



Institute
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Longevity Bulletin

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Longevity Modelling

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Contents

1. Introduction by our President, Fiona Morrison	3
2. Summary by Joseph Lu, Chair of the IFoA's Mortality Research Steering Committee	4
3. Historical analysis of longevity improvements, Richard Willets	6
4. Coronary heart disease mortality improvement, Madhavi Bajekal	8
5. Mortality and the Irish Economy 2000-2013, Mary Hall	12
6. Future mortality improvement: can ageing be treated? Richard Faragher	14
7. Assessing high age mortality, Steve Bale	16
8. Mortality projections by using extrapolative models, Andrew Cairns	18
9. Mortality projections by cause of death and risk factors, Chris Martin	22
10. Recent developments and events	26

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1. Introduction by our President

The last century had seen significant improvements in mortality rates and the continuing increases in life expectancy have brought to the fore the critical importance of longevity modelling. It is not surprising that the understanding of longevity risk and factors affecting future longevity, such as lifestyle, medical advances, and health care policy, are attracting more attention. This issue of the Longevity Bulletin discusses drivers of longevity improvements and considers characteristics of two broad families of models – extrapolative and causal models. You can also read about the updated CMI Mortality Projection Model and forthcoming consultation on page twenty-seven of this Bulletin.

I have a great pleasure in introducing the seventh issue of the Longevity Bulletin. I wish to thank all the contributors and authors for their thought-provoking and informative articles on these crucial subjects of longevity trends and modelling.

We hope that this issue will be read with interest by all those with a technical, professional or personal interest in longevity, ageing and population change.



A handwritten signature in black ink, consisting of the letters 'FM' followed by a long horizontal line.

Fiona Morrison

President, Institute and Faculty of Actuaries

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2. Summary

Joseph Lu, Chair of the IFoA's Mortality Research Steering Committee

The amount of the world's wealth dedicated for retirement has been valued at an astronomical £85.8 trn (Marin, 2013). The adequacy of these funds will depend on the future longevity of the individuals who will be relying on these funds to meet their retirement needs. With pension liabilities of the UK private sector defined benefit pension schemes estimated at £2 trillion (Hymans Robertson, 2015), it could cost these schemes some £60 billion if their pensioners were to live on average one year longer than assumed. Therefore a good understanding of population longevity trends and modelling is crucial for the world's financial system and global economy. This Longevity Bulletin discusses some potential drivers of longevity trends and considers aspects of longevity modelling.

Why are we living longer?

Life expectancy at age 65 for males in England and Wales has risen dramatically by six years between 1970 and 2013, in contrast to a rise of around one year between 1841 and 1970 (Human Mortality Database, 2015). Some 70% of the post-1970 rise in life expectancy is linked to an unprecedented drop in mortality rates related to circulatory diseases such as coronary heart disease (CHD) and stroke.

The IMPACT model was developed to estimate how much of the observed change in CHD mortality over a period could be explained by population-level changes in CHD risk factors and treatments. Between 1981 and 2000 it concluded that half of the fall in CHD mortality in England and Wales could be explained by the net change in risk factors, including smoking, blood pressure, cholesterol, diabetes and obesity. A further 40% could be explained by ground-breaking medical treatments such as statins and coronary artery-bypass graft. The remaining 10% was not explained by the model (Bajekal et al., 2012).

But little was known about whether differences in treatments or risk factors were contributing to widening inequality in CHD mortality between socio-economic groups. A follow up model (IMPACTsec) was developed to analyse this for England between 2000 and 2007. It concluded that changes in the uptake of medical treatments have contributed to about 52% of the fall in CHD deaths, a figure that was similar across all five socio-economic groups categorised by the level of deprivation of their area of residence (Bajekal et al., 2012). Changes in risk factors contributed to only 33% reduction, much less than previous decades' 40%, to CHD mortality reduction overall. The contribution of changes in risk factors was



socio-economically related, explaining 20% of CHD mortality fall for people in the most affluent quintile and 45% in the most deprived quintile. About 14% of CHD mortality reduction was not explained by the model.

The results highlight the important roles of risk factors and medical treatments on longevity. They demonstrate that the relative contribution of risk factors and medical treatments can be different for sub-populations and can change over time. Additionally, they suggest that public health policies that target risk factor reduction and improve treatment uptake could be influential in longevity trends.

