes are exactly those available for NDC. In the FDC context an effort is made to project a life expectancy factor that is expected to give long-run system solvency.[12]

In sum, as countries convert to DC schemes, life expectancy projections are used to *calculate individual pensions* in national systems. The question is whether the state of the art in projecting mortality can meet new demand created by large public schemes that are turning towards annuities based on life expectancy.

Final Comments

With conversion to public NDC and FDC schemes in Sweden,[13] as well as elsewhere, individuals have been given greater responsibility to plan their own working careers and saving with respect to a desired level of resources

---

[10] The judgment is the product of a white paper written by a leading demographer and a discussion in an official committee of demographers and actuaries set up for this purpose. This is discussed in Krumins, Palmer, Svensson and Vanovska (2001).

[11] The procedure was initiated in 1999, and in the first three years thereafter, no revision of the long-term projection had been made.

[12] In FDC schemes there is a trade off between the rate of return and life expectancy in the sense that inaccuracies in the projection of the life expectancy factor can be counterbalanced by a better rate of financial return on funded capital. In the Swedish NDC construction, since positive labor force gains are undistributed, i.e. indexation is with (1+g)(1+p), these together with good returns on the buffer fund(s) can counterbalance the clear inaccuracy in the projection of life expectancy at retirement resulting from not attempting to account for some additional increase in life expectancy of cohorts after retirement.

[13] Note that Sweden, as other countries that have introduced NDC and FDC schemes, has a minimum guarantee, which has not been discussed in the present context.

during retirement. In the DC framework resources during retirement are linked to contributions during the whole working career, and the level of a benefit is also determined by life expectancy at the age chosen for retirement. In this framework, knowledge about the development of cohort life expectancy becomes an important informational input into the economic plans of individuals.

One of the goals of policy makers is to loosen up the idea of "a" pension age at which everyone is expected to exit the labor force, that is the concept of the "statutory" or "mandatory" pension age that implanted itself so deeply in the minds of employees, unions and employers since the 1960s. The focus is to shift from the "right" to leave the labor force at age, for example 60, with a defined lifelong benefit, to the "right" to work as an older worker – but in a work environment that is friendly to older workers.

The transition from national DB to DC schemes presupposes a future where people can freely choose between work and retirement – combining a partial or full benefit with partial or full retirement. It also presupposes that individuals invest enough in their human capital (personal health, education and training) to be able to remain in the labor force longer. Today the *de facto* age of exit from the labor force for men and women together is below 60 in the OECD countries. There is evidence that this has been influenced by national benefit schemes (Gruber and Wise 1999). Countries are now aiming to raise this age. National DC (NDC or FDC) schemes are a tool that can help promote this goal, and this is an important reason why countries are introducing them.[14]

To conclude, in order to plan for retirement in a DC environment, individuals need good information about their cohort's life expectancy. Work career and saving plans will then be formulated in accordance with this information, thereby determining lifetime resources and their distribution over the life cycle.

References

Feldstein M. and A. Samwick (1997). "The Economics of Prefunding Social Security and Medical Care Benefits". NBER Working Paper, no. 6055. Cambridge, Mass: NBER.

[14] Another is that the value of pension rights equals the account value at any given time. This makes it easy to move between jobs, occupations and countries without losing rights, eliminates the potential locking in effect of some formulations of DB schemes, for example, DB final-salary schemes.

Feldstein M. and A. Samwick (1998). "Two Percent Personal Retirement Accounts: Their Potential Effects on Social Security Taxes and National Saving". NBER Working Paper no. 6540. Cambridge, Mass: NBER.

Fox L. and E. Palmer (1999). "Latvian Pension reform." Social Protection Discussion Paper No. 9922.Washington D.C.: The World Bank.

Fox L. and E. Palmer (2001). "New Approaches to Multi-pillar Pension Systems: What in the World Is Going On?" in J. Stiglitz et al. (eds.) *New Ideas about Old Age Security.* World Bank. Chicago: University of Chicago Press.

Frennberg P. and B. Hansson (1992). "Swedish Stocks, Bonds, Bills and Inflation", *Applied Financial Economics*, 2.

Góra M. and E. Palmer (2002). "Shifting Perspectives in Pensions," Working Paper. Warsaw: Center for Economic and Social Research (CASE).

Gruber J. and D. A. Wise (1999). *Social security programs and retirement around the world*. Chicago: University of Chicago Press.

Krumins J., E. Palmer, I. Svenssson and I. Vanovska (2001). "Life expectancy trends and calculations of projected life expectancy at retirement – from transition to post-transition – the case of Latvia." Paper presented at the European Population Conference, Helsinki Finland. June 7-9.

Palmer E. (1999). "Individual Decisions and Aggregate Stability in the NDC System". Mimeograph.

Palmer E. (2000). "The Swedish Pension Reform: Framework and Issue". World Bank Pension Primer. Washington DC: The World Bank.

Palmer E. (2002). "Swedish Pension Reform – How Did It Evolve and What Does It Mean for the Future?" in Feldstein, M and H. Siebert (eds.) *Coping with the Pension Crisis: Where Does Europe Stand?* Chicago: University of Chicago Press.

Settergren O., N. Holmgren, J. Leander and B. Mikula (2000). *The Automatic Balance Mechanism in the Swedish Pension System*. RFV Analyserar 2000:1. Stockholm: The National Social Insurance Board.

Settergren O. (2001). "The Automatic Balance Mechanism of the Swedish Pension System – A Non-Technical Introduction", Wirtschaftspolitische Blätter 4.

Valdés-Prieto S. (2001). "The Financial Stability of Notional Account Pensions", *Scandinavian Journal of Economics*, vol. 102, no. 3, pp. 395-417.

Vaupel J. (2002). Paper in this publication.

World Bank (1994). *Averting the Old Age Crisis: Policies to Protect the Old and Promote Growth*. Washington, D.C.

Types of Models for Projecting Mortality

Nico Keilman

Professor of Demography at University of Oslo, Norway

The purpose of this short paper is to present a systematic overview of various types of models for projecting mortality. The framework is that of population forecasts of the cohort-component type for the elderly at the national level. This means that regional aspects are disregarded. However, since the elderly migrate relatively little (except when necessary to obtain care), any national mortality model for the elderly can equally well be applied at the regional level, provided the data exist.

I decided not to present any new synthesis; there are already many good reviews, for instance the excellent recent book edited by Tabeau, van den Berg Jets and Heathcote (2001), and furthermore papers by Gomez de Leon and Texmon (1992), Murphy (1990), Willekens (1990), Olshansky (1988), and Pollard (1987).

As a demographer, I first thought of distinguishing between demographic and non-demographic models (Sections 1 and 2). Demographic models are rather mechanistic, although they involve varying degrees of judgment. In this category, I distinguish between extrapolation models and interpolation models (Sections 1.1 and 1.2). Non-demographic models are much more causal in nature than demographic ones. I differentiate between macro models (Section 2.1) and micro models (Section 2.2). Finer distinctions and further qualifications will be provided below.

1 Demographic/actuarial Models

Models for mortality projection used by actuaries are included in this first group of demographic models. They are typically *macro* (aggregate) models, describing the evolution over time of the mortality risks of *groups* of individuals. A second characteristic is that the mortality risks are broken down by *demographic* variables only: usually sex and age, often cause of death (COD), and sometimes other characteristics (for example marital status) as well. Since time is a critical factor in any projection model, the combination of time with age raises the question whether one should base the mortality

model on synthetic cohorts (period approach) or real cohorts (cohort approach). I will come back to that issue below.

1.1 Extrapolation Models

Extrapolation models are based on time series for one or more observed indicators. The type of time-series model employed may be extremely simple, for instance constant future levels of the indicators (as observed for a certain base period), or constant slopes for those indicators. At the other extreme, we find sophisticated models of the ARIMA-type, for example a random walk with drift for life expectancy at birth. Another issue is the choice of indicators. It may be death rates or death probabilities by age and sex (possibly by COD), or a summary indicator such as the standardized mortality ratio (SMR), life expectancy at birth, or the parameters of an age schedule (Gompertz, Heligman-Pollard, Lee-Carter, etc.). Tabeau et al. (2001) offer an example of a (deterministic) time-series model for the extrapolation of mortality by age, sex, and COD. The authors analysed and extrapolated mortality in France, Italy, the Netherlands, and Norway. For Norway, they performed three types of analysis on the historical data. First, they used a period approach and fitted Heligman-Pollard (H-P) curves to death rates by age and sex. Second, in a cohort approach, they used death rates by age and sex for the population aged 40 and over to fit a modified Gompertz curve (as applied in the H-P curve). Third, they fitted various exponential and double exponential models to the age patterns of period death rates by age, sex, and COD (ten groups). In all three cases, the parameters were extrapolated by means of deterministic trend models (linear decline or increase, quadratic decline, hyperbolic decline or increase, log-normal decline or increase, constant).

1.2 Interpolation Models

Extrapolative models described above take future mortality levels, as predicted by the method, as given. In practice, one inspects the predictions and assesses their plausibility. If the future levels are unrealistic, a different extrapolation technique may be used, or the extrapolation period may be reduced. However, extrapolative models are of limited value for long-term forecasts, such as 50 years ahead. It is highly probable that the future, in whatever sense it is regarded, will not look like the recent or remote past. Therefore, subjective and qualitative statements must be made about mortality levels for the distant future. Long-term forecasts call for *interpolation* models, in which one first specifies the value of one or more mortality indicators in a future year, the so-called target year. Next, one interpolates between recently observed values and target values. The target values are usu-

ally determined on the basis of international comparisons, by referring to "more advanced" (*i.e.* lower-mortality) populations, or to an "optimal" life table under ideal conditions.[1] Helge Brunborg presents in his paper an example for Norway: Statistics Norway has specified the life-expectancy values for men and women in 2050 as 80 and 84.5 years, respectively. Next, life expectancy values for shorter forecast periods were obtained. In the forecasts of 1993 and 1996, this was done by deterministic trend interpolation; see Statistics Norway (1994, 1997). By contrast, the forecast of 1999 employed age-specific rates, which were interpolated from age-specific deterministic trends; see Statistics Norway (1999, 2001).

1.3 Comments

- In all projection models discussed above, the mortality indicators in the future are interpreted as *deterministic* variables. In other words, one is only interested in the *expected* level of mortality, not in its *prediction* interval. In recent years, research in the field of *stochastic* cohort-component forecasting has resulted in various models for stochastic mortality forecasts (*e.g.*, Lee and Tuljapurkar 1994; Alho 1998).
- One important issue is the level of aggregation for the projection models. Is a breakdown by age and sex sufficient, or should one also include cause of death? There is some literature on this subject (Murphy 1990; McNown and Rogers 1992; Alho 1991; Wilmoth 1995; Caselli 1996; Tabeau *et al*. 2001). From a theoretical perspective, it is important to note that under certain conditions, cause-specific mortality projections tend to result in lower future values for life expectancy than do all-cause projections. This is the case when a linear trend model is used. Second, existing COD-models assume that the causes are statistically independent, which is obviously not realistic. From a practical point of view, one should note the danger of misclassification of deaths by cause (*e.g.*, primary or secondary cause), particularly for the very oldest individuals. Furthermore, rapidly changing CODs, when used in non-linear models, may produce implausible results. This does not mean, however, that COD projections are of little value. When a non-linear COD model is used, one may obtain both higher and lower forecasts of life expectancy, compared to an all-cause

[1] Interestingly, Alho (1992) views this type of models as extrapolation models, too. He proposes that the target value be interpreted as data (with measurement error, as expressed by the expected variance of the target value), and that a time-series model be fitted on the basis of all "data", incorporating relevant variances. Next, the time-series model may be used for (possibly out-of-sample) predictions.

model. Second, when the weight of certain causes in overall mortality has changed over time, it may be easier to interpret historical trends for cause-specific mortality than for all-cause mortality. Needless to say, short-term forecasts for COD, and alternatively with the causes combined, will result in similar mortality levels.

- A separate issue is whether one should project cohort mortality or period mortality. In other words, are mortality trends, adjusted for age structure, best reflected in cohort effects, or in period effects? The answer is not straightforward. First, it is logically impossible to identify uniquely the three types of effects (age, period, and cohort effects). Various solutions have been proposed, but there is no agreement in the literature as to the best choice. At the same time, choosing one solution or the other may have a strong impact on the results. The second problem is the empirical fact that the interaction between age, period, and cohort should be included in order to obtain a satisfactory fit, in addition to the three main effects. Finally, estimates of cohort effects for recent cohorts are uncertain because most of their mortality has yet to come. Yet, the cohort perspective acknowledges life-long effects of health-related behaviour and life styles, which the period perspective cannot do. In practice, however, very little attention is given to cohort effects in projecting age-specific mortality, and the period perspective predominates. A notable exception is the work of Tabeau and colleagues referred to above.
- I have not tried to assess the usefulness of different "laws of mortality", *i.e.* mathematical representations of age-specific mortality – most often (but not exclusively) in terms of the death rates. A large number of such laws have been proposed in the literature, a process that started with De Moivre in 1725 and has continued to the present day (Hannerz 2001). Some of those laws are applicable only to adult or old-age mortality (Coale-Kisker, Himes-Preston-Condran, Gompertz, Perks, Weibull). Tabeau (2001) and Boleslawski and Tabeau (2001) compare some 27 of such laws. Relational models, such as Brass' logit model, and the Lee-Carter model, should also be considered.

2 Non-demographic/Causal Models

Age is not an explanatory factor in the causal sense, and sex very seldom is, either. Most often, the causal mechanism works through underlying factors. This explains why non-demographic models are often of the causal type. Insights from epidemiology and biomedical research are used. Covariates employed here (in addition to age, sex, and COD) include various indicators that reflect health, the environment, life style, access to health care, social

support, or socio-economic status. A further distinction is that between macro and micro models.

2.1 Non-demographic Macro Models

Epidemiological models use disease processes and related risk factors as the basis for modelling mortality and morbidity for groups of individuals. They often take the form of statistical regression models. In a time-series or cross-sectional approach, mortality or morbidity indicators are selected as the dependent variable and linked to such covariates as smoking behaviour, education, or income. Prominent examples of this approach include the Global Burden of Disease Study (GBD), which explains Disability Adjusted Life Expectancy worldwide based on socio-economic covariates, and smoking behaviour (Murray and Lopez 1997), and the lung cancer/smoking model of Alderson and Ashwood (1985) for England and Wales. An obvious advantage of the latter type of models, compared to the extrapolation models discussed above, is that the latency period of several decades between the start of smoking and the incidence of lung cancer is explicitly modelled. Mechanical extrapolations based on the conspicuous trends in the 1960s and 1970s, but overlooking the trends in smoking during the last 20 to 25 years (decrease for men, increase for women), will yield projected lung cancer levels that are too high for men and too low for women.

2.2 Micro Models: Ageing and Disease Processes

At the micro level, stochastic models have been developed for ageing and disease processes for individuals.

2.2.1 Models for Ageing Processes in Individuals

Yashin (2001) has recently provided a useful overview of models for individual ageing and mortality, including frailty models, physiological ageing models, and models for DNA repair. He states that models incorporating age-dependent frailty have considerable potential for mortality forecasting because they enable researchers to model the impact of unobserved and observed stochastically changing covariates on individual chances of survival. To the best of my knowledge, however, models of this kind have not been used so far in actual mortality forecasting.

2.2.2 Micro Simulation Models

A number of micro simulation models that describe the morbidity and mortality history of individuals have been developed. These include the Canadian POHEM (Population Health Model), models for the outbreak and spread of infectious diseases (HIV/AIDS, STDs), and Manton's stochastic risk-factor-change models. The common feature of these models is that they are based on the concept of a state space. Examples of states in addition to "alive" and "dead" might include the following: "susceptible" and "infected", or belonging to pre-specified classes of risk factors (such as blood pressure, cholesterol, smoking, body-mass index). Individuals move between states according to a set of transition rates, which could be exogenous or linked to covariates. Simulation is necessary to avoid the Markov assumption often used in analytical state-space models.

2.3 Comment

An important problem in epidemiological research is the dependency between risk factors. Factors such as smoking behaviour, blood pressure, body-mass index, physical activity and dietary habits are clearly interrelated. One consequence is that the benefits of risk-factor interventions may be substantially overestimated when the health gain is calculated for each factor separately.

3 Instead of a Conclusion

I shall not attempt to indicate whether models belonging to one type are more useful for mortality forecasting than models of a different type. The reason is that the notion "useful for mortality forecasting" has many dimensions. Does the model result in accurate predictions? Does it contain the type of information needed for policy making? Does it suggest unwanted future developments that require policy action? Does it offer insight into underlying processes? Can it easily be transferred from one data situation to another? The conclusion reached by the reviews of the literature referred to above is that no single type of model scores best on all or most of these criteria.

Yet, one cannot deny that the extrapolative and interpolative types of models discussed in Section 1 are used most frequently, possibly because many demographers simply lack the experience of working with causal mortality models.

References

Alderson M. and F. Ahwood (1985). "Projection of Mortality Rates for the Elderly", *Population Trends* 42, 22-29.

Alho J.M. (1991). "Effect of Aggregation on the Estimation of Trends in Mortality", *Mathematical Population Studies* 3(1), 53-67.

Alho J.M. (1992). "Estimating the Strength of Expert Judgement; the Case of US Mortality Forecasts", *Journal of Forecasting* 11, 157-167.

Alho J. M. (1998). A Stochastic Forecast of the Population of Finland. Reviews no.4. Helsinki: Statistics Finland.

Boleslawski L. and E. Tabeau (2001). "Comparing Theoretical Age Patterns of Mortality Beyond the Age of 80", in Tabeau, E. A. van den Berg Jets, and C. Heathcote (eds.) (2001) *Forecasting Mortality in Developed Countries: Insights from a Statistical, Demographic and Epidemiological Perspective.* Dordrecht: Kluwer Academic Publishers.

Caselli G. (1996). "Future Longevity Among the Elderly", 235-265 in G. Caselli and A. Lopez (eds.) *Health and Mortality Among Elderly Populations.* Oxford: Clarendon Press.

Goméz de Leon J. and I. Texmon (1992). "Methods of Mortality Projections and Forecasts", in Keilman N. and H. Cruijsen (eds.) *National population forecasting in industrialized countries.* Amsterdam: Swets and Zeitlinger.

Hannerz H. (2001). Manhood trials and the law of mortality. *Demographic Research* 4, article 7.

Lee R. and S. Tuljapurkar (1994). "Stochastic Population Forecasts for the United States: Beyond High, Medium, and Low", *Journal of the American Statistical Association* 89(428), 1175-1189.

McNown R. and A. Rogers (1992). "Forecasting Cause-Specific Mortality Using Time Series Models", *International Journal of Forecasting* 8, 413-432.

Murphy M. (1990). "Methods for Forecasting Mortality for Population Projections", in *Population Projections: Trends, Methods and Uses*. Occupational Paper 38. OPCS, London.

Murray C. and A. Lopez (1997). "Alternative Projections of Mortality and Disability by Cause 1990-2020: Global Burden Disease Study", *Lancet* 349, 1498-1504.

Olshansky S. J. (1988). "On Forecasting Mortality", *The Milbank Quarterly* 66(3), 482-530.

Pollard J. (1987). "Projection of Age-Specific Mortality Rates", *Population Bulletin of the United Nations* 21/22, 55-69.

Statistics Norway (1994). *Population Projections 1993-2050. National and Regional Figures*, Official Statistics of Norway, NOS C176, Oslo: Statistics Norway.

Statistics Norway (1997). *Population Projections 1996-2050. National and Regional Figures*, Official Statistics of Norway, NOS C414, Oslo: Statistics Norway.

Statistics Norway (1999). "Continued population growth. Population Projections. National and Regional Figures, 1999-2050".[Internet www.ssb.no/english/subjects/02/03/folkfram_en].

Statistics Norway (2001). *Population Projections 1999-2050. National and Regional Figures*, Official Statistics of Norway, NOS C693. Oslo: Statistics Norway. [Internet www.ssb.no/emner/02/03/ nos_c693/nos_c693.pdf].

Tabeau E. (2001). "A Review of Demographic Forecasting Models for Mortality", in Tabeau E., A. van den Berg Jets and C. Heathcote (eds.) *Forecasting Mortality in Developed Countries: Insights from a Statistical, Demographic and Epidemiological Perspective*. Dordrecht: Kluwer Academic Publishers.

Tabeau E., P. Ekamper, C. Huisman and A. Bosch (2001). "Predicting Mortality from Period, Cohort, or Cause-Specific Trends: A Study of Four European Countries", in Tabeau E., A. van den Berg Jets and C. Heathcote (eds.) *Forecasting Mortality in Developed Countries: Insights from a Statistical, Demographic and Epidemiological Perspective*. Dordrecht: Kluwer Academic Publishers.

Willekens F. (1990). "Demographic Forecasting: State-of-the-Art and Research Needs", in Hazeu C. and G. Frinking (eds.) *Emerging Issues in Demographic Research.* Amsterdam: Elsevier.

Wilmoth J. (1995). "Are Mortality Projections Always More Pessimistic When Disaggregated by Cause of Death?" *Mathematical Population Studies* 5(3), 293-319.

Yashin A. (2001). "Mortality Models Incorporating Theoretical Concepts of Ageing", in Tabeau E., A. van den Berg Jets and C. Heathcote (eds.) *Forecasting Mortality in Developed Countries: Insights from a Statistical, Demographic and Epidemiological Perspective*. Dordrecht: Kluwer Academic Publishers.

Experiences from Forecasting Mortality in Finland

Juha M. Alho

Professor of Statistics at the University of Joensuu, Finland

1 Modeen and Törnqvist

The first official cohort-component projection of the population of Finland was prepared by Gunnar Modeen (1934a), an actuary with the Central Statistical Office of Finland at the time. Modeen's work had elements of genuine forecasting in that he commented on past trends in fertility, mortality, and migration, and discussed their possible long-term implications (Modeen 1934b). On the other hand, the work was rather schematic in nature. In particular, age-specific mortality was assumed not to change during the projection period, although Modeen was aware of its declining trend since the late 19th century (Modeen 1934a, 38). Unable to pinpoint the future rate of decline exactly, Modeen rejected any alternative assumption as speculative (cf., Modeen and Fougstedt 1938).

Analyses of mortality trends in presumably more advanced countries were used as *leading indicators* in the United States by Whelpton *et al.* (1947), for example. In Finland, Leo Törnqvist (1949) proposed similar methods. In particular, he used Swedish mortality as a target towards which he assumed Finnish mortality to converge. Both series were first transformed via a logistic type transformation. Then, the curves were aligned, and the Finnish curve was prolonged in accordance with the Swedish development.

A problem with Modeen's projection was that it soon became outdated. Fertility started to increase at the time the projection was published, and mortality continued to decline. Modeen's calculations suggested that the Finnish population would never exceed four million, but this mark had already been crossed by 1950. Törnqvist's collected works (Viren *et al*. 1981) do not mention the error of past forecasts as a motivation for his own early work. Nevertheless, the future statistics professor, a specialist in time-series analysis (among other fields, cf. Nordberg 1999), was well aware that forecasts cannot be made without error. He appears to have been the first to formulate the problem of uncertainty in population forecasting in probabilistic terms in

Törnqvist (1949). Later, Törnqvist also conducted what must be one of the earliest assessments of the empirical accuracy of Finnish forecasts (including his own!) in Törnqvist (1967).

In this note, we will outline current developments in Finnish mortality forecasting. In Section 2, we describe the methods used by official forecasters. These derive mainly from the tradition of early cohort-component forecasters (cf.. DeGans 1999). In Section 3, we discuss how uncertainty can be taken into account using probabilistic models and present-day computing facilities. We conclude in Section 4 by commenting on some applications for which mortality forecasts are particularly relevant.

2 Official Forecasts [1]

The arithmetic underlying cohort-component forecasts was understood a hundred years ago (DeGans 1999). Since the method relies on detailed assumptions concerning future age-specific rates, the real key to forecast accuracy lies with those assumptions. One would think that major improvements would have occurred during the past century, judging from the way the assumptions are formulated. Yet, the methods were essentially perfected by Whelpton back in the 1940s.

The two producers of official population forecasts in Finland are Statistics Finland and the Social Insurance Institution of Finland (or KELA, an abbreviation of the Finnish name). Since the forecasters of the two institutions cooperate on an informal basis, the forecasts have many similarities.

Both institutions produce forecasts approximately every three years. More frequent updates are made if unexpected developments occur. Both disaggregate the population by sex and single years of age (0, 1, 2, ..., 99, 100+). Currently both organizations forecast until 2050.

KELA produces a national forecast only, whereas Statistics Finland forecasts the population of every one of the 448 municipalities of Finland. In the case of mortality, the country is divided into three relatively homogeneous areas: Northern and Eastern Finland, which have a high level of mortality (due in particular to cardio-vascular diseases among males); the Swedish-speaking

[1] The author would like to thank Mr. Matti Saari, Statistics Finland, and Mr. Markku Ryynänen, KELA, for information on the practice of forecasting. Any misunderstandings are the sole responsibility of the author.

coastal areas, with low mortality; the rest of the country, with intermediate mortality. The reason for the low mortality among the Swedish speakers has not been established, but both socio-economic and lifestyle factors apparently play a part (Koskinen and Martelin 1995).

Neither organization uses cause-specific mortality data in the preparation of their assumptions. This is contrast with the U.S. Office of the Actuary, for example (*e.g.*, Wade 1987). However, we have argued elsewhere that cause-specific information cannot be expected to increase forecast accuracy unless one of two conditions are met: either leading indicators can be identified in the preparation of forecasts, or structural changes can be anticipated based on other available information (as in the case of AIDS, for example) (Alho 1991).

Both organizations use trend extrapolation as a basis for their mortality forecast. Starting from a target value for life expectancy at birth, e_0, Statistics Finland adjusts future age-specific mortality rates so that the implied increase in life expectancy gradually slows down until the target of e_0 is reached. Age-dependent proportional adjustment is used to modify the jump-off rates. In KELA the starting point is a classification of individual ages into aggregates with similar mortality levels. Regression analysis is used in the log-scale to estimate rates of decline that gradually decelerate. The assumption, made by both organizations, that the rate of decline eventually falls off, is far from self-evident. In fact, we have used U.S. data to show that such an assumption has historically made the U.S. mortality forecasts *worse* than simpler trend extrapolations (Alho 1990).

Neither organization formulates their targets on a cohort basis although both occasionally examine cohort trends to see whether there are any irregularities. A current example of such an irregularity was reported by KELA: the female cohorts born in the 1950s appear to have higher mortality than cohorts born earlier, during WWII.

The methods of trend extrapolation used by the organizations blend judgment and empirical analysis. Neither organization has experimented with the method proposed by Lee and Carter (1992). Its performance in regard to ages 65+ in Finland, was investigated in a University of Joensuu *pro gradu* thesis by Eklund (1995), who found that a one-dimensional singular-value decomposition produced a good fit to the data. Because of random variation, however, the resulting forecast was not always an increasing function of age.

In addition to the trend forecast, KELA produces another mortality variant in which it is assumed (as Modeen did) that mortality will remain at the jump-

off level. Statistics Finland limits itself to a single variant even though high and low variants have previously been used in national forecasts.

3 Predictive Distribution of Mortality

A major contribution by Törnqvist (1949) was that he was apparently the first to maintain that since the future values of a vital rate cannot be totally known, they must be treated as random variables. The actual future values are then "samples" from their distributions. In modern terminology, the uncertainty of the future value is expressed in terms of a predictive distribution that represents both our best guess and its uncertainty. The distribution is conditioned on all information available at the jump-off time of the forecast (*e.g.*, Gelman *et al*. 1995, 9).

Törnqvist's contribution may have been ahead of its time. In particular, correct formal treatment of the predictive distribution would have been difficult before the availability of high-speed computing. In recent years, the potential usefulness of a probabilistic approach to uncertainty has been noted on several occasions.[2] At the University of Joensuu, we have written a computer program, PEP (Program for Error Propagation), which is capable of simulation samples from a wide range of predictive distributions.

The main concept of PEP is that it allows us to describe the uncertainty connected with a forecast at the time it is being made. All sources of uncertainty – age-specific fertility and age and sex-specific mortality and migration – are taken into account and propagated throughout to derive the predictive distribution of the population. In this sense, PEP is merely a stochastic version of the cohort-component bookkeeping system. The usefulness of the results depends on the assumptions underlying the calculations. The user of PEP must specify a point forecast for each of the vital rates for all future years, just as in ordinary cohort-component forecasting. An additional step is required in the form of specifying the uncertainty surrounding the forecast.

Suppose R(j,t) is the mortality rate for age j = 0, 1, ..., ω in a future year t = 1, 2, ..., T. PEP assumes that

---

[2] Review of Land (1986); "Special Section on Statistical Analysis of Errors in Population Forecasting and Its Implications on Policy," *Journal of Official Statistics*, September 1997; "Frontiers of Population Forecasting," *Population and Development Review*, 1998 Supplement; review of U.N. forecasts by the National Research Council (2000).

$$R(j, t) = \exp(\hat{r}(j, t) + X(j, t)),$$

where $\hat{r}(j, t)$ is the point forecast of the log-rate, and X(j,t) is a random error with a mean of E[X(j,t)] = 0. The random error can always be written in the form

$$X(j, t) = \varepsilon(1, t) + ... + \varepsilon(j, t).$$

In PEP, the *error increments* ε(j,t) are *assumed* to be of the form

$$\varepsilon(j, t) = S(j, t)\,(\eta_j + \delta(j, t)),$$

where the S(j,t)'s are known scale factors that can be chosen to match any sequence of error variances Var(X(j,t)) that increases with t. Fixing j, we can think of the terms η_j as representing errors in forecasted trends. In the case of mortality, the trend corresponds to the rate of decline, for example. Since the terms δ(j,t) are independent for any fixed j, they represent unpredictable random variation. The relative roles of the two types of uncertainties derive from the assumption $\eta_j \sim N(0, \kappa_j)$, and $\delta(j,t) \sim N(0, 1 - \kappa_j)$, where $0 \le \kappa_j \le 1$. The terms η_j are assumed to be independent of the terms δ(j,t). Finally, the terms η_j can either have a constant correlation across j, or an AR(1) type correlation. The same is true for the δ(j,t)'s, when t is fixed. This *scaled model* for error was introduced in Alho and Spencer (1997).

In Alho, (1998) we provide details of the application of PEP to the population of Finland for 1999–2050. The point forecasts for each vital rate were as specified by Statistics Finland. We now present some details on the treatment of uncertainty in the mortality forecast.

Age-specific mortality data in 5-year age-groups 0–4, 5–9, ..., 75–79, and 80+ were available for the years 1900–1994. After a preliminary analysis, the data were aggregated into the broader age groups 0–4, 5–34, 35–59, 60–79, and 80+ by adding the age-specific rates together. This increased the stability of the trends. The analysis was carried out in terms of the logarithm of the sum (cf. Alho 1998, Figures 5a–e, pp. 19–21). The unusual values produced by the civil war in 1918 and WWII in 1939–1944 were smoothed using values from the previous year. For each of the five broad age groups, we produced baseline forecasts as follows:

- Starting from year y = 1915, we used the data for the previous 15 years (y, y - 1, ..., y - 15) to calculate a trend forecast for all future years until 1994.
- A linear trend was estimated from the first and the last observation of the 15-year data period.

- In case the linear trend was positive, it was replaced by a constant value *(i.e.*, slope = 0).

For each y = 1915, 1916, we calculated the empirical forecast error for lead times t = 1, 2, ..., 50. For each lead time t, we could then estimate the standard deviation of the error around zero (*i.e.*, assuming that the forecasts are unbiased). This would give us estimates of Var(X(j,t)) directly, from which the scales S(j,t) could be deduced. However, it turned out that especially for younger ages the estimates were somewhat erratic because of the large random (Poisson-type) variation in the counts. Therefore, final estimates were produced by averaging the estimates from the six time series corresponding to the three broad age groups of 35–59, 60–79, and 80+ for males and females. The resulting estimate of the standard deviation of the relative error starts from approximately 0.06 at t = 1 and increases in a linear fashion to about 0.6 at t = 50. Otherwise expressed, the relative error one might expect for a single age group increases from 6 per cent to roughly 60 per cent in 50 years (cf., Alho 1998, Figure 6, p. 22). These estimates were used for all ages.

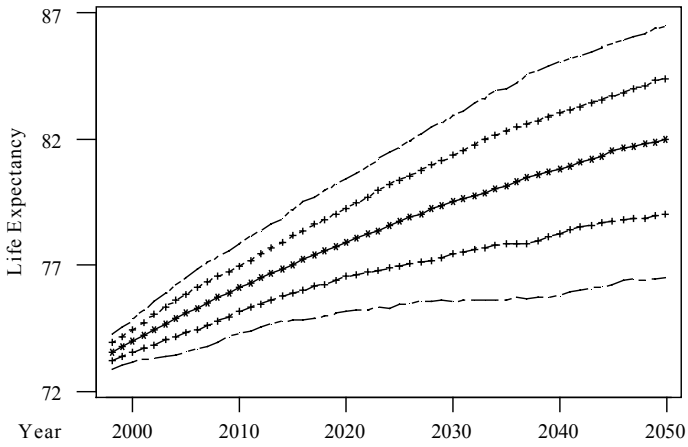
The results were checked by fitting an ARIMA(1,1,0) model to the data series, and similar results were obtained (Alho 1998, Figure 6, p. 22).

The parameter κ was estimated by the least-squares method. The single value $\kappa = 0.149$ was applied for all ages.

An AR(1) process was used to model the autocorrelation of the error terms η_j and $\delta(j,t)$ across age j. Otherwise expressed, the correlation was assumed to be $\rho^{|i-j|}$ for any two single years of age i and j, where the empirical estimate $\rho = .945$ was used for η_j's and $\rho = .977$ was used for $\delta(j,t)$'s. Finally, a parameter for contemporaneous crosscorrelation between the error of male mortality and the female mortality was estimated as .795.

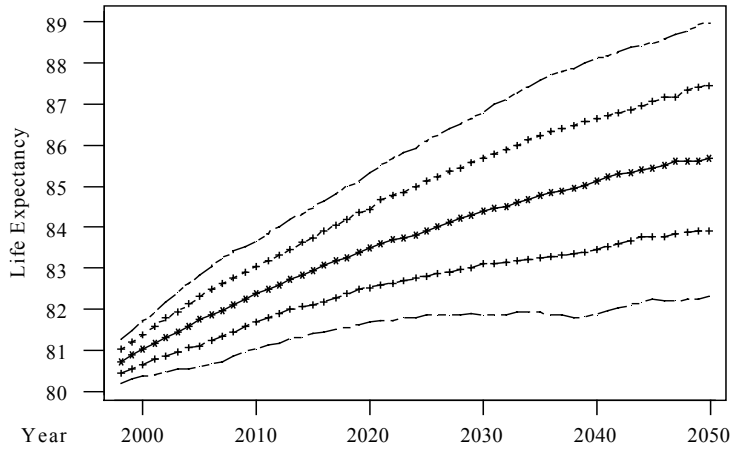
The details are fairly complex. One way to assess the reasonableness of the procedures is to consider their implications for life expectancy. Figure 1 has a predictive distribution for male life expectancy at birth, and Figure 2 has a plot for female life expectancy. The median of the predictive distribution is 82.0 for males and 85.6 for females in 2050. A 50 per cent prediction interval (or interval between the first and third quartile) is [79.0, 84.4] for males and [83.9, 87.5] for females. An 80 per cent prediction interval for males is [76.5, 86.5] and [82.3, 89.0] for females. The narrower spread for females is probably due to their lower level of mortality.

Figure 1 Predictive Distribution of Male Life Expectancy in Finland in 1998–2050



Two concerns can be raised concerning the intervals. First, the long-term point forecasts are based on an eventual slowdown of the decline in mortality; this may make the Finnish forecast too conservative, as it did in the U.S. earlier. However, we may note that the life expectancy implied by the current Swedish forecasts for males is 82.6 and for females 86.5 years in 2050. In the intermediate variant of the Norwegian forecast, the corresponding ages assumed are 80.0 and 84.5 years. We see that despite the assumption of a slowdown in the mortality decline in Finland, the Finnish forecast is the most optimistic of the three in terms of improvement, since the current life expectancy in Finland is the lowest. Even though the Finnish point forecast may be too low at the end of the forecast period, from this perspective the Finnish forecast appears less conservative.

Figure 2 Predictive Distribution of Female Life Expectancy in Finland in 1998–2050



Second, in view of the vast potential for new medical advances, one could argue that the range of uncertainty expressed by the widths of the intervals might be overly narrow. Two arguments seem relevant here. For the U.S. (both sexes combined), Lee and Carter (1992, p. 660, Figure 1.) calculated model-based 95 per cent intervals for life expectancy 50 years ahead. The width of these intervals was approximately 8.4 years. In a normal model, the corresponding width for an 80 per cent interval would be approximately 5.5 years. Thus, our intervals are clearly wider. In a discussion of the paper by Lee and Carter, we noted that by including all sources of variation, the Lee and Carter intervals would have been approximately one half wider (Alho 1992). This would have resulted in estimates close to ours. (One could also argue that in a large country with heterogeneous sub-populations there might be some offsetting variation, resulting in a national average more stable than in a small homogeneous country. While conceivable, this possibility does not seem to be an adequate reason for inflating the Finnish intervals, since it does not show up in the Finnish time series.)