Longevity disruption

To mark the launch of this Longevity Bulletin at the IFoA's 2015 Life Conference in Dublin, Mary Hall of University College Dublin and a group of practitioners share insights on the impact that the 2008 financial crisis could have had on mortality rates in Ireland. The authors warned that the crash might have increased the death rates of the younger population aged 25 to 44 by more deaths from suicide, circulatory diseases and cancers. More statistical analyses and international comparison would clarify the role of economy on longevity.

Ageing is associated with an increased risk of many life-threatening diseases. Can a drug be designed to treat ageing? Not now. But things are changing. Currently, drugs are

designed specifically for diseases such as CHD, cancer and stroke mainly because the US Food and Drug Administration (FDA) recognises these diseases as separate, valid targets for medicines. This is being challenged by a new clinical trial that uses a relatively safe, common and cheap diabetic drug, metformin, to study if it can delay the development of age-related disease. The goal is to convince the FDA that ageing can be a drug target, paving a way for future medicines. Actuaries responsible for managing longevity risks should monitor the potential role of age-delaying therapies that might markedly extend life expectancy.

Longevity modelling

The lack of reliable or credible population data at higher ages, such as above age 90, has been a long-standing challenge for estimating mortality rates at higher ages internationally. In the UK, the number of people in each age is derived from the decennial population numbers. Between censuses, census counts are aged forward and adjusted to account for population change including births, deaths and migration to derive annual population estimates. Potential errors in estimates could arise from deficiencies in the data sources used to calculate change assumptions.

Unlike the estimated numbers for people alive, the UK's data on the deceased are collected and recorded more reliably and accurately. Using death data of 'closed' cohorts, where everyone is expected to have died, the number of people alive in each age in previous calendar years can be reconstructed to compare against official estimates. However, death data can also face problems such as late reporting and registration of wrong age. These could skew the estimates of mortality rates at higher ages.

In addition to the challenges of analysing recent and current mortality rates, actuaries have to derive assumptions for future changes in mortality rates. We discuss two broad families of models for the projection of future mortality rates – extrapolative and causal models.

Extrapolative models normally aim to fit historical data and project future total mortality rates. Examples would include the Lee-Carter, Lee-Carter variant and Cairns-Blake-Dowd models. They are purely driven by data and assume that past trends are the best guide to the future. They provide central estimates with measures of uncertainty around it – a feature that is much appreciated in longevity risk management. However, they don't account for the drivers of longevity, such as risk factors or treatments, or differences in cause of death trends.

Explanatory models that link risk factors and treatments to mortality rates of different causes of death have been used in medicine for various reasons. They have the potential to be adopted for actuarial work to understand potential impact of changes in risk factors and treatments on future longevity. Random events could be modelled to help users understand uncertainty around forecasts.

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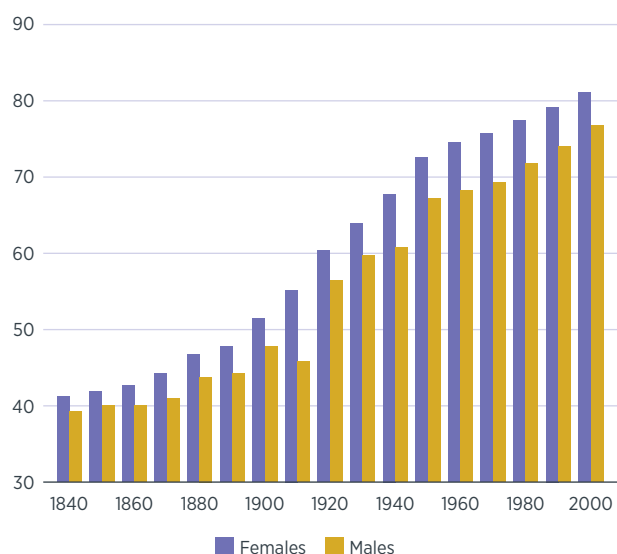
3. Historical analysis of longevity improvements

Richard Willets, Longevity Director, Partnership

What can we learn from looking at the history of longevity improvement in the UK?