A related criticism suggests that future advances in medical knowledge may be so unprecedented that intervals based on past outcomes are too narrow. However, in 1915, at the start of our observation period, both life expectancy for both males and females was substantially lower – 43.2 and 49.2 years (Kannisto and Nieminen 1996); the improvements by the end of the 20th century were 27.5 and 29.5 years, respectively. Our estimates reflect the variation in this turbulent period of major improvement. The future can be

even more volatile, but the advantage of our intervals is that they correspond to actual past variation, rather than to a subjective assessment. Of course, a subjective assessment may be used as a basis for other calculations that would complement our own.

4 Applications

Now that we have the analytical capability to produce predictive distributions for future vital rates and future population, it is of some interest to consider how they might be applied. There are two aspects to this.

First, it is critical that we understand how the predictive distribution can be understood. As noted, *e.g*., in Alho (1998), predictive distributions can be based on (1) formal statistical models, (2) errors of past forecasts can be used to estimate the error of future forecasts, (3) errors of baseline forecasts can be used to estimate future error, and (4) error specification can be purely subjective. Of course, any mixture of the four is also a possibility. The results we have shown rely primarily on (3), although they have elements of (1) and (4), as well. The aim was to provide an empirical assessment of the difficulty of forecasting (or ”forecastability”) of the vital processes for different times. As such, the *results for mortality correspond to the uncertainty in mortality forecasting during the 20th century*. One can reasonably question whether forecasting is now easier, or more difficult, than in the past, but at least we now have a quantitative empirical assessment of how things were before.

Second, the predictive distribution can be used to address numerous social-policy issues that depend on future population and its age structure. For example, in Alho *et al*. (2001) we review an example in which output from PEP was used in combination with the Finnish overlapping-generations model to devise alternative pension-funding rules, and another example in which output from PEP was used to assess the stability of the current rules for state aid to municipalities. In a University of Joensuu *pro gradu* thesis, Polvinen (2001) used PEP to form a predictive distribution of the so-called generational accounts. All these questions are of fundamental concern for the long-term planning of the social-support systems in Finland. In no case has the effect of uncertain population age-distribution previously been recognized. Other applications have been presented by Lee and Tuljapurkar (1998) for the Social Security system of the United States, for example. Further research opportunities are discussed in Auerbach and Lee (2001).

References

Alho J. (1990). "Stochastic Methods in Population Forecasting". *International Journal of Forecasting*, 6, 521–530.

Alho J. (1991). "Effect of Aggregation on the Estimation of Trend in Mortality". *Mathematical Population Studies*, 3, 53–67.

Alho J. (1992). "Comment". *Journal of the American Statistical Association* 87, 673–675.

Alho J. (1998). "A Stochastic Forecast of the Population of Finland". *Reviews* 1998/4. Helsinki: Statistics Finland.

Alho J. and B.D. Spencer (1997). "The Practical Specification of Expected Error of Population Forecasts". *Journal of Official Statistics*, 13, 203–225.

Alho J., J. Lassila, M. Salo, B.D. Spencer and T. Valkonen (2001). "Predictive Distributions for Future Population with Applications to Aging". Manuscript.

Auerbach A.J. and R.D. Lee (Eds.) (2001). *Demographic Change and Fiscal Policy*. Cambridge University Press.

Collected Scientific Papers of Leo Törnqvist. Series A7. Helsinki: The Research Institute of the Finnish Economy.

DeGans H.A. (1999). *Population Forecasting 1895–1945*. Dordrecht: Kluwer.

Eklund K. (1995). *Kuolevuuden mallittaminen ja ennustaminen Suomessa vanhusväestön keskuudessa*. Pro Gradu Thesis. University of Joensuu.

Gelman A., J.B. Carlin, D.B. Rubin and H.S. Stern (1995). *Bayesian Data Analysis*. London: Chapman-Hall.

Kannisto V. and M. Nieminen (1996*). Revised Life Tables for Finland. Population 1996:2.* Helsinki: Statistics Finland.

Koskinen S. and T. Martelin (1995). Why Is Mortality Low Among the Swedish Speaking Minority in Finland? Paper presented at the 11. Nordic Demographic Symposium, June 1995, in Helsinki, Finland.

Land K.C. (1986). "Methods for National Population Forecasts: a Review". *Journal of the American Statistical Association*, 81, 888–901.

Lee R.D. and L. Carter (1992). "Modeling and Forecasting the Time Series of U.S. Mortality". *Journal of the American Statistical Association* 87, 659–671.

Lee R.D. and S. Tuljapurkar (1998). "Uncertain Economic Futures and Social Security Finances". *American Economic Review*, May, 237–241.

Modeen G. (1934a). *Finlands framtida befolkningsutveckling*. Tilastokatsauksia n:o 10. Helsinki: Tilastokeskus.

Modeen G. (1934b). *Suomen väkiluvun tuleva kehitys ja sen taloudelliset seuraukset.* Kansantaloudellinen aikakauskirja VI, 351–378.

Modeen G. and G. Fougstedt (1938). *Laskelmia Suomen vastaisesta väestönkehityksestä*. Sarja VI, Väestötilastoa 89. Helsinki: Tilastokeskus.

National Research Council (2000). Beyond Six Billion: Forecasting the World's Population.

Nordberg L. (1999). "Leo Törnqvist – the "Grandfather" of Finnish Statistics" in Alho J. (ed.) *Statistics, Registries and Science. Experiences from Finland.* Helsinki: Statistics Finland.

Polvinen A. (2001). *Stokastisen ennusteen käyttö julkisen talouden kehityksen arvioinnissa*. Eläketurvakeskuksen raportteja 2001:23. Helsinki: Eläketurvakeskus.

Törnqvist L. (1949). "Näkökohdat, jotka ovat määränneet primääristen prognoosiolettamusten valinnan" in Hyppölä, J, A. Tunkelo and L. Törnqvist (1949) *Suomen väestöä, sen uusiutumista ja tulevaa kehitystä koskevia laskelmia.* Tilastollisia tiedonantoja 38. Helsinki: Tilastokeskus.

Törnqvist L. (1967). "The Post-War Development of Finland Compared with Predictions Made After the War" in *World Population Conference, 1965, vol. III, Projections, Measurement of Population Trends*. New York: United Nations.

Viren M., K Alho, T. Teräsvirta, P. Vartia and Y. Vartia (1981) *Collected Scientific Papers of Leo Törnqvist*. Series A7. The Research Insititute of the Finnish Economy. Helsinki.

Wade A. (1987). *Social Security Area Population Projections 1987*. Baltimore: U.S. Office of the Actuary.

Whelpton P.K., H.T. Eldridge and J.S. Siegel (1947). *Forecasts of the Population of the United States 1945–1975*. Washington D.C.: U.S. Bureau of the Census.

Mortality Projections in Norway[1]

Helge Brunborg

Senior Research Fellow at Statistics Norway, Oslo, Norway

1 A Brief Description of the Norwegian Population Projection Model

The official population projections for Norway are produced and published by Statistics Norway. As in Finland, though not in Denmark and Sweden, the national statistical agency makes regional projections as well. The smallest geographical units projected are the 435 municipalities (*kommuner*), which range in size from about 250 (Utsira) to about ½ million (Oslo) people.

Using the projection model BEFREG, the population by age and sex is projected one year at a time by the cohort-component method. The method employs a migrant pool approach for migration between the 90 economic regions of Norway (NUTS), with 1–19 municipalities in each region. The regional projection results are subsequently broken down into results for individual municipalities according to the size and historical growth rate of broad population age groups in each municipality. Thus, the cohort component method is generally not applied at the municipal level. The national population figures are found by aggregating the regional projections, *i.e.* the bottom-up principle.

The model has been virtually unchanged during the last 15–20 years; see Rideng, Sørensen and Sørlie (1985) for a general description and Hetland (1998) for a technical description of the computer system.

In the most recent projections, for the period 1999–2050 and with the registered population as of 1 January 1999 as the initial population, we assumed

[1] After this paper was written, a new set of population projections for Norway for the period 2002-2050 was published in December 2002, see http://www.ssb.no/folkfram/. One important change compared to the 1999–2050 projections is that the assumed life expectancies up to 2050 are considerably higher than previously. The method for projecting the age-specific death probabilities is the same as that for the 1999–2050 projections.

three variants for each of the following demographic components: fertility, mortality, net immigration and the degree of centralisation for internal migration (plus a variant with zero migration). This would yield 144 different population projections; however, only a few of these combinations have been computed and published. The populations of municipalities were projected until 2020, those of the counties were projected until 2050 (but published only for the years up to 2030), and the population for the entire country was computed until 2050.

All data for the projections come from registers through the population-statistics system BESYS, which is used to build up a structure of aggregate data for population, births, deaths, domestic and international migration, where the smallest unit is age*sex*municipality.

As mentioned above, BEFREG projects the population only by age, sex and region. Previously, Statistics Norway has also made national projections by age, sex and marital status (Brunborg, Mønnesland and Selmer 1981; Kravdal 1986) and by age, sex and household status (Keilman and Brunborg 1995). The last two of these publications used mortality rates by formal marital status, whereas the first did not. Moreover, the stochastic microsimulation model MOSART projects a sample (from one to ten per cent) of Norway's population by age, sex, marital status, household status, educational activity and level, labour-market earnings and public-pension status/benefits (including disability and old-age). In this model the mortality probabilities have been estimated from 1993 data with sex, age, marital status, educational attainment and disability status as covariates (Fredriksen 1998). MOSART has a complicated data structure that is infrequently updated.

2. A Short History of Mortality Projections in Norway

Since 1969, thirteen sets of regional and national projections have been prepared and published, usually every three years.[2] In the first projections the mortality rates were kept constant for the entire projection period and set equal to the most recently observed rates, usually for a period ranging from two to five calendar years for national rates and ten-year periods or more for regional rates and time trends. The use of observations for several years is

[2] See Texmon (1992) for a survey of Norwegian population projections for the period 1969–1990.

done to reduce random variations due to Norway's small population (about four million).

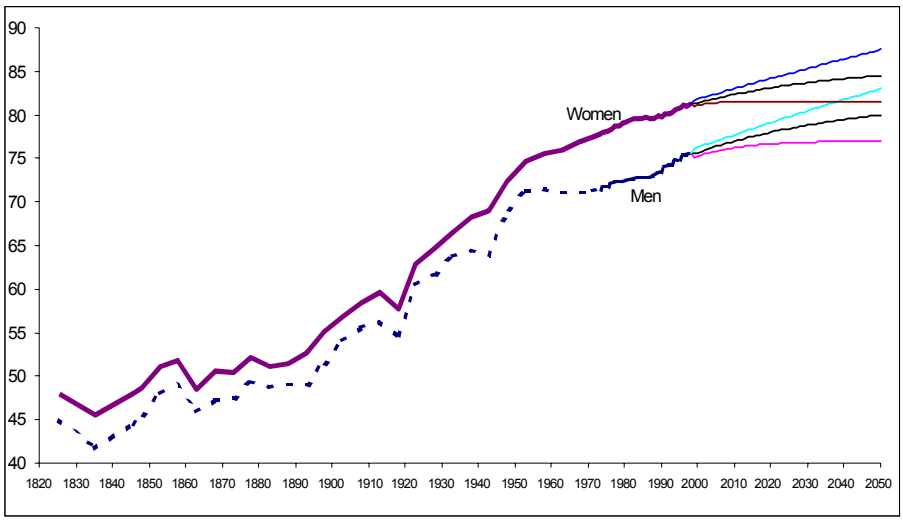
Gradually the projections of the age- and sex-specific mortality rates have become more sophisticated:[3]

- In the projections of 1977 and 1979, the observed rates were reduced by two per cent and three per cent, respectively, and kept constant for the entire projection period (until 2010).
- Regional mortality rates (specific for each of the 19 counties) were introduced in the projections of 1979. This affects overall mortality if, for example, net migration flows go primarily from high to low-mortality areas.
- A declining trend in future mortality rates was assumed for the first time in the 1982 projection, when all rates were reduced by one per cent per year for the first ten years but kept constant for the rest of the projection period.
- The Brass logit life-table model was used for the projections of 1987 (but not later).
- A model with time- and age-specific death rates was used for the period 1990–2050 (but not later); see Statistics Norway (1991) and Goméz de Leon and Texmon (1992).
- Alternative mortality projections were introduced in the 1993 projections, when sets of rates yielding low, medium and high life expectancies were assumed (Statistics Norway 1994).
- Target life expectancies for the final projection year were also introduced in the 1993 projections (Statistics Norway 1994).
- Age- and sex-specific reductions in future death rates were used in the 1999–2050 projections.

Most of these changes were made because it was discovered that mortality had been persistently overestimated. It is perhaps surprising that it took so long to realise this, since Norway had experienced an almost uninterrupted mortality decline since the 1820s (though with some stagnation for men in the 1950s). The decline has been particularly rapid since the 1960s – see Figure 1.

[3] For more detail, see the publication for each set of projections, with text in both Norwegian and English, the most recent being Statistics Norway (2002).

Figure 1 **Life Expectancy at Birth for Women and Men. Registered 1825–1998 and Projected 1999–2050: Low, Medium and High Assumptions of Life Expectancy**



3 Current Methodology of Mortality Projections

In this section, we will describe the methodology and thinking behind the current mortality projections, focusing on a number of separate issues that need to be considered.

3.1 Target Life Expectancies

The assumed target life expectancies at birth for the final projection year, 2050, vary from 77 to 83 years for men and from 81.5 to 87.5 years for women *(i.e.* the same as for the previous projections, those of 1996) – see Figure 1. There is no theory or profound thinking behind these assumptions, which have been based on the development in Norway and elsewhere:

- The assumed future increase of e_0 – 7.5 years for males and 6.3 years for women – in the most optimistic alternative from 1998 to 2050 is of the same magnitude as the *actual* increase during the previous 50-year period from about 1950 to 1998 (5.5 and 7.7 years, respectively).
- In the medium UN series, it is assumed that e_0 for Norway will increase to 80.5 years for men and 86.4 years for women in 2040–2050 (United Nations 1999), *i.e.* well within our range. The long-term UN projections to 2150 assume that e_0 will increase to 85.2 for men and 91.3 for women in

Europe and to 87.1 and 92.5 in North America; these constitute the upper limits of the projections.

- The most recent Swedish projections assume life expectancies for 2050 that are slightly lower than in our high alternative, 82.6 for men and 86.5 for women (Statistics Sweden 2000).

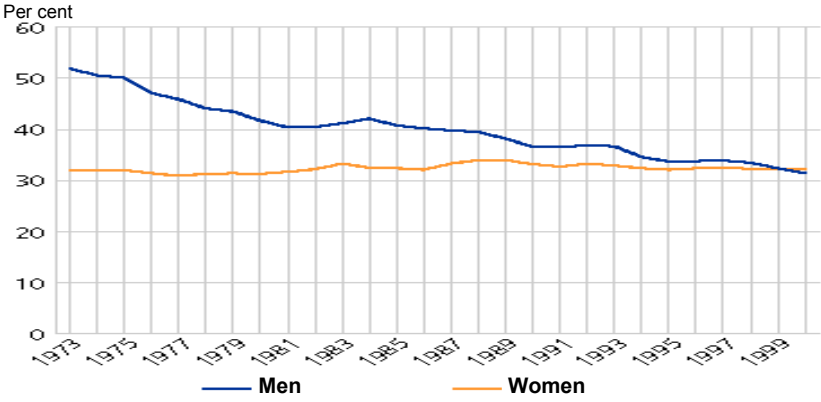
We conclude that our assumptions about target life expectancies in 2050 are not extreme and perhaps even slightly conservative. In our next projections, we will consider assuming a wider range of life expectancies. It would be useful to base this range on probabilistic considerations, for example, the work done by Juha Alho and Nico Keilman – see *e.g.*, Keilman, Pham and Hetland 2002.

3.2 Difference in Target e_0 for Males and Females

We have assumed a continued gradual decline in the difference between female and male life expectancies, to 4½ years in 2050. This is consistent with historical trends, where the difference increased from about 3½ years around 1950 to almost 7 years in the 1980s, declining later to 5.7 years in 1998. This decrease, which has been observed in most European countries, is usually explained as due to a narrowing of the gender differences in life style. A similar assumption has been made in Sweden, where the differential is reduced to 3.9 years for 2050.

The most specific manifestation, as well as the principal reason, for this tendency is probably the change in smoking behaviour. The proportion of daily smokers among men 16–76 in Norway has decreased from about ½ in 1973 to ⅓ in 2000, while it has remained constant at ⅓ for women. In 2000, smoking had become more common for the first time among women than among men (32 per cent versus 31 per cent) – see Figure 2.

Figure 2 Proportion of Daily Smokers among Men and Women 16–74 Years of Age, 1973–2000



Source: http://www.ssb.no/emner/03/01/roy/

3.3 Life Expectancies in the First Projection Year

If we had assumed a smooth development of e_0 from the most recent observations to the target, there would have been practically no difference between the alternatives for the first years of the projection. We would like, however, to have a reasonably wide range of e_0 to cover the possibility of random year-to-year fluctuations in mortality. For this purpose, we chose, as described in Section 5, a parameter that yields a change in e_0 for the first projection year (*i.e.* from 1998 to 1999) that is similar to the largest observed annual change in e_0 during the period 1965–1998. In other words, we chose the largest annual *decrease* for the low alternative – about 0.4 years for men 0.2 years for women – and the largest annual *increase* for the high alternative – about 0.6 years for each sex (see Table 1). In 1996 a similar approach was taken to widen the mortality range in the first projection year, but then the standard deviation of the difference between observed and *projected* life expectancies was subtracted (or added) to yield the low (or high) values for the first projection year. Actual and assumed life expectancies for the first projection years are shown in Figures 3 and 4. It can be seen that the observed values are close to the medium variant in both rounds of projections, with a slightly faster improvement for men than for women.

Table 1 **Change Factors and Target Life Expectancies, Population Projections 1999–2050***

	First projection year (1999)		*Subsequent projection years (2000 ... 2050)*	
Life expectancy variant	*Parameter α_s*	*Assumed life expectancy*	*Parameter β_s*	*Target life expectancy*
Males				
L (low)	3.315	75.1	0.927	77.0
M (medium)	0.741	75.5	0.982	80.0
H (high)	–3.026	76.1	1.005	83.0
Females				
L (low)	1.417	81.0	0.791	81.5
M (medium)	–0.212	81.2	0.978	84.5
H (high)	–4.195	81.7	1.006	87.5

* The observed life expectancy at birth in 1998 was 75.5 years for males and 81.2 years for females.

Figure 3 **Life Expectancy at Birth for Men, Registered 1994–2000 and Projected to 2001**

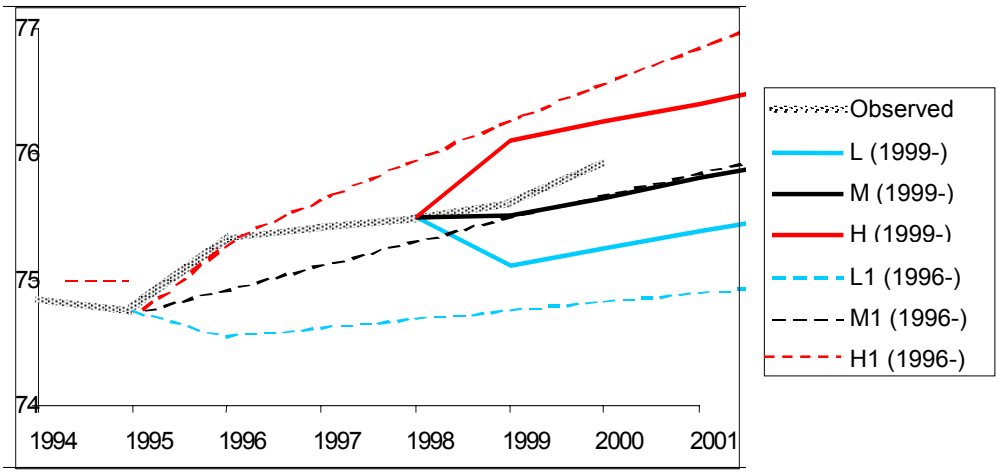
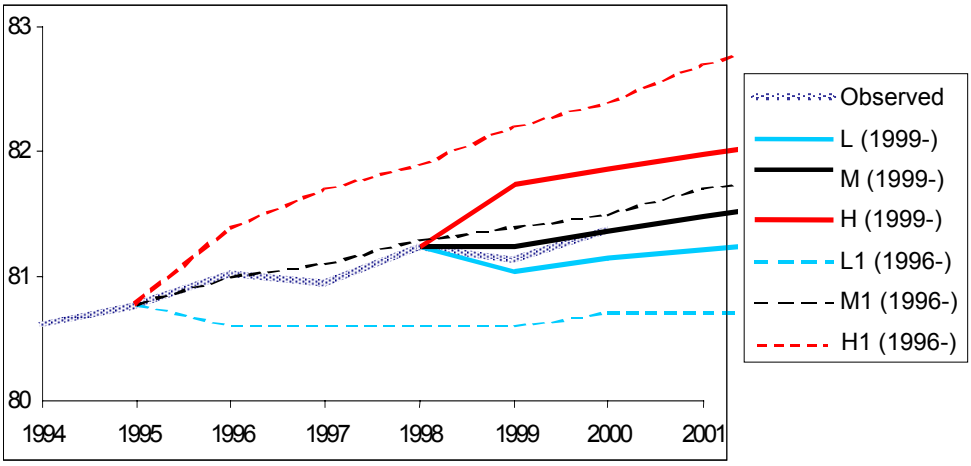


Figure 4 Life Expectancy at Birth for Women, Registered 1994–2000 and Projected to 2001



3.4 Path of e_0 from the Initial until the Target Year

In Section 5, we describe the method used to project the mortality rates in the 1999–2050 population projections for Norway. The e_0 trajectory is found by interpolating between the life expectancies assumed for the initial and target years. This is done by assuming a dampening of the annual age- and sex specific rates of change in death rates. Figures 5 and 6 show e_0 for males and females for each of the three mortality alternatives in the two most recently completed projections, *i.e.* for 1996–2050 and 1999–2050.

We notice that the 1999 projections generally assume a more linear development of e_0 than the 1996 projections, except for the low alternative. The main reason is that we did not impose the restriction of no mortality change in the target year, as discussed below. It is, however, not yet possible to determine whether the 1999 paths are more realistic then the 1996 paths – only time will tell!

Figure 5 Life Expectancy at Birth for Males, Registered 1970–1999 and Projected to 2050

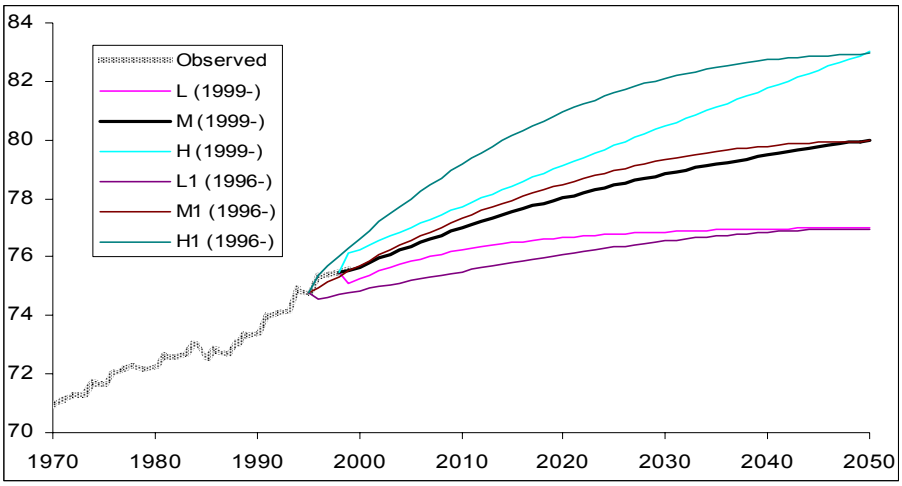
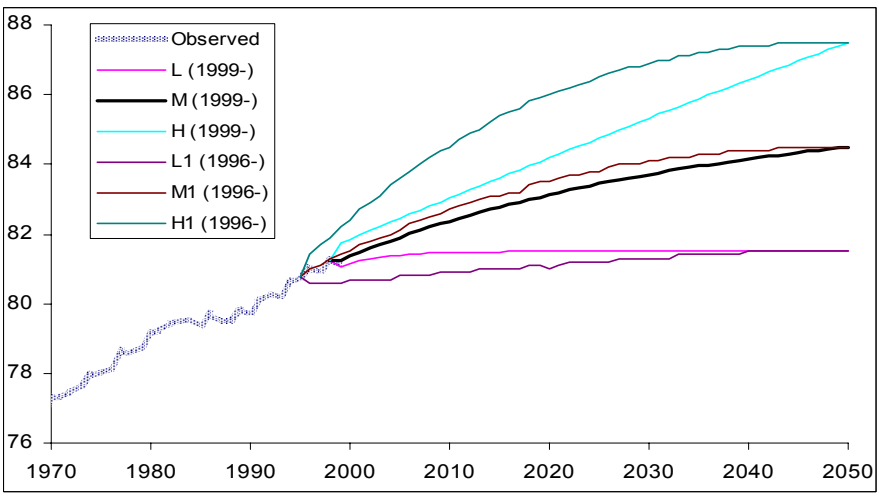


Figure 6 Life Expectancy at Birth for Females, Registered 1970–1999 and Projected to 2050



3.5 Slope of e_0 in the Target Year

In the previous round of projections, for 1996–2050, it was assumed that life expectancy would cease to decline in 2050 due to high uncertainty about mortality in such a distant future. In the most recent projections, however, we

have not been concerned about the slope of e_0 in the target year. This is due partly to the high uncertainty, but also to the likelihood – in our opinion – that mortality will continue to decline, even after 2050.

3.6 Alternative mortality assumptions

As mentioned in the introduction, Statistics Norway did not begin to introduce alternative mortality assumptions before 1993. Although future fertility (and migration) may seem more uncertain than future mortality, there are a number of uncertainties related to the development of mortality. To mention briefly just a few: technological breakthroughs in the diagnosis and treatment of diseases, new epidemics and other diseases, pollution and other environmental problems, increasingly unhealthy life styles, etc.

Thus, it seems wise to base population projections on several mortality alternatives. The alternatives should cover a realistic future range of variation. If the range is too small, there are in reality no alternatives; if it is too large, the projections may cease to be interesting. Ideally, the range should be based on a probability distribution.

The number of alternatives is another choice that needs to be considered. The number should be greater than one but not so large that it becomes confusing and complicated to present and use the population projections. Thus, three seems to be a sensible number. In the last three rounds of projections, we have assumed one low, one medium and one high alternative for life expectancy. Note that "low", "medium" and "high" do not refer to mortality levels as such but to life expectancies, for consistency with the assumptions about other demographic components; for example, "low" implies low population growth.

3.7 Age Groups

The current version of BEFREG has 100 age groups, 0,1,2,...,98 and 99+. We are considering including more single-year age groups, at least age 99, in the next version of the model for two reasons: First, there is an increasing number of (and interest in) centenarians. Second, the lack of a decline in mortality for the oldest of the old, as described in Section 4, may necessitate a more differentiated approach for this particular group.

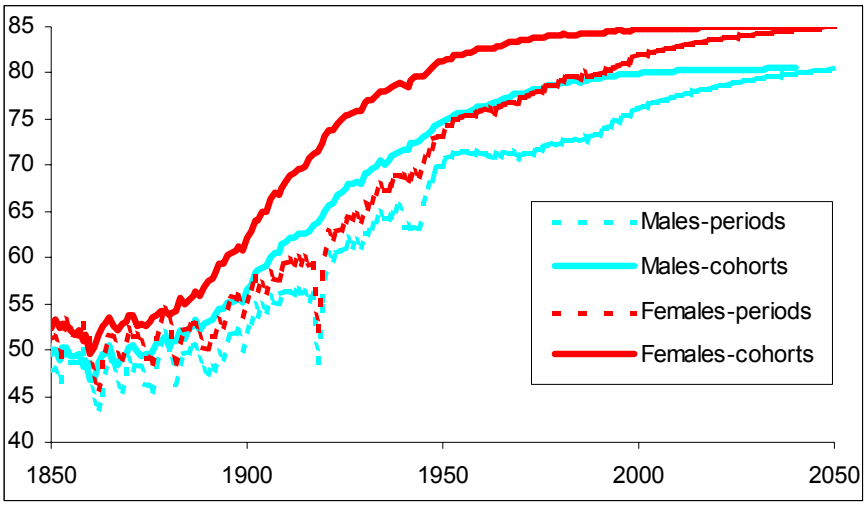
3.8 Cohort Mortality

Cohort mortality is not directly considered in our mortality assumptions. However, we have calculated the implications of our assumptions for cohort mortality – see Figure 7.[4] We may note that projected cohort mortality approaches projected period mortality; this tendency may be expected in view of the assumptions made. We also see that the difference between cohort and period mortality is particularly high for the 1960s–1980s.

4 Age-specific Trends in Mortality Rates

The future age pattern of mortality has been given little attention in previous Norwegian population projections. The reason may be that the past mortality decline was implicitly assumed to be about the same for all ages and perhaps also that the age pattern of mortality was not considered to matter so much for the population projections – the only important factor being the level of mortality as measured by life expectancy at birth.

Figure 7 Life Expectancy at Birth for Periods and Cohorts, Registered and Projected



[4] The life expectancy for cohorts born in 1950 and later and having members still living in 2050 has been estimated from extrapolated death probabilities. For example, q(101, c1950) = q(101, c1949).

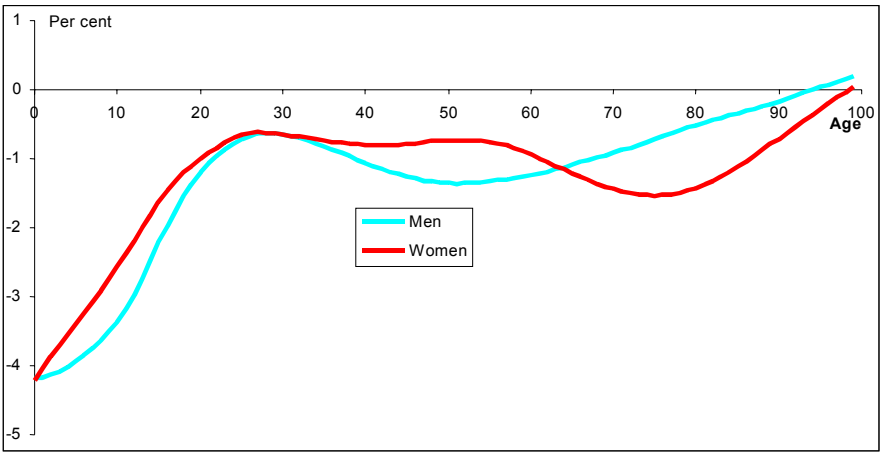
It is clear, however, that the mortality decline has varied greatly by age and sex. It is well known that historically the decline was largest for infants and children. To learn more about recent trends, we have studied the development of age-and sex-specific mortality rates for the past 30–40 years.

Let q(x,t,s) be the probability of death at age x, time t and sex s. The annual relative rate of change during the period from t_1 to t_2 is

(1) $\check{r}(x, s) = \ln(q(x, s, t_2) / q(x, s, t_1)) / (t_2 - t_1)$.

We estimated the rate of change for x = 0,1,...99; t_1 = 1965–1968, t_2 = 1998; and s = male, female.[5] As these rates of change exhibit a rather ragged appearance we applied a smoothing procedure. The rate of change r(x,s) is graduated using a 21-term weighted moving average, with coefficients from Hoem (1995). The smoothed rates of change for males and females are shown in figure 8.

Figure 8 **Annual Change in Age-Specific Death Probabilities, 1965–1998. Per cent. Men and Women by Age. Graduated by 21-term Weighted Moving Average**



As expected, the decline has been the most rapid for children, but it has also been very impressive for adolescents and for men and women between 40

[5] We experimented with different periods, finding similar patterns, and chose the oldest single-year age-specific death rates that were available to us when we performed this analysis, i.e. for 1965.

and 80. On the other hand, it has been nearly zero for persons over 90. There is even a tendency of *increasing* mortality for the oldest individuals, especially men over age 95, see Figures 9–10. Thus, mortality rates for the oldest in Norway may have stagnated or started to increase in recent years. This is contrary to the trends in most other countries, but similar to those in the Netherlands (Nusselder and Mackenbach 2000). There are also indications of stagnating old-age mortality trends in Denmark, especially for men (Danmarks Statistik 2000), and in the USA (Kranczer 1997).

Figure 9 **Expected Remaining Years of Life for Elderly Men at Selected Ages**

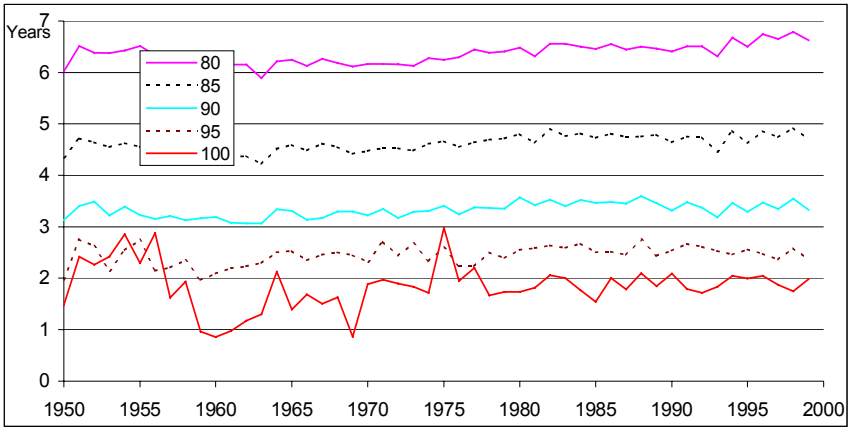
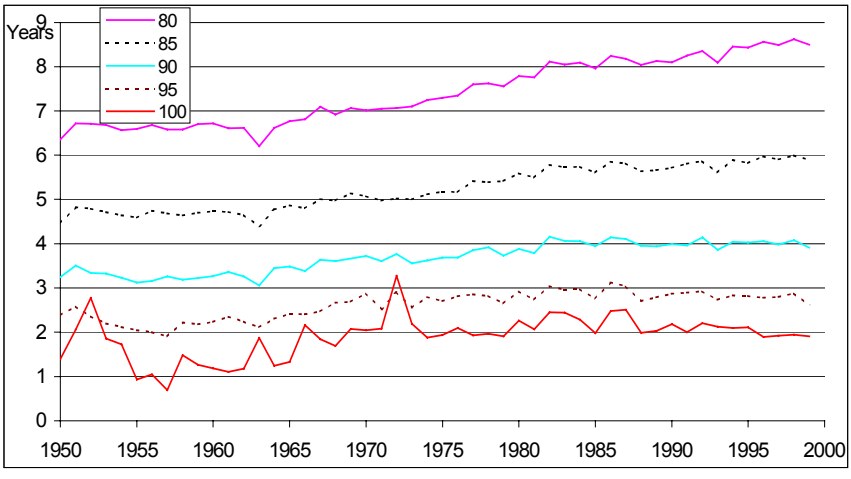


Figure 10 **Expected Remaining Years of Life for Elderly Women at Selected Ages**



5 Projections of Age-Specific Mortality Rates

In the most recent projections, we assume that the age-specific mortality declines are consistent with the patterns shown in Figure 8 and as estimated in (1). To project the mortality probabilities q(x, t, s) by age x, time t and sex s, we multiply them by a factor depending on the rates of change ř (x, t, s) estimated for the period 1965–1998, as shown in Figure 8. These rates of change are also changed, however, one year at the time, through multiplication by the parameters α_s or β_s for each age.

Thus, for the first projection year, 1999, we find

(2) q(x, s, 1999) = q(x, s, 1997.5) * (1 + r(x, s, t)), where

(3) q(x, s, 1997.5) = (q(x, s, 1997) + q(x, s, 1998))/2

and 1997.5 denotes the average of 1997 and 1998. For 1999 the change factors are

(4) r(x, s, 1999) = ř(x, s) * α_s.

For the subsequent years we compute

(5) q(x, s, t) = q(x, s, t–1) * (1 + r(x, s, t)) for t = 2000,...,2050, where

(6) r(x, s, t) = ř(x, s) * β_s for t = 2000 and

(7) r(x, s, t) = r(x, s, t–1) * β_s for t = 2001,...,2050.