Well, for a start, we can see that enormous change has occurred since the first mortality statistics were collected. Life expectancy at birth in the UK has - more or less - doubled in the period since the 1840s and - as Figure 1 illustrates - the average lifespan has increased reasonably steadily (Human Mortality Database, 2015).

Figure 1: Period life expectancy at birth, in England & Wales, by decade and gender.



However, what this doesn't show is that the forces that have driven increased life expectancy have shifted substantially over time.

Until relatively recently, the pace of improvement was far higher for children and young adults, than for older people. Improved sanitation and control of infectious diseases - such as measles, diphtheria and tuberculosis - led to massive reductions in mortality rates at younger ages. Table 1 shows that, by the beginning of the 1970s, mortality rates for young infants had already reduced by 98% (Human Mortality Database, 2015).

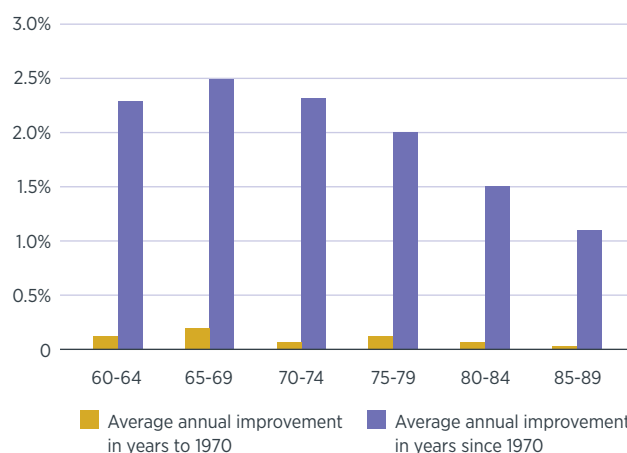
Table 1: Mortality rate in 1970 as a proportion of the equivalent rate in 1841, England & Wales population, for sample ages.

Age	Females	Males
1	2%	2%
21	5%	11%
41	16%	21%
61	43%	79%
81	69%	90%
101	101%	100%

However, the picture was very different for the elderly, especially males, and at the most advanced ages there was little apparent evidence of any significant change. As a consequence, life expectancy for males at state pension age (i.e. 65) in 1970 was barely higher than it was in the middle of the 19th Century, having increased from about 11 to 12 years.

Since 1970 we have witnessed a spectacular shift in the pattern of improvement. Figure 2 contrasts the average pace of improvement in mortality rates for men in their 60s, 70s and 80s before and after 1970 (Human Mortality Database, 2015).

Figure 2: Average annual rate of mortality improvement, males in England & Wales, by age group, periods from 1841-1970 and 1970-2013.



Overall, for men in the 60-89 age range, the average annual improvement rate has been around 20 times higher in the period since 1970 than during period up to 1970. Life expectancy at age 65 has jumped upwards by 6 years.

What has caused this to happen? An analysis of mortality rates by cause of death is helpful. Figure 3 shows how the age-standardised mortality rate for men in England & Wales, split into major cause group, has reduced since 1970 (Office for National Statistics, 2015).

It is clear that the dramatic reduction in circulatory disease mortality (i.e. deaths from causes such as heart disease and stroke) has been the primary driver. In fact around 70% of the overall reduction has been due to fewer deaths in this cause group.

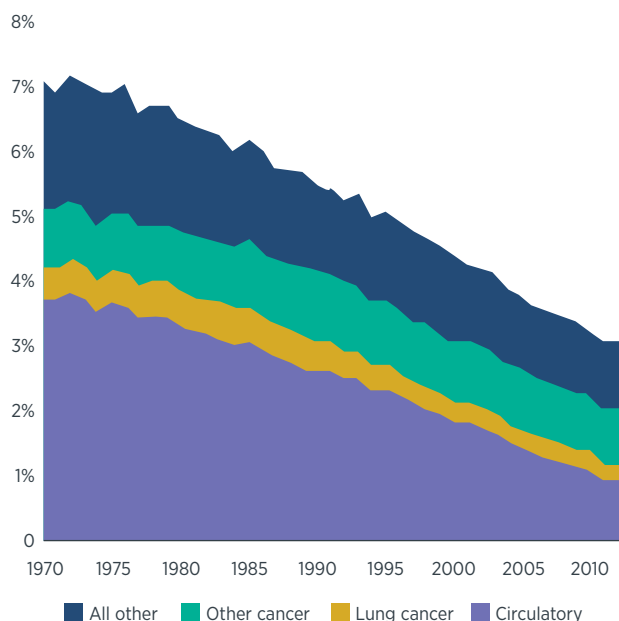
Research has shown that a number of factors have contributed towards this fall; with reduced cigarette smoking being the most significant.

A key question facing actuaries now is whether the recent slowdown in improvement rates at older ages, apparent in data since 2012, is just a 'blip' in the trend or whether we are moving into a new era of more modest change.

Is there still potential for further significant improvement in circulatory disease mortality? Are we seeing an evitable slowing down in aggregate improvement as the main driver of a change runs out of steam? What role are adverse lifestyle factors (such as increased obesity) playing? Has the global recession and economic austerity had a role in shaping recent trends?

The history of mortality improvement tells us that we shouldn't forget that patterns of change and their drivers, that have remained steady for long periods of time, can alter relatively quickly.

Figure 3: Age standardised mortality rate for ages 60-89, males in England & Wales, by cause of death group, 1970-2013.



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4. Coronary heart disease mortality improvement

Dr Madhavi Bajekal, Senior Research Fellow, University College London

Coronary heart disease (CHD) mortality in England has fallen by a remarkable 70% for both men and women in the three decades between 1981 and 2011, and is estimated to have contributed about half of the increase in adult life expectancy in England over the latter half of the 20th Century (Vallin and Mesle, 2004). The overall annual rate of CHD mortality improvement for England was slowest in the 1980s (2.2% per year for men and 1.3% per year for women); gathered pace to 4.0% per year in the 1990s for both men and women; and greater acceleration to 5.8% per year for men and 6.2% per year for women in the 2000s (Table 1).

CHD mortality improvements by socio-economic deprivation, 1981-2010

The rate of mortality decline has usually been described in terms of national age adjusted rates, and little attention has been paid to differentials in terms of age and socio-economic characteristics. When analysed by deprivation, a rapid decline in age-adjusted CHD mortality rates was observed in all deprivation groups (Figure 1) (Bajekal et al., 2013). Thus, the absolute gap in age-adjusted death rates between the most and least deprived groups fell by almost half for men (from 300 per 100,000 in 1982 to 160 per 100,000 in 2010) and more than halved for women (from 161 to 70 per 100,000, respectively).

But the narrowing of the absolute inequality gap was accompanied by a significant widening in relative inequality (or the rate ratio) between the most and least deprived groups. Despite lower initial mortality levels, the annual pace of CHD mortality decline in the most socially advantaged areas was the fastest, in each of the three decades (Table 1). This socially graded pattern of fall was not restricted to just old-age mortality when most deaths occur: we see the same pattern repeated in younger age groups (Figure 2).

Table 1: Average annual rate of CHD mortality decline, by sex and deprivation quintiles. (Notes: based on 3-year moving averages of age-standardised rates, for ages 35 and over, using data from 1981-2011)

	Males			Females		
	1980s	1990s	2000s	1980s	1990s	2000s
Least deprived	2.7	4.0	6.3	1.7	4.1	6.7
Q2	2.4	4.2	6.2	1.7	4.1	6.2
Q3	2.3	4.1	6.0	1.4	3.8	6.3
Q4	2.2	3.8	5.4	1.1	3.7	5.9
Most deprived	1.4	3.6	4.9	0.8	3.9	5.5
England	2.2	4.0	5.8	1.3	4.0	6.2

Figure 1: Trends in age standardised CHD mortality rates in England by deprivation quintiles, 1982 -2010 (ages 35 and over, 3-year moving averages)