Equation (7) determines the path of e_0 from the most recently observed values of q_x (the mean of 1997 and 1998) to the assumed target values for 2050 (see Table 1). The estimates of parameters α_s and β_s employed here imply a dampening of the annual rates of change of the age- and specific death probabilities, see Table 1. The parameters are determined in the following way, for each sex and mortality alternative (low, medium and high):

- The parameter for the first projection year, αs, is chosen on the following assumptions about the *change* in life expectancy from 1998 to the first projection year (1999), to obtain a wide range of death probabilities rates for the first year:
  - It is set equal to the largest *decline* in e_0 observed during 1965–1998 in alternative L
  - It is set equal to zero in alternative M

  - It is set equal to the largest *increase* in e_0 observed during 1965–1998 in alternative H

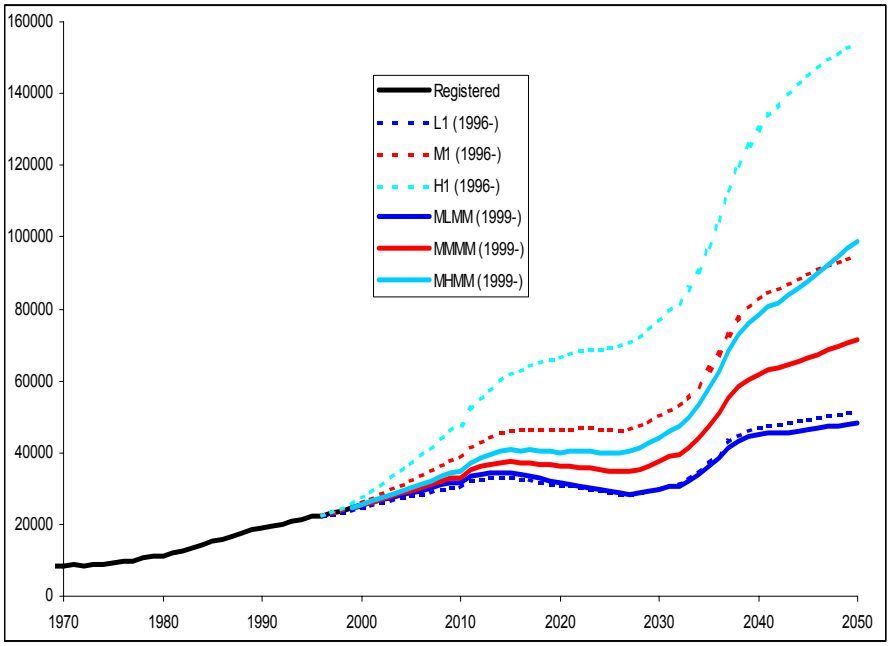
- The parameter for the rest of the projection years, β_s, is chosen such that the resulting life expectancy in 2050 is equal to our target life expectancy for that year – see Table 1. This is done through iteration. (The procedure is made easy by using an Excel spreadsheet function that lets the user set the target whereby the program estimates the value that yields the desired value of the target.)

6 Projection Results

The two most recent sets of population projections for Norway, *i.e*. those for 1996–2050 and 1999–2050, are very similar with regard to most of the assumptions. The fertility and migration assumptions are almost the same, and the target life expectancies in 2050 are identical although the trajectories are slightly different. As mentioned above, the greatest difference is that we have assumed a different age-pattern of mortality change for the projection period. This difference, and in particular the slower mortality decline for the oldest, result in significantly lower numbers of old people than in the previous projections (Figure 12).[6] The results are very similar for the low alternatives, where only a very small mortality decline has been assumed. However, the *high* alternative of the 1999 projections yields about the same number of people 90+ as the *medium* alternative of the 1996 projections. For 2050, for example, the projected population 90+ varies from 50,000 to 150,000 in the previous projection to 50,000–100,000 in the most recent projections.

[6] The three different projections from 1999 shown here differ only with regard to the mortality assumptions. The 1996-projections are also different with regard to fertility and migration, however, but for the age groups and period considered here, the effects of these differences are marginal. Fertility differences for 1996–2050 will obviously not affect the number of persons 90+ for this period. The youngest age in 1996 of persons to enter age group 90+ in the projection period 1996–2050 is 36 years. Since persons aged over 35 accounted for only 12 per cent of net immigration in 1996–99, differences in net immigration can only have marginal effects on the number of persons 90+ projected for the period 1996–2050.

Figure 12 Number of Persons 90+. Registered 1970–2001 and Projected 1996–2050 and 1999–2050



We conclude that the specification of the age-structure of mortality decline has significant effects on the projected number of old people. Thus, the analysis of mortality trends for the oldest members of the population is important for population projections.

References

Brunborg H., J. Mønnesland and R. Selmer (1981). *Projections of the population by marital status 1979-2025.* Rapporter 81/12. Oslo-Kongsvinger: Statistics Norway.

Danmarks Statistik (2000). *Befolkningens bevægelser 1999*. Copenhagen: Statistics Denmark.

Fredriksen D. (1998). "Projections of Population, Education, Labour Supply and Public Pension Benefits: Analyses with the Dynamic Microsimulation Model MOSART". *Social and Economic Studies 101.* Oslo-Kongsvinger: Statistics Norway.

Goméz D.L., J. and I. Texmon (1992). "Methods of Mortality Projections and Forecasts" in Nico Keilman and Harri Cruijsen (eds.). *National Population forecasting in industrialized countries.* Amsterdam/Lisse: Swets & Zeitlinger.

Hetland A. (1998). *Systemdokumentasjon for BEFREG.* Interne dokumenter 98/4. Oslo-Kongsvinger: Statistics Norway.

Hoem J. M. (1995). "Moving-Average Graduation of the Timing of the Historical Demographic Transition in Sweden" in C. Lundh (ed.). *Demography, Economy and Welfare / Scandinavian Population Studies 10*. Lund: Lund University Press.

Keilman N., D. Q. Pham, and A. Hetland (2001). "Norway's Uncertain Demographic Future". Social and Economic Studies 105. Oslo: Statistics Norway. http://www.ssb.no/emner/02/03/sos105/.

Keilman N. and H. Brunborg (1995). "Household Projections for Norway, 1990-20 Part I: Macrosimulations". Report 95/21. Oslo-Kongsvinger: Statistics Norway.

Kranczer S. (1997). "Record-High U.S. Life Expectancy". *Statistical Bulletin* Oct-Dec: 2-8.

Kravdal Ø. (1986). "Framskriving av folkemengden etter kjønn, alder og ekteskapelig status 1985-2050". Rapporter 86/22. Oslo-Kongsvinger: Statistics Norway.

Nusselder W. J. and J.P. Mackenbach (2000). "Lack of Improvement of Life Expectancy". *The Netherlands. International Journal of Epidemiology* 29: 140-148.

Rideng A., K. Ø. Sørensen and K. Sørlie (1985). "Modell for regionale befolkningsframkrivinger". Rapporter 85/7. Oslo-Kongsvinger: Statistics Norway.

Statistics Norway (1991). *Population Projections 1990-2050. National and Regional Figures.* Official Statistics of Norway, NOS B983. Oslo-Kongsvinger: Statistics Norway.

Statistics Norway (1994). *Population Projections 1993-2050. National and Regional Figures.* Official Statistics of Norway, NOS C176. Oslo-Kongsvinger: Statistics Norway.

Statistics Norway (1997). *Population Projections 1996-2050. National and Regional Figures*. Official Statistics of Norway, NOS C414. Oslo-Kongsvinger: Statistics Norway.

Statistics Norway (2002). *Population Projections 1999-2050. National and Regional Figures*. Official Statistics of Norway, NOS C693. Oslo-Kongsvinger: Statistics Norway.

Statistics Sweden (2000). *Sveriges framtida befolkning. Befolknings-framskriving för åren 2000-2050*. Demografiska rapporter 2000:1. Stockholm: Statistics Sweden.

Texmon I. (1992). "Norske befolkningsframskrivinger 1969-1990" in O. Ljones, B. Moen and L. Østby (eds.). *Mennesker og modeller. Livsløp og krysløp*. Sosiale og økonomiske studier nr. 78. Oslo-Kongsvinger: Statistics Norway.

United Nations (1999). *World Population Prospects*. The 1998 Revision. Volume I: Comprehensive Tables, ST/SER.A/177, United Nations, New York.

Mortality Assumptions for Sweden. The 2000–2050 Population Projection[1]

Hans Lundström

Senior Statistician, Statistics Sweden

Mortality Projection in Sweden

A new population projection for Sweden is prepared every third year with minor updates in between. Statistics Sweden is responsible for the projections.

Ideally, mortality projections should be made in a process-oriented manner. In practice, however, they are mainly based on trend extrapolations of period mortality rates for the last 50 years. The critical component in the assumptions concerns the ages of 50 and above. The problem is to handle both short-term and long-term developments, especially when there is a trend shift like the one today with a temporary slow-down in the mortality decline for middle-aged women. Does this change mark a new trend or not? Most users are mainly interested in the short-term development, *i.e.* in the next year or the next five years. Others have a time horizon of 50 years or longer. There are three alternative assumptions: no change in mortality, mortality decline according to the main alternative, and a more pronounced mortality decline.

The assumption of the future mortality decline is based not only on the observed trend but adjusted for other information such as smoking behavior.

For the coming projection we plan to base the analysis more on cohort mortality.

Mortality in Sweden has fallen ever since the mid-19th century. In the beginning, the change was mainly due to a reduced risk of dying of infectious dis-

[1] To a large extent, this is a translation from Swedish of Chapter 3.3 in the publication Sveriges framtida befolkning. Befolkningsframskrivning för åren 2000–2050. Demografiska rapporter 2000:1 SCB. (Sweden's future population. Population forecast for the years 2000–2050. Demographic reports 2000:1, Statistics Sweden). ISBN 91-618-1068-1.

eases and deficiency diseases. The factors underlying the greater chances of survival were economic, social and sanitary improvements, and, not least, medical advances such as the introduction of vaccines and antibiotics.

More recent developments – say, since 1950 – have brought a continued decline in mortality. Changes in the last few decades have largely concerned chronic illnesses, including cardiovascular diseases and cancer, which are the major causes of death. The reasons for the changes in what are sometimes called the diseases of wealthy societies are a transition to a healthier lifestyle and improved medical care, leading to a considerable increase in survival at advanced ages. The decline in mortality at ages above 65 began considerably earlier for women than for men.

Sharply Lower Mortality in 1950–1999

In the second half of the 20th century, mortality fell sharply. The risk of death has been reduced by more than half among men below the age of 50 and women below the age of 80. The drop in infant mortality has been particularly dramatic. In 1950, 21 of 1,000 children born died before their first birthday. In 1999, just three per thousand live-born children died in their first year.

Among adult men, however, the risk of death changed relatively little in the period 1950–1975. For part of this period, there was even an observable increase in mortality, largely among middle-aged men. Since 1975, however, male mortality has declined substantially, by an average of about 2 per cent per year among men between young middle age and the age of about 80, (see Figure 1).

Among women, we see a decline in mortality throughout the post-1950 period. The annual rate of mortality reduction has averaged 1–2 per cent. At ages in excess of 85, the reduction of mortality has been somewhat lower. However, over the last ten years the rate of mortality reduction among upper-middle-aged women has slowed slightly, (see Figure 2).

Figure 1 **Annual Reduction of Risk of Death at Different Ages for Two Periods, 1950–1999. Men**

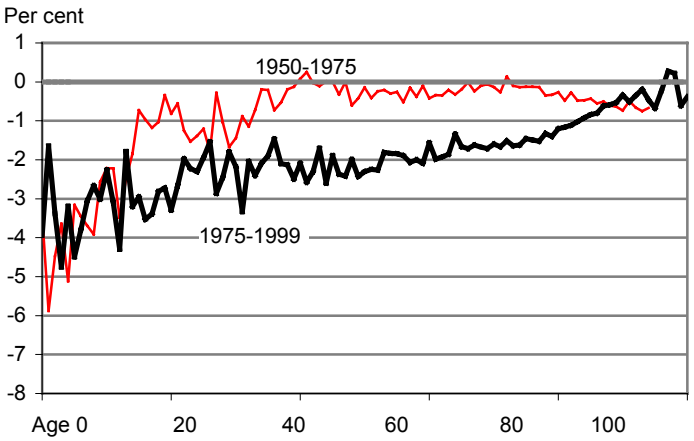
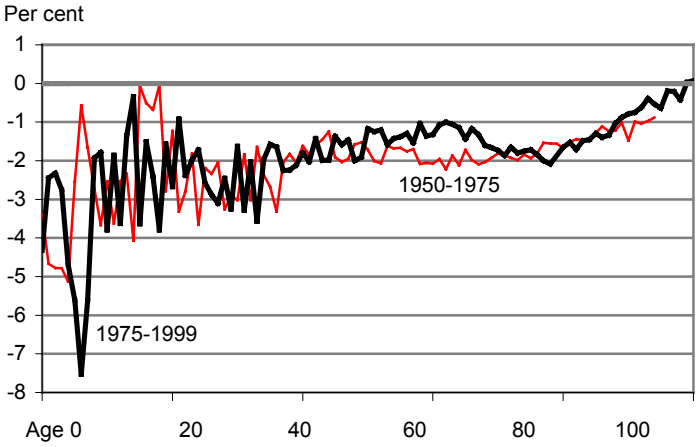


Figure 2 **Annual Reduction of Risk of Death at Different Ages for Two Periods, 1950–1999. Women**



We may add that in recent years severe influenza epidemics have occurred more frequently. This has had a serious effect on many elderly people. The long-term rise in women's annual average life expectancy has therefore slowed temporarily after each influenza epidemic. One reason why women's average life expectancy is affected most is that a larger proportion of elderly women than men reach advanced ages.

Reasons for the Decline in Mortality in 1980–1999

Assumptions about future mortality trends are based on changes in mortality over the past two decades. During this period, the decline in male mortality has strongly resembled the decline in female mortality. In spite of this close correspondence, around 1980 the situation differed for men and women aged over 50 – and these are critical ages in the assumptions used in the forecast.[2]

The rising mortality observable for men until the end of the 1970s was caused by an increase in deaths due to cardiovascular diseases and cancer. Changes in lifestyles helped to break this trend in the 1980s, when mortality began to fall. The proportion of people smoking every day has declined since the end of the 1970s, a tendency with a significant impact on the trends for cardiovascular diseases and cancer. Less fatty food and increased exercise have probably been other important factors behind the decline in cardiovascular diseases. Alcohol consumption has also fallen during this period.

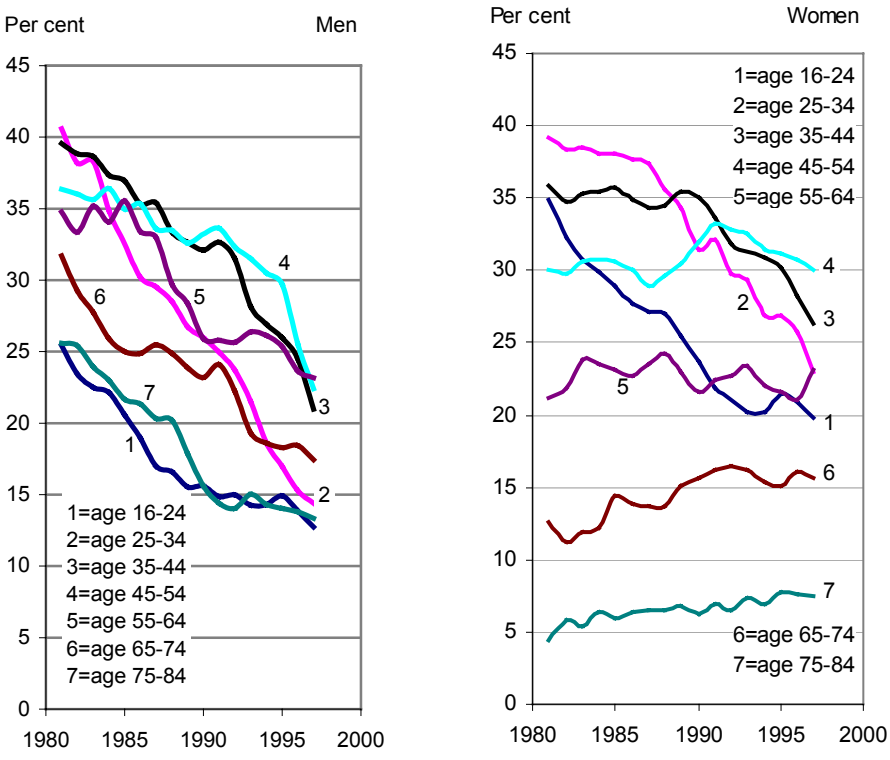
The evident rise in mortality for men until the end of the 1970s was not observable among women. However, a slight tendency towards higher mortality due to ischemic diseases has been discernible, along with a relatively sharp rise in mortality from lung cancer. Nevertheless, total mortality fell, though at a lower rate. The reasons for the reduction in female mortality over the last 20 years have probably been about the same as those affecting male mortality, but with one major exception: the proportion of smokers has continued to increase gradually at more advanced ages. This is due to a generational change involving smokers. The proportion of women who have smoked at some time increases in each successive generation over the age of about 30, (see Figure 3). In spite of this, mortality from cardiovascular diseases has diminished, whereas mortality due to lung cancer, which is linked more closely to smoking habits, has continued to rise.

The medical treatment of cardiovascular diseases in particular has improved, and this has had a significant impact on the decline in the risk of death. A simple indicator of the changes is that mortality due to cardiovascular diseases (heart attacks) has fallen considerably more rapidly than the risk of falling ill (incidence).

---

[2] Except in the very long term, assumptions on relative changes in younger age groups have a limited impact on the numerical strength of the population, since mortality is so low at these ages.

Figure 3 Proportion of Daily Smokers by Sex, 1981–1997 *



*) Moving averages (3-year).
Source: Living Conditions Survey. (ULF, Statistics Sweden)

Higher Average Life Expectancy in 1950–1999

Mortality trends since 1950 have resulted in an increase in the average life expectancy of men from 69 to 77 years, an average increase of 0.16 years per calendar year. For women average life expectancy went up from 72 to 82 years, an average increase of 0.20 years per calendar year.

Table 1 below shows changes in life expectancy between different periods in the second half of the 20th century. Table 1 also reveals the positive impact on average life expectancy (at birth) of the improvements in mortality at different ages. The gains for men in recent years consist mainly of mortality improvements among young and middle-aged men, but improvements among the elderly have also made a major contribution. For women, the trend among the elderly accounts for most of the increase in average life expectancy over the same period.

Table 1 Change in Average Life Expectancy at Birth Between Different Periods (in Years). Total Change and Distribution by Different Age Intervals

Sex / *Period*	*Change (in years)*			
	Total	*of which in age group*		
		0–19	*20–64*	*65–*
Men				
1951–55 to 1956–60	0.7	0.3	0.3	0.1
1956–60 to 1961–65	0.4	0.3	0.1	0.0
1961–65 to 1966–70	0.3	0.3	–0.1	0.1
1966–70 to 1971–75	0.2	0.3	–0.1	0.1
1971–75 to 1976–80	0.4	0.3	–0.0	0.1
1976–80 to 1981–85	1.1	0.2	0.5	0.4
1981–85 to 1986–90	0.8	0.0	0.4	0.4
1986–90 to 1991–95	1.2	0.2	0.6	0.5
1991–95 to 1998 *)	1.3	0.1	0.6	0.5
Women				
1951–55 to 1956–60	1.2	0.3	0.6	0.4
1956–60 to 1961–65	1.0	0.2	0.3	0.5
1961–65 to 1966–70	0.9	0.3	0.2	0.5
1966–70 to 1971–75	1.1	0.2	0.2	0.7
1971–75 to 1976–80	0.9	0.2	0.1	0.5
1976–80 to 1981–85	1.0	0.1	0.3	0.6
1981–85 to 1986–90	0.7	0.1	0.1	0.5
1986–90 to 1991–95	0.8	0.1	0.2	0.5
1991–95 to 1998 *)	1.0	0.1	0.3	0.6

*) 1998 is the last point in time (the distance from the middle year of the previous period is five years here, as in the other cases).