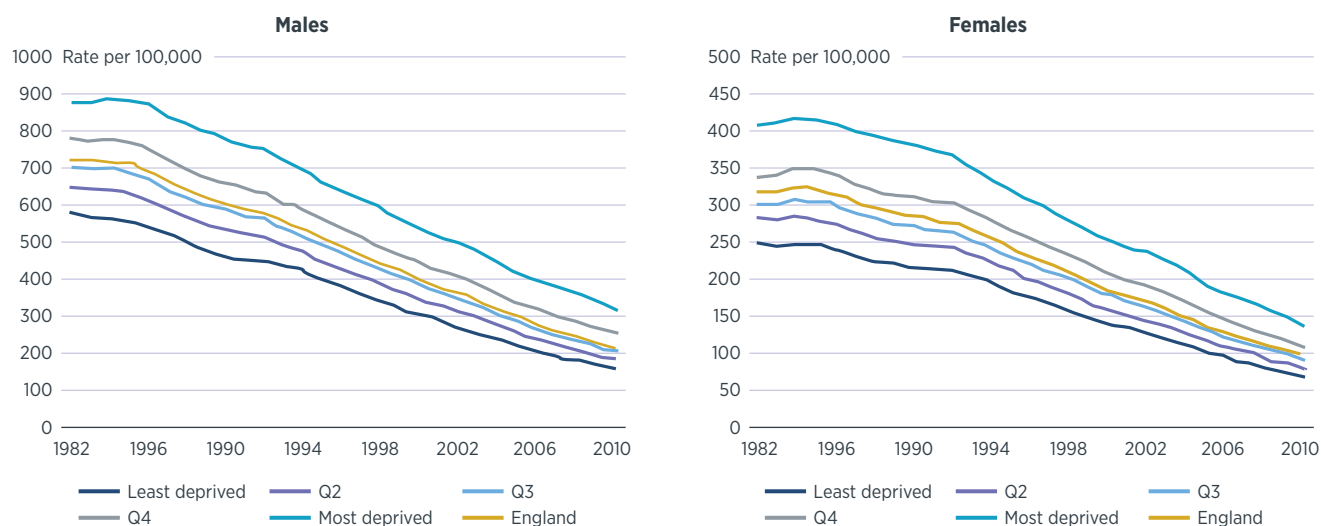
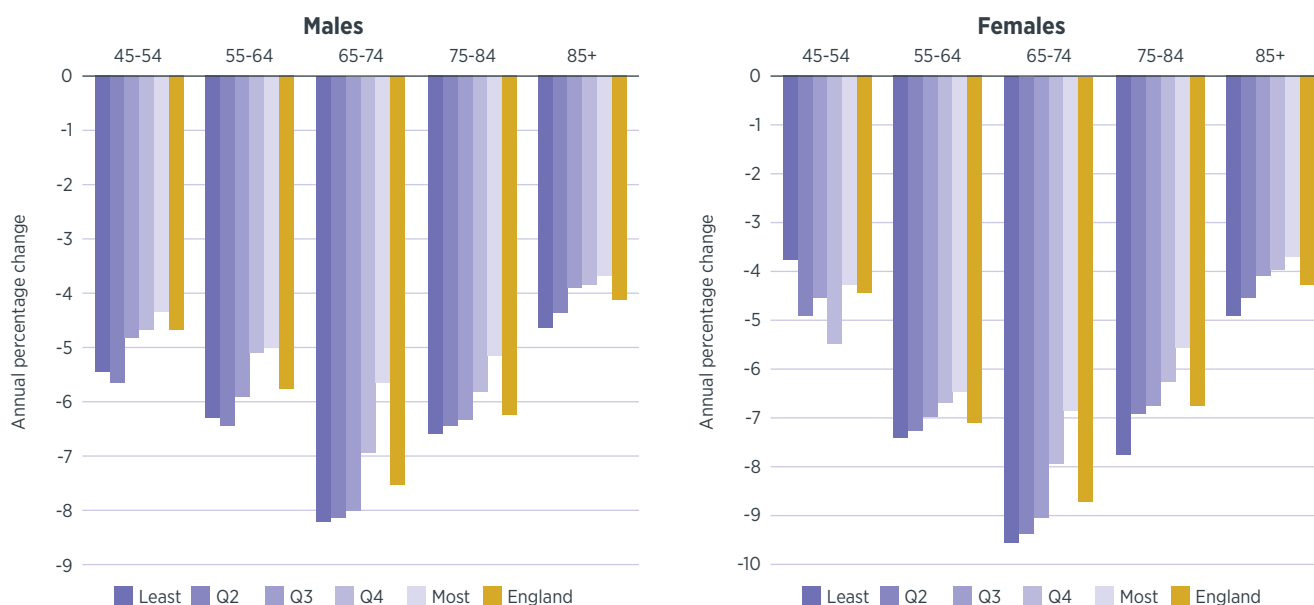


Figure 2: Average annual rates of CHD mortality fall in England, by sex, age group and deprivation,



So why was CHD mortality declining so rapidly, and were residents in deprived areas falling behind because of slower adoption of healthy lifestyles or was it poorer access to modern treatments or both?

Modelling the drivers of CHD mortality decline, 2000-2007

To answer this question we used a model (IMPACTsec) to quantify the variation by socio-economic circumstances in the relative contributions of modifiable population-level risk factors and evidence-based individual treatments to the fall in CHD mortality during the period 2000 to 2007 (Bajekal et al., 2012). This decade saw the introduction of several national initiatives to improve the quality of services provided for CHD prevention, diagnosis, treatment and rehabilitation (Department of Health, 2000; The NHS Information Centre for Health and Social Care, 2004); and public health measures to reduce risk factors across the entire population were introduced including the ban on tobacco advertising (2003); smoke free legislation (2007), and voluntary agreements to reduce salt and artificial trans-fats in processed food (UK Food Standards Agency, 2006; UK Food Standards Agency, 2007).

Previous research by Unal et al. (2004) had examined the fall in CHD mortality between 1981-2000 for England and Wales and concluded that half of the national fall was attributable to the net effect of lifestyle changes, with the uptake of new medical therapies for CHD (like statins and coronary artery-bypass graft) explaining another 40%. The remaining 10% was unexplained.

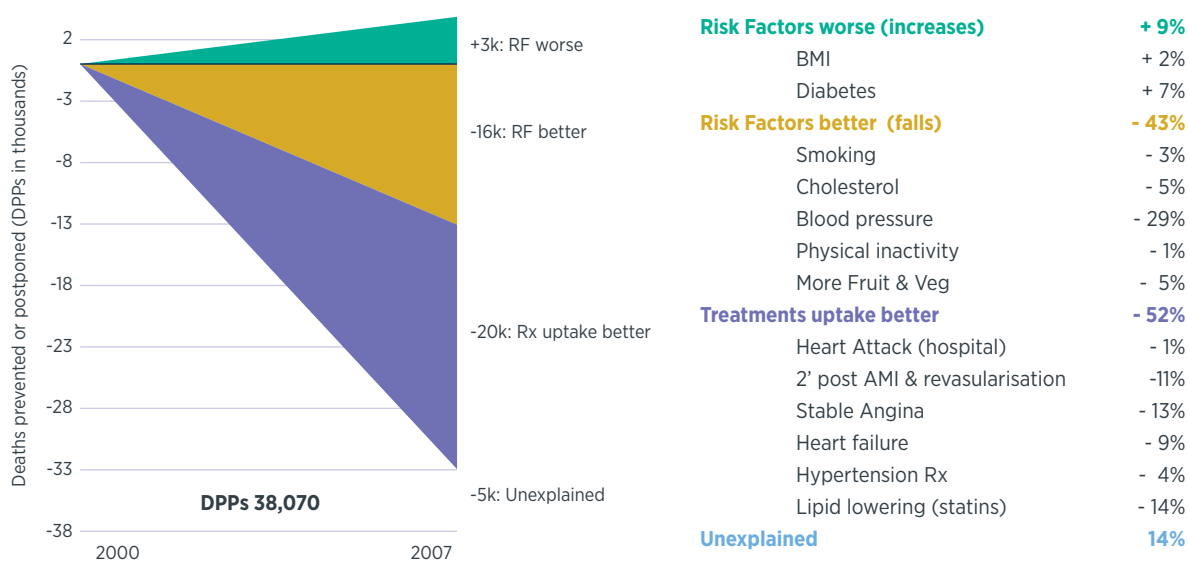
These findings were replicated across a range of countries, including the US, New Zealand, Canada and Scandinavia, indicating that healthier behaviours played a larger role than

better treatment in the CHD decline, with quitting smoking being the most prominent. But in the mid-1990s, after falling continuously from the 1970s, the prevalence of smoking began to level off. At the same time, adverse risk factors such as diabetes and high body mass index (BMI) increased relentlessly, especially in deprived areas. Emerging evidence suggested that CHD mortality rates in young people under 45 were flattening or even reversing. And there were few new therapeutic breakthroughs after the mid-1990s. But despite these adverse trends in the drivers of CHD mortality, rates had continued to fall.