Future Mortality

As stated above, improved living conditions in a range of areas are significant factors underlying the decline in mortality in recent decades. Given present trends, there is reason to hope for continued improvements in living conditions and lifestyles. We know that fewer and fewer young people are taking up smoking and increasing numbers are exercising regularly in their spare time, factors that are important to health and life expectancy. It is worth noting that even if no major improvements were to occur in future, the long-term (longitudinal) impact on mortality at a given age would be similar to that observed to date (perhaps for several decades). In certain cohorts, people could enjoy a favourable life expectancy throughout their entire lives, assuming the levels attained in the 1980s and 1990s are sustained (for factors like consumption, exercise, and men's smoking habits).

Nevertheless, there are lifestyle factors that give rise to concern. Even if smoking is now becoming less common among young people, there is a considerable difference between the smoking habits of elderly and middle-aged women, (see Figure 3). At present, relatively few elderly women are smokers or former smokers. The number will increase during the forecast period, as those who are middle-aged grow old, and this may put a brake on the decline in mortality. As a result, we have assumed that the long-term decline in mortality will be less marked for women than for men. The increasing proportion of people who are overweight, greater stress in professional life, and a possible rise in alcohol consumption in the future are some examples of trends that could slow the decline in mortality. Better information on health matters and improvements in workplace organisation, in the broad sense of this term, may moderate such effects.

Medical progress has had a positive impact on mortality trends. In all probability, the positive trend observed until now in the medical area will continue, and these medical advances may help to improve quality of life and increase life expectancy. The possible impact of potential breakthroughs in genetic engineering and biotechnology surpasses our present comprehension. However, as serious illnesses become more curable, a higher proportion of elderly people will have previously had such illnesses. Despite successful treatment at the time, this factor may have a negative impact on mortality among the very oldest.

Thus, numerous trends may potentially have a – positive or negative – impact on mortality. However, it is hardly possible to quantify the effect of these factors with any precision. We should bear in mind that until now, mortality has changed slowly. Accordingly, we assume that in the immediate future,

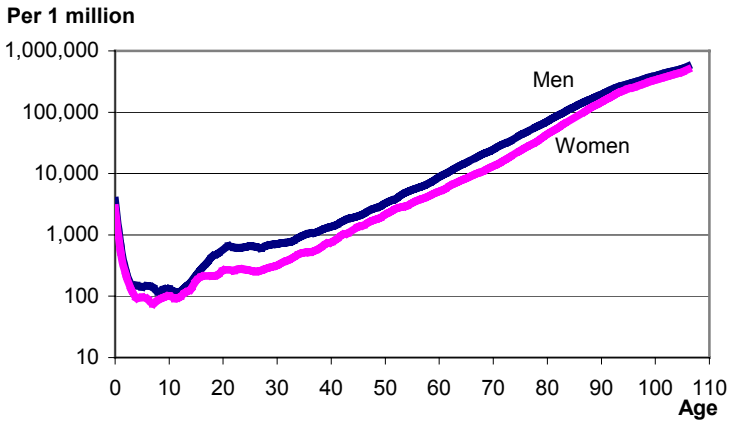
mortality will continue to follow the trend prevailing up to this point. In the longer term, we assume that the reduction in the risk of death will continue throughout the forecast period[3] but will be slowed somewhat by the negative risk factors indicated above. It is far from clear when this slowdown in the reduction of mortality will set in and how significant it will be. Our assumptions have been guided in part by the growing uncertainty of assessments as the time elapsed increases. Here we have assumed a reduced decline in mortality for women from 2010 onwards and for men from 2015 onwards. The difference between men and women is due in part to our assumption that longitudinal effects will cease to be felt sooner among women than among men, since the decline in mortality started earlier among women than among men. We also put a brake on the decline towards the end of the forecast period. The reason is that the overall picture of causes of death may change by then. We should bear in mind that most of the extrapolated reduction in mortality is connected with cardiovascular diseases. In 30–40 years time, this cause-of-death category may well be considerably reduced, even at relatively advanced ages. The other causes of death, which are declining more slowly, will thus acquire greater significance in relative terms and will then automatically entail a slower decline in total mortality.

[3] It may be noted that this assumption is very far-reaching in statistical terms, since in effect we are extrapolating 50 years forward in time from a 20-year trend (at least for men).

Assumptions Used in the Forecast for the Immediate Future

We have based our assumptions regarding mortality in the immediate future on observed risks of death during the period 1995–1999, extrapolated until 2000, (see Figure 4).

Figure 4 Risks of Death in 2000 by Age and Sex. Per million



We assume that risk of death will subsequently be reduced according to the pattern shown in the figures below. Among men, we assume that the risk of death will decline by 1.5 per cent per year at ages below 45, at a somewhat faster rate between 50 and 75, and at a gradually declining rate at more advanced ages. These reductions in the risk of death largely correspond to the trend observable in the 1990s among middle-aged and older men. We assume that this reduction in risk of death will continue unchanged until 2015.

For women, the risk of death has diminished over time in about the same way as for men. For the period until 2010, we have assumed an annual reduction in the risk of death of 1.5 per cent up to the age of 80, in accordance with the trends observed in the 1990s.

It should be noted that the change in the rate of reduction in mortality at different dates proceeds in stages (linear progressive reduction). The transition to a new rate of reduction occurs over a four-year period (for men in 2015–2018 and for women in 2010–2013).

Assumptions Used in the Forecast for the Longer Term

Among men, we assume that the annual rate of reduction during the period 2018–2039 will be 75 per cent of its original level. After this, the rate of reduction will gradually decline over a four-year period until it reaches 50 per cent of the original level (due to the change in the overall composition of causes of death, see Figure 5).

Among women, we assume that the risk of death will be reduced at a slightly slower pace beginning in 2010. We set the rate of reduction at 75 per cent of its original level over the period 2013–2034 and at half its original level in 2038–2050, (see Figure 6).

Behind these assumptions, there is substantial uncertainty regarding the speed at which the chances of survival are capable of changing over so long a period of extrapolation. However, the future may bring both a more rapid slowdown in the decline in mortality and new medical advances resulting in sharply lower risks of death.

Figure 5 **Annual Reduction of Risk of Death Among Men, by Age. Per cent**

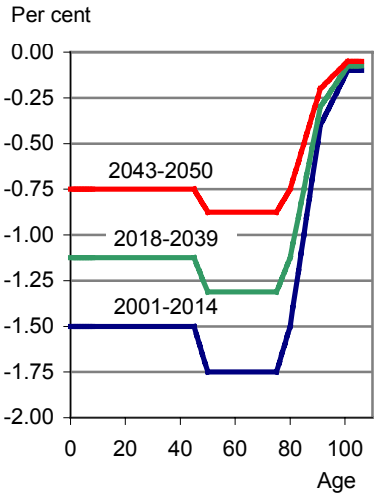
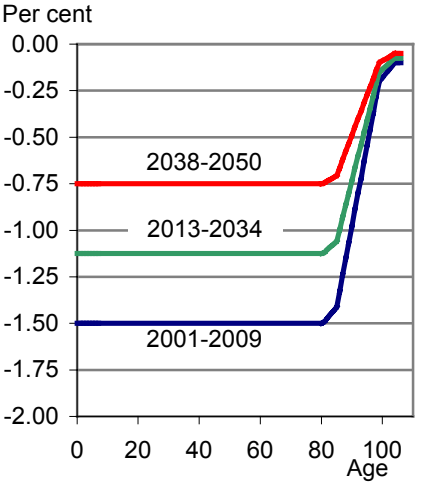


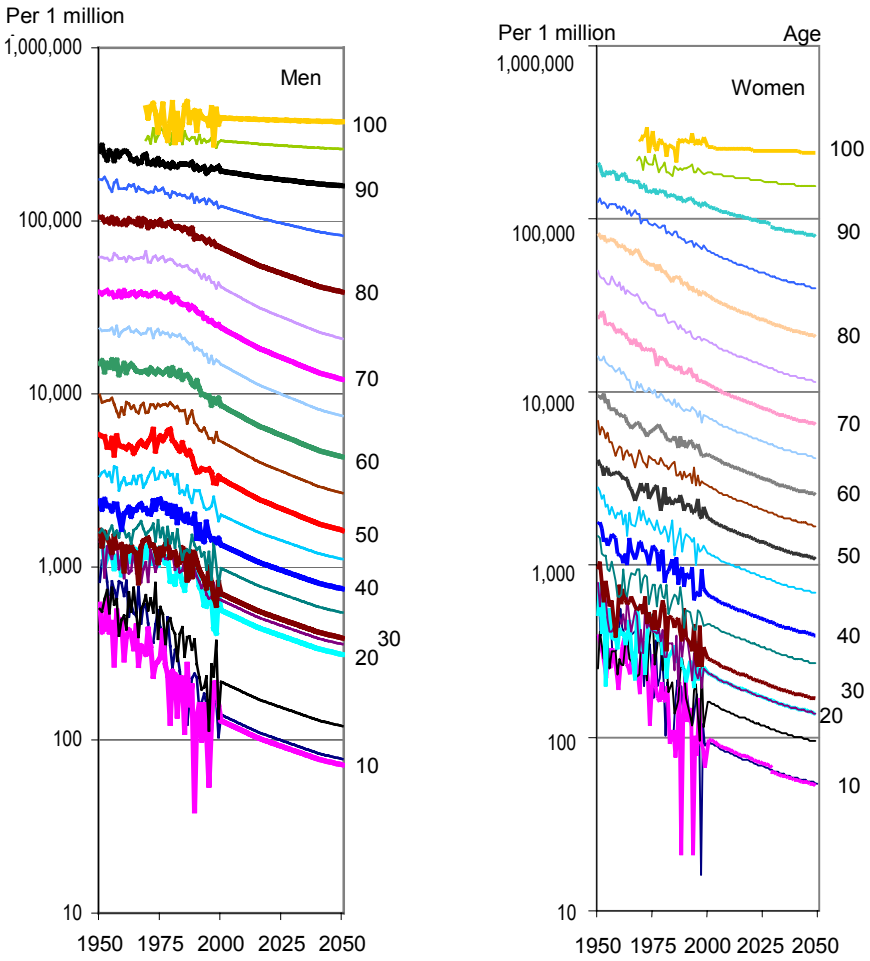
Figure 6 **Annual Reduction of Risk of Death Among Women, by Age. Per cent**



Mortality Trends over the Period 1950–2050

Figure 7 summarizes mortality trends between 1950 and 2050. A logarithmic scale has been used, thus making it possible to compare the mortality trends for different ages. The fact that the curves have the same slope shows that the percentage change in the risk of death has been the same.

Figure 7 **Mortality Trends by Age 1950–1999 and Assumed Mortality 2000–2050. Men and Women. Per 1 million**



Higher Average Life Expectancy

According to our estimates, average life expectancy for men will rise from 77.1 in 2000 to 82.6 in 2050, while the corresponding figures for women are 82.1 and 86.5. As shown in Table 2, we are forecasting a slower increase in average life expectancy over the coming 50-year period than we have observed over the past 50 years. We estimate that average life expectancy at 65 will rise by 3.8 years for men and 3.4 years for women over the next 50 years.

Table 2 Average Life Expectancy at Birth and at Age 65, 1951–2050

Year	*At birth*		*At age 65*	
	Men	*Women*	*Men*	*Women*
1951–60	70.9	74.1	13.9	15.0
1961–70	71.7	76.1	13.9	16.1
1971–80	72.3	78.1	14.1	17.5
1981–90	74.0	79.9	14.9	18.7
1991–95	75.6	81.0	15.7	19.4
2000	77.1	82.1	16.5	20.1
2010	78.7	83.4	17.6	21.1
2020	80.0	84.4	18.4	21.8
2030	81.0	85.2	19.1	22.5
2040	82.0	86.0	19.8	23.0
2050	82.6	86.5	20.3	23.5

Assumptions Regarding Mortality Trends in Some Countries

For the sake of comparison with assumptions regarding future mortality trends in other countries, we have provided average life expectancies according to population forecasts for a number of countries in Europe and for the USA and Japan, (see Tables 3 and 4).

There is wide variation between the different countries. In France and Belgium, it is assumed that average life expectancy for men will increase by nearly eight years over the next 50 years, while in Japan it is expected to rise by just two years. In Sweden, the predicted increase is 5.5 years.

For women, too, there is a considerable range in assumed future mortality. France, Belgium and the USA are predicting that average life expectancy will go up by seven years, whereas Japan and the Netherlands are anticipating a gain of barely more than two years. In Sweden, the rise is expected to be 4.4 years.

Table 3 **Average Life Expectancy for Men, 2000–2050. Forecasts in Different Countries**

Country	*Average life expectancy at birth, in years*					
	2000	*2010*	*2020*	*2030*	*2040*	*2050*
Sweden	77.1	78.7	80.0	81.0	82.0	82.6
France	74.6	76.4	78.0	79.5	80.9	82.2
Belgium	74.4	75.7	77.1	78.7	80.3	82.1
Austria	75.1	76.9	78.5	80.0	81.0	82.0
Switzerland	76.1	77.5	78.8	79.8	80.6	81.3
Finland	73.8	75.6	77.3	78.7	80.0	81.2
USA	74.2	75.5	76.8	78.3	79.8	81.2
Netherlands	75.3	76.6	77.8	78.8	79.5	80.0
Norway	75.8	77.3	78.5	79.4	79.8	80.0
Japan	77.4	78.1	78.6	79	79.2	79.4
UK	75.1	76.6	77.6	78.2	78.7	78.9
Italy	75.9	77.1	78.3	78.3	78.3	78.3
Denmark	73.4	73.5	73.5	73.5	73.5	73.5
Ireland	73.7	75.2	76.4			
Spain	74.1	75.3	76.0			
Germany	73.7	74.8	75.7	76.7	76.9	
Iceland	77.3	77.5	77.5	77.5		

Source: USA, US Bureau of Census; Japan, Ministry of Health and Welfare; Sweden, Forecast 2000–2050. Other countries: Eurostat, June 2000.

Table 4 **Average Life Expectancy for Women, 2000–2050. Forecasts in Different Countries**

Country	*Average life expectancy at birth, in years*					
	2000	*2010*	*2020*	*2030*	*2040*	*2050*
France	83.0	84.8	86.5	87.9	89.2	90.4
Belgium	81.1	82.3	83.6	85.0	86.5	88.1
Austria	81.3	82.8	84.2	85.5	86.2	87.0
Switzerland	83.0	84.4	85.6	86.0	86.4	86.9
USA	79.9	81.4	82.9	84.2	85.4	86.6
Sweden	82.1	83.4	84.4	85.3	86.0	86.5
Japan	84.1	85.1	85.6	86.0	86.3	86.5
Finland	80.9	82.0	83.1	84.0	84.8	85.5
Italy	82.3	83.5	84.7	84.7	84.7	84.7
Norway	81.5	82.7	83.5	84.1	84.4	84.5
UK	80.1	81.5	82.6	83.2	83.6	83.8
Netherlands	80.6	81.1	81.6	82.2	82.7	83.0
Denmark	78.5	78.6	78.6	78.6	78.6	78.6
Spain	81.9	83.0	83.7			
Iceland	81.9	82.1	82.1	82.1		
Ireland	79.4	81.0	82.4			
Germany	80.1	81.1	81.9	82.6	82.9	

Source: USA, US Bureau of Census; Japan, Ministry of Health and Welfare; Sweden, Forecast 2000–2050. Other countries: Eurostat, June 2000.

Alternative Assumptions

The purpose of alternative assumptions is to attempt to capture some of the uncertainty in the principal assumption that we have already presented.

Under an alternative assumption with lower mortality, the declining trend in mortality decline during the 1990s will continue uninterrupted throughout the forecast period until 2050. We assume continuous improvements in lifestyle throughout the period. Moreover, further improvement in medical care and treatment is required (over and above the improvement in the principal assumption), particularly with regard to diseases other than cardiovascular diseases.

In an alternative with higher mortality, we assume no changes in mortality at all in future. Positive and negative lifestyle factors offset each other. This alternative provides a base level for the impact on the population of assumptions regarding mortality; *i.e.*, it functions as a form of sensitivity analysis.

In the first alternative, life expectancy rises from 77.1 in 2000 to 86.1 in 2050 for men and from 82.1 to 89.0 for women. In the second alternative, the figures remain at their initial level throughout the period.

Forecasting Life Expectancy: The SCOPE Approach

James W. Vaupel

Professor and Founding Director, Max Planck Institute for Demographic Research, Rostock, Germany

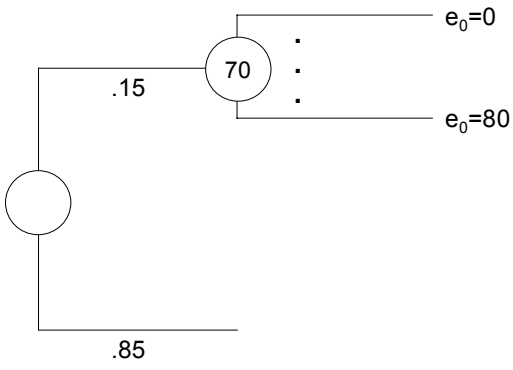
This note outlines a method for forecasting life expectancy. The method is based on the idea of structured conditional probabilistic estimation; it "scopes" out the range of possibilities the future may hold. I first described this SCOPE method at a workshop organized by Juha Alho several years ago in Finland.[1] It is a kind of scenario method – with probabilities attached to scenarios, with scenarios structured conditionally, and with the possibility of stochastic scenarios. It is a simple method, and it is by no means original; many other people have used a similar approach in various settings. This method might be helpful to those who want to forecast life expectancy. This note summarizes my presentation.

To be concrete, suppose the goal is to forecast female life expectancy at birth in Sweden in 2050. In the year 2000, the expected life span for Swedish women was just over 82 years. What will it be in the middle of the 21st century?

A central point of uncertainty is whether the kind of progress and development in Sweden that has marked the past couple of centuries will continue until 2050. Perhaps the future will be characterized by poverty, misery, and a shorter life expectancy. Coming decades could bring nuclear war, massive biochemical terrorism, epidemics more deadly than the AIDS epidemic, catastrophic environmental change, lasting economic depression, or some other disaster or combination of disasters that might cause female life expectancy in Sweden to plummet far below its current level of more than 82 years, perhaps even down to zero. These possibilities are indicated in the figure below. My "guesstimate," given my current knowledge and the limited amount of time I have spent researching and thinking about this question, is that there is

[1] In addition, I very briefly presented one simple version of it in the last paragraph on page 195 of Lutz, Vaupel and Alberg (eds.) (1998).

a 15 per cent chance that life expectancy will decline in the future. If it does, then my best guess is that the mean value – of the range of possible life expectancies in 2050 – is 70 years, which is close to the current value of female life expectancy in the world as a whole. Discussions among a group of experts and systematic consideration of various scenarios would undoubtedly produce values different from 15 per cent and 70 years, but these values illustrate the approach.



Suppose calamity is averted, with probability 0.85. Then the next major uncertainty would seem to be whether life expectancy is approaching a looming limit. This limit does not have to be an ultimate cap that will hold forever. It simply has to be some ceiling that Swedes will not be able to exceed by 2050. Perhaps there really is some biological limit to life expectancy of 85 or so. Current evidence suggests that this is unlikely, but it might be true. More plausibly, perhaps it will be impossible to make much progress in reducing death rates at very old ages. To achieve such reductions, new kinds of biomedical breakthroughs will be required, and these breakthroughs may not be forthcoming, at least over the next half century. Furthermore, there may be practical impediments to further reductions in mortality. For instance, taxpayers may not be willing (or able) to pay for the required interventions if there are too few workers to support increasing numbers of retired people. For illustrative purposes, suppose the probability of some such scenario is 20 per cent and that, conditional on this, life expectancy in 2050 will be 85 for Swedish females. Of course, it might not be precisely 85, but suppose that 85 is the average value of the fairly narrow range of possibilities permitted by this line of thinking. Again, debate and systematic calculation would lead to values other than 20 per cent and 85 years, but these values provide a suggestive example.

The final possibility, in my simple probability tree, is that the future will be roughly the same or perhaps even better than the past. The uncertainty here might be structured as follows:

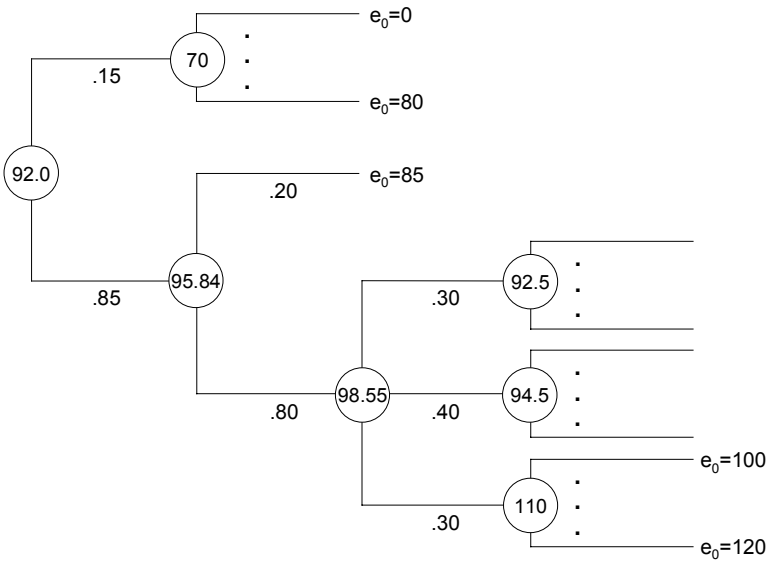
- One possibility is that the pace of age-specific mortality improvement over the next half century will be similar to the pace of improvement over the last fifty or hundred years. This is the general idea underlying the Lee-Carter-Tuljapurkar time-series method of forecasting life expectancy and Juha Alho has also done research along this line.[2] I am not sure what the latest results are if this approach is applied to Swedish females, but to be specific, suppose the mean value of predicted life expectancies in 2050 is 92.5.
- A second possibility is that the pace of life-expectancy increase over the next half century will be similar to the rate of increase over past decades. Jim Oeppen and I studied the rise in the expectation of life over the last two hundred years. We discovered that "best-practice" life expectancy, *i.e.*, female life expectancy in the country that held the record, had risen linearly by three months per year from 1840 to 2000. Over this entire period, Sweden has been either at or near the top. Currently, the life expectancy of Japanese women is almost 85 years, whereas that of Swedish females is a bit more than 82 years. Thus, the current gap is between two and a half and three years. If the trend of the past 160 years continues for the next half century, then life expectancy in 2050 will reach a record 97.5. If time-series methods are applied to the data, then there will be some small band of uncertainty around this value; the band will be narrow because the trend over the past 160 years has been remarkably linear. I have not applied time-series methods to the data, however, and I am not sure exactly how wide the range of uncertainty will be. In any case, a greater source of uncertainty will involve the gap between Swedish and record life expectancy in 2050. In the figure below, I assume that the mean value of this gap is three years, yielding a life-expectancy estimate of 94.5.
- Finally, the third possibility is that mortality improvements will accelerate in the future. Biology and biomedicine may be on the verge of unprecedented breakthroughs in knowledge about specific diseases and about the aging process itself – many knowledgeable scientists are of this opinion. Specifically, instead of increasing by 2.5 years per decade, life expectancy may increase by 3, then 4, and then 5 years per decade over the next three decades and perhaps by 6, 8, or even 10 years per decade in the 2030s and

[2] See Lee and Carter (1992: 659); Tuljapurkar, Li and Boe (2000); Alho (1998).

2040s. Since the sum of 3+4+5+7+9 is 28, female life expectancy in Sweden in 2050 may be 110 years rather than the current 82 years or so. In the figure below, I suggest a range from 100 to 120 years if mortality improvements accelerate, with a mean value of 110. This is really just a guess, and some informed discussion and structured debate might yield quite a different picture. For illustrative purposes, however, a value of 110 may not be inappropriate.

What is the chance that the future will be like the past in terms of age-specific mortality change, that the future will be like the past in terms of life-expectancy change, or that the future will bring an accelerated rate of increase in life expectancy? The trend in best-practice life expectancy is so regular that I assigned a probability of 40 per cent to a continuation of this trend, and I gave each of the other two possibilities a 30 per cent chance. Folding the tree back, these values lead to a mean life expectancy of 98.5 if the future is like the past or even better. If there is no disaster, then the mean is 95.84. All factors considered, the mean is 92.0. (The calculations just happened to produce a value close to this nice round number).

The future is enveloped in uncertainty, and there is a wide probability distribution around this value of 92.0, stretching from 0 to 120 in the tree below. This predictive distribution could be estimated. Some components of uncertainty could be assessed by expert judgement. Other components, as noted above, could be estimated by time-series methods. A structured conditional probability tree, of the kind shown below, could be used to organize the forecasting problem. And that is the concept underlying the SCOPE approach.


e_0=0
70
.15
e_0=80
92.0
e_0=85
.20
95.84
.85
92.5
.30
98.55
.80
94.5
.40
e_0=100
110
.30
e_0=120

References:

Alho J. (1998). "A Stochastic Forecast of the Population of Finland", *Reviews* 1998/4. Helsinki: Statistics Finland.

Lee R.D. and L. Carter (1992). "Modeling and Forecasting U.S. Mortality", *Journal of American Statistical Association* vol. 87: 419, 659-671.

Lutz W., J.W. Vaupel and D.A. Ahlburg (eds.) (1998). "Ways to Improve Population Forecasting: What Should Be Done Differently in the Future?" in Frontiers of Population Forecasting, *Population and Development Review*, supplement to vol 24, 191-198.

Tuljapurkar S., N. Li and C. Boe (2000) "A Universal Pattern of the Mortality Decline in the G7 Countries", *Nature,* vol. 405, 789-792.

Mortality Forecasts. Comments on How to Improve Existing Models – an Epidemiologist's Perspective

Kaare Christensen

Professor of Epidemiology at the University of Southern Denmark, Denmark

From an epidemiologist's perspective, one way to improve mortality forecasts is to gain insight into the causes and predictors of mortality. If we know the "risk profile" of the current cohorts compared to the previous cohorts, then our forecasts may improve.

If we also include genetics in our perspective, the initial task will be to quantify the contribution of genes and environment to lifespan. Therefore, the first question will be, "Are the lifespans of relatives correlated?" and if so, "Is the correlation due to a shared environment or to shared genes?" It is important to pose these questions initially because the answers determine whether it is worthwhile to seek the causes and predictors of mortality in the environment as well as in the genetic make-up.

Are the Lifespans of Relatives Correlated?

Traditional family studies suggest a correlation in lifespan within families. However, studies have generally found only small correlations in lifespan between parents and offspring (0.01–0.15) (Pearl 1931; Cohen 1964; Wyshak 1978), whereas correlations between siblings tend to be higher (0.15–0.35) (Cohen 1964; Wyshak 1978). Heritability estimates based on regression analysis were in the range of 0.10–0.33 for parents-offspring and 0.33–0.41 for siblings, constantly over a period of 300 years (Meyer 1991), but these estimates include both genetic factors and shared environmental factors. Some family studies have found a stronger maternal than paternal effect (Abbott et al. 1974), but not all (Wyshak 1978). The lower correlation found for parents and offspring than for siblings, suggests that genetic non-additivity (genetic effects due to gene interaction which are not passed from one generation to the next) is present. However, it may also reflect a higher degree of shared environment among siblings than among parents and offspring; the latter constitute two generations living under very different conditions.

The Relative Effects of Genetic and Environmental Factors on Lifespan

Twin studies are designed to separate the effects of additive and non-additive genetic factors, as well as shared and non-shared environmental factors. However, most of the early twin studies had methodological problems due to left-truncation of the cohorts included, selection bias, lack of zygosity diagnosis, or heavy right-censoring. Carmelli and Andersen (1981) included a sample of 2,242 Mormon pairs of twins born 1800–1899 in which both twins had died, a criterion met by 60 per cent of the original sample. Wyshak (1978) followed 972 Mormon twin-pairs (possibly included in the study of Carmelli and Andersen) until death. Unfortunately, since both studies lacked zygosity diagnosis, heritability estimates could not be provided. However, similarity in length of life was found. The similarity was more pronounced for twins of the same sex (including both MZ and DZ twins) than for twins of the opposite sex (always DZ twins), suggesting genetic influences on lifespan. Jarvik et al. (1960) followed a sample of 853 pairs of twins for 12 years; the sample included only pairs with at least one twin surviving to age 60. At the end of the follow-up period, both twins had died in only 35 per cent of the pairs. The mean intra-pair difference in lifespan was found to be higher for DZ than in MZ twins, suggesting genetic influences on lifespan. Hrubec and Neel (1981) followed a sample of 31,848 male twin veterans born 1917–27 for 30 years to ages 51–61. Around 10 per cent were deceased at the time of analysis. To avoid censoring problems, longevity was analyzed as a categorical variable (dead/alive). In this study, the heritability of "liability" to die was estimated to be 0.5.

The first non-censored and population-based twin study that could provide an estimate of the magnitude of genetic influences on lifespan was conducted by McGue et al. (1993). It covered 600 Danish pairs of twins born 1870–1880. Using path analysis, a heritability of 0.22 was found, with genetic influences being mainly non-additive. Later this study was expanded by Herskind et al. (1996) to include more than 2800 twin-pairs with known zygosity born 1870–1900. These cohorts were followed from age 15 to death. The study confirmed that approximately a quarter of the variation in lifespan in this population could be attributed to non-additive genetic factors, while the remaining three-quarters were due to non-shared environmental factors.

Ljungquist et al. (1998) studied the 1886–1900 Swedish cohorts of twins and concluded that around a third at most of the variance in longevity is attributable to genetic factors.

Hence, it seems to be a rather consistent finding in the Nordic countries that approximately 25 per cent of the variation in lifespan is caused by genetic differences. It is interesting that animal studies have revealed similar estimates for a number of species not living in the wild (Curtsinger et al. 1995; Finch and Tanzi 1997).

Hence, the conclusion from these studies is that it is worthwhile to seek the causes and predictors of mortality in the environment as well as in the genetic make-up. However, the results from family studies with low correlations between family members suggest an absence of common genes with a substantial impact on lifespan.

Prediction of mortality

From a forecast perspective, which focuses on reduction of mortality (rather than on sudden increases in mortality due to new diseases, war, etc.), there is little interest in estimating survival at younger ages, when the room for improvement is very limited. What is important for future mortality trajectories is mortality among elderly people (Vaupel et al. 1998).

A number of risk factors seem to lose their importance with age, probably because of heterogeneity and selection, *e.g.*, smoking, obesity, diseases and SES (socio-economic status). However, this does not mean that the survival rate of the elderly, including the very oldest, cannot be raised. In the latter category, the fraction dying every year is high (for example, 1/4 – 1/3 among nonagenarians), and there is both practical and theoretical evidence that intervention can have substantial positive effects on both quality of life and survival.

In relation to forecasting, it is important to note that certain predictors remain valid even at the highest ages, *e.g.*, self-rated health as well as physical and cognitive abilities (Nybo *et al*. 2001). This may provide an opportunity to improve forecasting if for example the physical abilities of new cohorts of elderly persons are assessed and compared to previous cohorts; *i.e*. "Are the new cohorts of the elderly healthier than the previous cohorts (and therefore expected to live longer)?" The first reports of this kind have been published based on US studies (Manton and Gu 2001; Manton *et al*. 1997). They indicate that the new cohorts of elderly are increasingly healthy.

Conclusion

The cohort differences in physical abilities among the elderly and the correlation between physical abilities and mortality may be the basis for improving the forecasting of mortality.

References

Abbott M. H., H. Abbey, D.R. Bolling and E.A. Murphy (1974). "The Familial Component in Longevity. A Study of Offspring of Nonagenarians. II. Preliminary Analysis of the Completed Study". *Johns Hopkins Medical Journal*, vol. 134, no. 1, 1-16.

Carmelli D. and S. Andersen (1981). "A Longevity Study of Twins in the Mormon Genealogy". *Progress in Clinical and Biolological Research*, vol. 69 Pt C, 187-200.

Cohen B. H. (1964). "Family Pattern of Mortality and Life-Span". *The Quarterly Review of Biolology*, vol. 39, 130-181.

Curtsinger J. W., H. H. Fukui, A. A. Khazaeli, A. Kirscher, S.D. Pletcher, D. E. Promislow, and M. Tatar (1995). "Genetic Variation and Aging". *Annual Review of Genetics*, vol. 29, 553-575.

Finch C. E. and R. E. Tanzi (1997). "Genetics of Aging". *Science*, vol. 278, no. 5337, 407-411.

Herskind A.M., B. Harvald, N. V. Holm, M. McGue, T. I. Sorensen and J. W. Vaupel (1996). "The Heritability of Human Longevity: a Population-Based Study of 2872 Danish Twin Pairs Born 1870-1900". *Human Genetics*, vol. 97, no. 3, 319-323.

Hrubec Z. and J. V. Neel (1981). "Familial Factors in Early Deaths: Twins Followed 30 Years to Ages 51-61 in 1978". *Human Genetics*, vol. 59,no. 1, 39-46.

Jarvik L., A. Falek, F. J. Kallmann and I. Lorge (1960). "Survival Trends in a Senescent Twin Population". *American Journal of Human Genetics*, vol. 12, 170-79

Ljungquist B., S. Berg, J. Lanke, G. E. McClearn, and N. L. Pedersen (1998). "The Effect of Genetic Factors for Longevity: a Comparison of Identical and Fraternal Twins in the Swedish Twin Registry". *The Journals of*

Gerontology. A Biological Science and Medical Science, vol. 53, no. 6, M441-M446.

Manton K.G., L. Corder and E. Stallard (1997). "Chronic Disability Trends in Elderly United States Populations: 1982-1994". *Proceedings of the National Academy of Sciences of the USA*, vol. 94, no. 6, 2593-2598.

Manton K.G. and X. Gu (2001). "Changes in the Prevalence of Chronic Disability in the United States Black and Nonblack Population above age 65 from 1982 to 1999". *Proceedings of the National Academy of Sciences of the USA*, vol. 98, no. 11, 6354-6359.

McGue M., B. Harvald, N. Holm and J. W. Vaupel (1993). "Longevity is Moderately Heritable in a Sample of Danish Twins Born 1870-1880". *Journal of Gerontology*, vol. 48,no. 6, B237-B244.

Meyer P. J. (1991). "Inheritance of Longevity Evinces no Secular Trend Among Members of Six New England Families Born 1650-1874". *American Journal of Human Biology: The Official Journal of the Human Biology Council*, vol. 3, pp. 49-58.

Nybo H., K. Andersen, K. Christensen, D. Gaist, B. Jeune, M. McGue, H. C. Petersen and J. W. Vaupel (2001). "Predictors of Mortality Among 2,249 Nonagenarians - The Danish 1905-Cohort Survey". Manuscript.

Pearl R. (1931). "Studies on human longevity IV. The Inheritance of Longevity". *Human Biology*, vol. 3, 245-269.

Vaupel J. W., J. R. Carey, K. Christensen, J. W. Curtsinger, N. V. Holm, I. A. Iachine, T. E. Johnson, V. Kannisto, A. A. Khazaeli, P. Liedo, V. D. Longo, K. G. Manton, A. I. Yashin and Y. Zeng (1998). "Biodemographic Trajectories of Longevity". *Science*, vol. 280, no. 5365, 855-860.

Wyshak G. (1978). "Fertility and Longevity in Twins, Sibs, and Parents of Twins". *Social Biology*, vol. 25, no. 4, 315-330.

The Need for Looking Far Back in Time When Predicting Future Mortality Trends

Tommy Bengtsson

Professor of Demography and Economic History at Lund University, Sweden

There are three reasons why we must look far back in time when predicting future mortality trends. Firstly, the mortality decline that we can observe today has its roots in improvements achieved long ago in living standards and diet, public health institutions, medicine, and other areas relevant to the physical well-being of the population. Speaking in general terms, living conditions improve from one period to the next. Such improvements are called *period* factors, since they relate to living conditions for the entire population during one specific period.

Secondly, the health and remaining life span for people living today is determined not only by contemporary period factors but also by living conditions earlier in life. Since even conditions in the foetal stage have an influence on longevity, improvements during early childhood could have an effect on mortality trends today. These are called *cohort* factors, since they relate to the conditions for a certain cohort, often birth cohort, while previous cohorts are unaffected. Since the oldest people living today were born a hundred or more years ago, we have to consider cohort factors far back in time in predicting future mortality.

Thirdly, predicting future mortality trends also requires a multivariate point of departure. No single factor, but a variety of factors, determines health and remaining life span, and we do not know a priori which one is the most important. Thus, predicting future mortality trends calls for a long-term multivariate, causal approach, in which both period and cohort factors are taken into account. This kind of holistic view has not always been in favour. Early in the twentieth century, cohort factors were considered most important, while multivariate period factors became more popular later on, followed in turn by a preference for single-period factors as the main determinant of the great mortality decline since the mid-eighteenth century. Now we are once again discussing cohort factors.