Our IMPACTsec model included the effect on mortality of change in seven of the major risk factors for CHD: smoking, systolic blood pressure, total cholesterol, BMI, diabetes, physical inactivity, and fruit and vegetable consumption between 2000 and 2007; plus effects of changes in the uptake of all 45 medical and surgical treatments currently in use in nine CHD-disease groups. Our model included the total population of England aged 25 and over in 2000 and 2007, segmented by sex, seven age bands and deprivation quintiles.

We found that after 2000, the relative contribution of medical therapies versus risk factors had swapped over: now modern treatments contributed 52% of the mortality improvement, while the net change in risk factors contributed 33% (see Figure 3). Although smoking rates started to drop again from 2001 onwards, the pace of decline was not as dramatic as in previous decades. Compared with the significant falls seen in population blood pressure, stopping smoking made a relatively small contribution. Interestingly, the falls in blood pressure were particularly large in people not taking medication to lower their blood pressure.

Figure 3: Contribution of risk factors and treatments to CHD deaths prevented in 2007, England



Abbreviations: AMI = acute myocardial infarction (heart attack); BMI = body mass index; Rx= Treatment; 2' = secondary prevention by drugs prescribed by GPs to prevent recurrence of heart attack

However, the benefits of improvements in health behaviours across all social groups were partially negated by sharp rises in BMI and diabetes, particularly in the most deprived groups (Scholes et al., 2012). Opposing trends in major risk factors, which varied substantially according to deprivation, meant that their net contribution accounted for just a third of deaths averted overall. In deprived areas, both the positive and the negative changes in risk factors were larger than in the affluent group and together explained about half of the fall. By contrast, risk factors explained only 30% of the fall in affluent areas, with about 20% unexplained by the model.

The contribution of improved treatments was very similar across all socio-economic groups (about 50%). This finding is consistent with equitable service delivery across the NHS, most likely resulting from the implementation of treatment protocols in hospitals and primary care and from giving GPs an incentive to prevent and manage CHD in the community. The increased contribution of treatments to improvements in CHD mortality resulted largely from the doubling of the uptake of effective drugs prescribed by GPs, such as statins, beta-blockers and angiotensin-converting-enzyme (ACE) inhibitors, rather than the introduction of new hospital treatments.

Socially patterned

Overall, the model was unable to explain about 15% of the fall in CHD mortality rates. This too was socially patterned: ranging from 3% in the most deprived quintile to 20% in the least deprived quintile (Figure 4).

Our findings suggest that while UK policies for salt reduction and tobacco control have been relatively effective, adverse trends in risk factors related to diet, including diabetes and BMI, continued unabated and rose fastest in the most disadvantaged groups.

In conclusion the research threw up two findings that were unexpected.