Thus, there has been a shift over time in our explanations for the mortality decline (Bengtsson 1998). Below I shall develop the reasons why I think we need to take a multivariate, period, and cohort approach. I will start with an overview of the great mortality decline, commencing several hundred years ago, and then move on to discuss how the explanations for the historical decline have changed over time.

Over the past 300 years, human physiology has undergone profound changes. These were made possible by numerous advances whereby humans have gained an unprecedented degree of control over their environment. The changes include increases in body size by over 50 per cent, in length of life by over 100 per cent, and in time lived in retirement by several hundred per cent, with obvious implications for expenditures on pensions and health care. The extension of the human life span has been gradual, starting a long time ago with what now is called the great mortality decline.

The timing of the great mortality decline was strikingly similar in the countries of Western and Northern Europe despite differences in economic structure and development. It started in the mid-eighteenth century, levelled off for a couple of decades in the mid-nineteenth century, and then continued. Over this period, life expectancy at birth rose from some 35 years to more than 70 years. The development in North America was similar.

Few scholars today will argue that the great mortality decline was due solely or primarily to a single factor. Economic growth, for example, was probably not a major determinant before or during the initial stages of the great mortality decline, and its effect may be far less than expected during later stages of the decline. Instead, the causes are multi-factorial and vary from the start to the end of the decline.

In countries for which long historical series of age-specific mortality rates (England, France, and some others, including the Nordic countries) are available, we know that the increase in life expectancy began with a fall in the rates of infant and child mortality. This was mainly due to a reduction in deaths from smallpox, which was a very common childhood disease in the eighteenth century.

While infant mortality continued to decline in Sweden throughout the nineteenth century, it levelled off in England and remained stable until the end of the nineteenth century, when it again dropped rapidly, as was the case in all other Western countries. Childhood mortality in both countries actually increased in the mid-nineteenth century before commencing a persistent decline. This exemplifies two patterns of the historical decline in infant and

childhood mortality: a northern pattern with declines essentially throughout the nineteenth century, and a western one with a levelling off during the nineteenth century and a further decline after the 1880s (Perrenoud 1984).

Adult and old-age mortality started to decline gradually at the beginning of the 19th century, possibly earlier for England. The decrease was generally sharper in the later years of the 19th century and even more so after the First World War, as was the case with mortality at all other ages. The decline slowed for adults and the elderly around 1950 but accelerated again in the 1970s.

With the great mortality decline followed a change in the leading causes of death, from pestilence, to receding pandemics, and then to man-made diseases (Omran 1971). In his theory of epidemiological transition, Omran identified three different development patterns: the classical or western pattern, described above, the accelerated pattern, and the delayed pattern. They are distinguished by differences in timing and speed. The decline that took 200 years in the West started 150 to 200 years later in the Third World but then took less than fifty years to complete. In many countries, the great mortality decline has not yet occurred, and the gains from low mortality are still to be reaped.

The great mortality decline can be viewed in light of two approaches, one based on *period factors*, the other on *cohort factors*. Most studies focus largely on period factors. In addition, factors based almost entirely on human actions should be distinguished from those beyond deliberate human control. One widely accepted multi-factorial explanation (United Nations 1953) is based on *period factors* that depend upon human activity: these include public health reforms, advances in medical knowledge, improved personal hygiene, and rising income and standards of living. This explanation is highly similar to the demographic-transition theory (Davis 1945; Notestein 1953; Bengtsson and Ohlsson 1994).

On a broader front, McKeown (1976) questioned the multi-factorial explanation of the great mortality decline. He argued that a single factor – better nutrition – could almost entirely explain the great mortality decline. His criticism was based on a study of cause-specific mortality in England and Wales from 1838 to 1947, where he observed that two-thirds of the mortality decline was due to a reduction in infectious diseases. In later work, he also analysed mortality rates and economic development for other countries and further back in time, though not in such detail as for England and Wales.

McKeown argued that medical advances had little influence on mortality trends before the breakthrough of sulphonamides and antibiotics in the 1930s and 1940s. Previously, the only curable disease had been diphtheria. Very few deaths, however, were due to diphtheria, and its incidence was already receding when the antitoxin came into use around 1900. For periods before 1838, McKeown held that inoculation and vaccination for smallpox had little or no impact on the general mortality decline. Vaccination started in England at the end of the eighteenth century, but it did not become widespread until after 1840, when it was made available at public expense. The history of vaccination in the Nordic countries is similar. There, smallpox mortality was declining even before vaccination started in the first years of the nineteenth century and well before it became common in the 1820s. However, not all scholars agree on this interpretation. Easterlin (1999) downplays the role of economic development and better diet, contending that advancement in medical knowledge is the principal reason for the mortality decline from the mid-nineteenth century onwards.

The improvement of personal hygiene may have had some effect on mortality in England and Wales after about 1880, when the incidence of intestinal infections declined, probably because of substantial improvements in water supply and sewage control at that time. Prior to 1870, the decrease in intestinal infections accounted for only a small part of the general decline in mortality, and according to McKeown, neither better personal hygiene nor measures to improve public health had any significant impact on the overall decline before 1870. Other public-health measures, such as the breast-feeding campaigns in Sweden in the 1830s and thereafter, were not discussed by McKeown. Fridlizius (1984) has argued that while these campaigns might well have had an impact on infant mortality in Sweden, they began much later than the decline in mortality. In fact, childhood mortality went up somewhat at the time of the first breast-feeding campaigns.

In 1957, Helleiner argued that the population of Western Europe must have increased from the mid-eleventh century to the late thirteenth century and from the mid-fifteenth century to the end of the sixteenth century (Helleiner 1957). The population increase observed in the eighteenth century was therefore unique only in that the mortality decline started from a higher level and went on longer than before. Helleiner and other scholars have contended that these changes in mortality were spontaneous (natural), and in 1973 the UN added natural factors as a fifth determinant of the great mortality decline (United Nations 1973). Later, others like Fridlizius (1984), Perrenoud (1984) and Schofield (1984) asserted that a change in the virulence of pathogens initiated the great mortality decline. Their basic assumption was that the virulence of pathogens changes spontaneously over time. Virulence in a

pathogenic organism is generally understood as its ability to overcome host defences. At this point, it is important to note that some pathogens develop more quickly in a malnourished host, whereas others do not depend on a weakening of the host to produce very high mortality.

McKeown's main argument on this subject, though not proved, is that the initial development of mortality in the late eighteenth century was an integral part of the great mortality decline. Since the decline continued for the next two centuries, it cannot be due to a spontaneous reduction in the virulence of pathogens; thus, according to McKeown, the only explanation left is better nutrition. Improved nutrition would explain not only the decline in infectious diseases from the mid-nineteenth century onwards, but also the initial decline. This reasoning is part of McKeown's attempt to find one single explanation for the entire mortality decline.

Fogel (1994) criticized McKeown's reasoning for considering only nutritional intake, or diet, while ignoring the needs of the body to maintain itself and build up cells. Thus, McKeown only took into account gross nutrition, rather than net nutrition; the latter must be more closely related to health and mortality. I will come back to this issue shortly.

Cohort explanations for the mortality decline refer to factors that initially affect only certain young age groups but may have a long-lasting impact on these groups. Such factors would consist mainly of improvements in childhood conditions, or even conditions during the foetal stage, that have lasting effects on health and on the life span. Net nutrition is seen as the principal determinant of cellular development, which is most rapid during the foetal stage and gradually diminishes until the body is fully developed around the age of 20. Net nutrition is what is left for the development of cells after the nutritional requirements of other life-sustaining functions and work have been met. Thus, low net nutrition could be due either to low nutritional intake or to additional, disease-related needs of the body for nutrition. Moreover, many diseases not only claim nutrients but also make it more difficult for the body to absorb nutrients in general, as is the case with infectious diseases. If cells and organs consequently fail to develop properly, a child's growth and development may be inhibited, and the child may be less healthy in general. Thus, we can differentiate between two basic types of cohort explanations for the mortality decline, namely (i) increased nutritional intake during the foetal stage and/or early years of life, and (ii) decreased needs for nutrition during the foetal stage or early years of life owing to less disease in the mother or the child.

The importance of early childhood conditions for later life has probably been well known since time immemorial. It is often assumed that each generation shows the same relative mortality from childhood to old age. Kermack, McKendrick and McKinley adopted this assumption in 1934 when scholars were starting to become aware of the great mortality decline (Kermack, McKendrick and McKinley 1934). Strangely enough, cohort factors were out of fashion when the UN in 1953 and 1973 made its synthesis of the causes of the great mortality decline. Over the last couple of decades, both medical and historical research on this matter has expanded rapidly (for an overview, see Elo and Preston 1992).

In this connection, the work by Barker (1994 and elsewhere) has been of major importance. He has summarised the medical evidence showing the importance of foetal and neonatal nutrition for adult health. In historical research, Fogel (1996) is probably the leading advocate of these ideas. In addition, Preston and van de Walle (1978) for urban France, and Fridlizius (1989) for Sweden, emphasised the importance of cohort factors for the mortality decline. Bengtsson and Lindström found that exposure to disease from airborne infections has a strong effect on mortality among the elderly (Bengtsson and Lindström 2000, 2001).

Steckel (1983) and Fogel (1996 and previous work) use final body height as a measure of net nutrition and health during childhood. Individuals who have had well-nourished and healthy mothers, and thus have been well nourished themselves during the foetal stage, have a lower risk of death during infancy. If they are well nourished and healthy, their cells and organs develop better, they grow taller, and they live longer. Since health is determined by net nutrition and not gross nutrition, there is no direct link between gross nutrition and height attained. Improvements in health and height may be due either to better nutrition (better diet) or to more limited claims on nutrition from disease. Thus, a decline in the prevalence of smallpox, for example, has a positive effect on height and extends the life span, everything else being equal. The problem is to evaluate how much of the improvement in health is due to diet and how much is due to less disease. Calculating diets for pre-modern populations is a difficult task (Fogel 1996), and it is even harder to calculate disease-related claims on nutrition. Still, historical records show similarities between trends in height and gross consumption of nutrition (Fogel 1994, 1996), indicating that trend in disease-related claims has been of minor importance. If that is the case, then McKeown's focus on gross nutrition may be justified.

Whether due to a low or badly composed nutritional intake, or to greater claims on nutrition from diseases, undernourishment may stunt growth in

height or weight and lead to illness, disease and mortality later in life more than in the immediate future. The immediate relationship, or period link, between the economy and mortality, therefore, is much weaker than Malthus believed, according to Fogel. The rather tenuous short-term relationship often found between prices and deaths for many European countries, as shown by Lee (1981, 1993), Galloway (1988) and others, supports this interpretation (Fogel 1994). Thus, cohort factors matter more to the mortality decline than period factors, according to Fogel.

To summarise, few scholars today will argue that any single factor is the primary determinant of the great mortality decline. Of course, it is no coincidence that the vast growth in resources resulting from the transformation of our economies in the eighteenth and nineteenth centuries was concurrent with the great mortality decline and with the fertility transition as well. In a millennial perspective, these events took place at about the same time. This is not to say, however, that there is a close relationship between the economy and the great mortality decline. On the contrary, economic growth has probably not been a major determinant, either before the great mortality decline or in its initial stages, and its impact may have been far less than expected during later stages of the decline. Instead, the causes are multi-factorial and vary from the start to the end of the decline.

Initially, the decline may well have been partly due to pure luck, for example spontaneously less aggressive smallpox pathogens as part of an old demographic pattern rather than the result of a modernisation process. Later development and compulsory use of vaccine surely prevented the re-emergence of the more aggressive virus. Improvements in nourishment and in the care of mothers and children had long-lasting effects on life span. Advancements in water supply and sanitation as well as better housing contributed to the decline from the second part of the nineteenth century onwards. Medical progress in the twentieth century prolonged life. The fact that health is determined by net nutrition – intake of nutrition minus claims on nutrition – and that claims are partly disease-related, makes it difficult to evaluate the determinants of the mortality decline. The influence of conditions in childhood – including temporary ones – on mortality at older ages adds further complexity. The impact of each individual factor is therefore very difficult to measure; to do so would require a long series of high-quality data. Several variables, such as the virulence of pathogens and the claims on nutrients due to disease can at best be estimated indirectly, if at all. The analyses based on highly aggregated longitudinal data have identified the problems and directed us toward the solutions. It is more doubtful, however, whether solutions will be found at that level of analysis. Perhaps the use of longitudinal micro data will serve as valuable complement, as it has in other areas.

In my opinion, the development of theories about the great mortality decline can be summarised in the following figure, which also could give us some guidance for the future:

Figure 1 Development of Models for Explaining the Great Mortality Decline and Population Forecasts

	Period Factors	**Cohort Factors**
Univariate Models	1970s McKeown *1940s forecasts*	1930s Kermack et al *1990s forecasts*
Multivariate Models	1950s UN	1990s Barker and Fogel *2000s mortality forecasts?*

Thus, the cause of the great mortality decline is clearly multi-factorial, and the importance of the various factors changes over time. Both period and cohort factors must be taken into account in analysing the decline. A long-term perspective is essential in predicting future mortality trends. Combining longitudinal information for individuals with information at the societal level is likely to provide important information about mortality determinants in the past and may be useful for predicting future trends as well. Previously, both population and mortality projections were based largely on periodic information about demographic trends. The use of demographic cohort information in the 1990s was a considerable advancement. The question now is whether we are ready to take a great step forward by using multivariate causal models that combine information at the individual and societal levels.

References

Barker D. J. P. (1994). *Mothers, Babies and Disease in Later Life.* London: BMJ.

Bengtsson T. (1998*).* "The Great Mortality Decline: Its causes and consequences" in Núñez, C.-E. (ed.) *Debates and Controversies in Economic History.* Madrid: Fundación Ramón Areces.

Bengtsson T. and M. Lindström (2000). "Childhood Misery and Disease in Later Life: The effects on mortality in old-age of hazards experienced in early life, southern Sweden, 1760-1894". *Population Studies*, vol 54, no 2, 263-277.

Bengtsson T. and M. Lindström (2001). "Airborne Infectious Diseases in Infancy and Mortality in Later Life, Southern Sweden 1766-1894". Paper presented at the Social Science History Association conference in Chicago, November 2001.

Bengtsson T. and R. Ohlsson (1994). "The Demographic Transition Revised" in Bengtsson, T. (ed.) *Population, Economy, and Welfare in Sweden.* Berlin: Springer Verlag.

Davis K. (1945). "The World Demographic Transition". *Annals of the American Academy of Political and Social Sciences,* 237, 1-11.

Easterlin R. A. (1999). "How Beneficent is the Market? A look at the modern history of mortality". *European Review of Economic History*, vol 3, part 3, 257-294.

Elo I. and S. Preston (1992). "Effects of Early-life Conditions on Adult Mortality: a review". *Population Index*, vol 59, no 2, 186-212.

Fogel R. W. (1994). "The Relevance of Malthus for the Study of Mortality Today: Long-run influences on health, mortality, labour force participation, and population growth" in Lindahl-Kiessling, K. and H. Landberg (eds.) *Population, Economic Development, and the Environment*. Oxford: Oxford University Press.

Fogel R. W. (1996). *The Escape from Hunger and Premature Death 1700-2100: Europe, America and the Third World.* The 1996 Ellen McArthur Lectures, Cambridge.

Fridlizus G. (1984). "The Mortality Decline in the First Phase of the Demographic Transition: Swedish experiences" in Bengtsson, T., G. Fridlizius and R Ohlsson, (eds.) *Pre-Industrial Population Change: The mortality decline and short-term population movements*. Lund: Amqvist & Wiksell International, pp 71-114.

Fridlizius G. (1989). "The Deformation of Cohorts: Nineteenth century mortality decline in a generational perspective". *Scandinavian Economic History Review*, vol 37, no 3.

Galloway P. R. (1988). "Basic Patterns in Annual Variations in Fertility, Nuptiality, Mortality and Prices in Pre-Industrial Europe". *Population Studies*, vol 42, 275-302.

Helleiner K. F. (1957). "The Vital revolution Reconsidered". *Canadian Journal of Economics and Political Science*, vol 23, 1-9.

Kermack W. O., A. G. McKendrick and P. L. McKinley (1934). "Death Rates in Great Britain and Sweden: Some regularities and their significance". *The Lancet*, March.

Lee R. D. (1981). "Short-Term Variation: Vital rates, prices and weather" in Wrigley, E. A. and R. S. Schofield (eds.) *The Population History of England, 1541-1871: a reconstruction*. London: Edward Arnold.

Lee R. D. (1993). "Inverse Projections and Demographic Fluctuations: A critical assessment of new methods" in Reher, D. and R. S. Schofield (eds.) *Old and New Methods in Historical Demography*. Oxford: Oxford University Press.

McKeown T. (1976). *The Modern Rise of Population.* London: Edward Arnold.

Notestein F. W. (1953). *Economic Problems of Economic Change*, in Proceedings of the Eight International Conference of Agricultural Economics. London.

Omran A. R. (1971). "The Epidemiological Transition. A theory of the epidemiology of population change", *Millbank Memorial Fund Quarterly*, vol 49:4, 509-538.

Perrenoud A. (1984). "The Mortality Decline in a Long Term Perspective" in Bengtsson, T., G. Fridlizius, and R. Ohlsson (eds.) *Pre-Industrial Population*

Change: The mortality decline and short-term population movements. Lund: Almqvist & Wiksell International.

Preston S. H. and E. van de Walle (1978). "Urban French history in the Nineteenth Century". *Population Studies*, vol 32.

Steckel R. (1983). "Height and Per Capita Income". *Historical Methods*, vol 16.

Schofield R. S. (1984). "Population Growth in the Century after 1750: the role of mortality decline" in Bengtsson, T., G. Fridlizius, and R. Ohlsson (eds.) *Pre-Industrial Population Change*. Lund: Almqvist & Wiksell International.

United Nations (1953). *The Determinants and Consequences of Population Trends.* Population Studies, no 17, New York.

United Nations (1973). *The Determinants and Causes of Population Trends.* Population Studies, no 50, New York.

Social Insurance Studies

No. 1: Perspectives on Mortality Forecasting
I. Current Practice

This volume is the first in a series on mortality forecasting reporting proceedings of a series of workshops, organized by the Stockholm Committee on Mortality Forecasting and sponsored by the Swedish National Social Insurance Board (RFV).

The current volume addresses general issues – the need for mortality forecasts in planning national pension schemes, types of models for forecasting mortality and forecasting models used by statistical agencies in Sweden, Finland and Norway. Current practice is discussed from various perspectives: demographic, epidemiological, and economic-historical.

Stockholm Committee
on Mortality Forecasting

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

RFV
RIKSFÖRSÄKRINGS
VERKET

ISBN 91-89303-24-5
ISSN 1651-680x