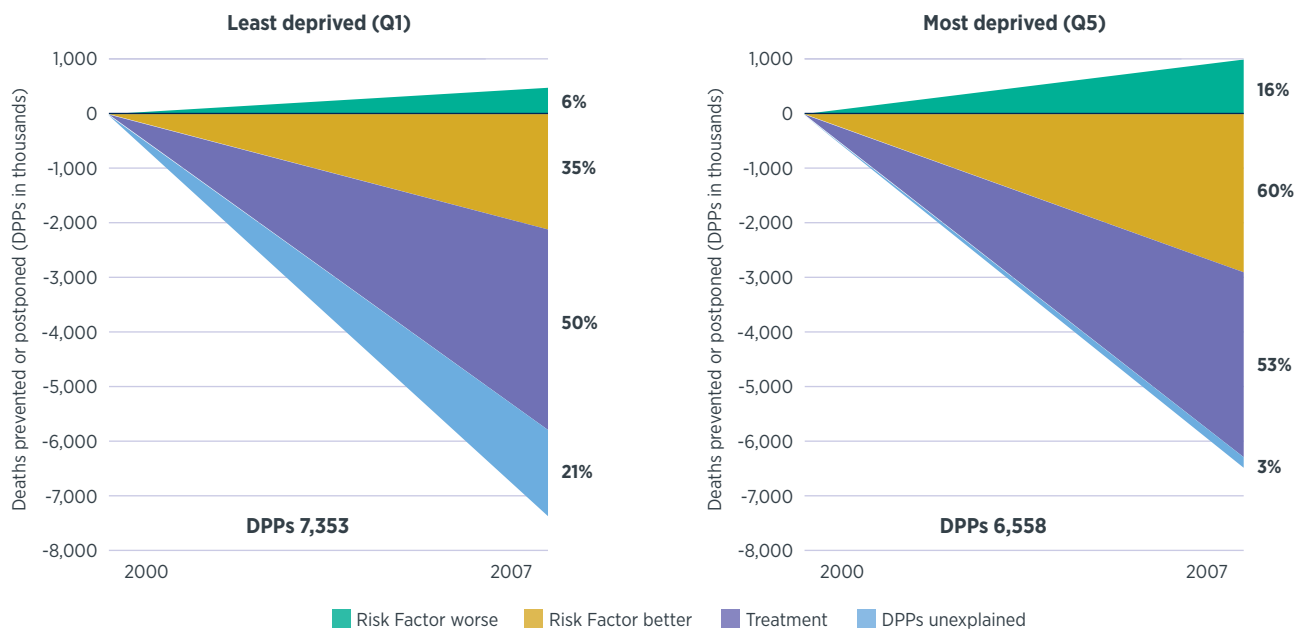
First was the markedly equitable uptake of treatment both in hospital and in the community, and its substantial contribution to falls in CHD mortality across all groups. England in the early 2000s saw a raft of government measures to both reduce social inequalities in health outcomes and to improve the quality and delivery of healthcare – through clinical guidelines, pay-for-performance schemes and the doubling of public expenditure growth. That these measures appear to have been effective, at least in reducing deaths from CHD, underscores the potential for health gain through proper implementation of what we know works, especially so in a country with a universal health service.

The second surprise was that, despite including all the known risk factors and treatments in the model, we were unable to explain a fifth of the improvement in CHD mortality in the most advantaged group (Figure 4). Why did mortality rates improve fastest in the most advantaged quintile, given that risk factor levels and mortality was already low, limiting the potential for gains compared with other quintiles?

There are several possible reasons, but perhaps the most plausible is that the adoption of a range of healthy behaviors across the life course has a synergistic effect in accelerating the fall in mortality rates in the most advantaged group that statistical models cannot capture.

Actuaries involved in pricing and reserving for annuities and pensions will no doubt have an interest in the future progress of mortality differentials, particularly as portfolios are quite often skewed, with a few very large cases in the top social groups having a significant impact on the overall trend.

Figure 4: Contribution of risk factors and treatments to CHD deaths prevented in 2007, least deprived quintile versus most deprived quintile



Source: Bajekal, Scholes, Love et al: Plos Medicine, 2012

Where does this knowledge of the causes of improvements in mortality leave us in terms of projecting the future? It is clear that explanatory models that focus on a major cause of death and attempt to unpack the contribution of each underlying risk factor and treatment to explain the observed change provide greater traction in projecting the likely pace and direction of future change.

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5. Mortality and the Irish Economy 2000-2013

Mary Hall, University College Dublin

Contributing authors: Shane Prendergast - Irish Life, Colm Murphy - Deloitte, John Flanagan - CSO, Aine Houlihan - JG Byrne Consulting Actuaries

Recent trends in the Irish economy

The mortality of a country is influenced by its economy through, for example, spending on health care and the general standard of living of the population. Ireland's economy experienced record growth during the so called "Celtic Tiger" years from the mid 1990's to 2007, with average GDP growth of almost 10% pa and 5.5% pa for the periods 1995-2000 and 2001-2007 respectively (Dept. of Finance, Ireland 2011). The global financial crisis of 2008-2009 had a severe impact on the Irish economy. Ireland entered recession in 2008 and was forced to accept an EU/IMF bailout in November 2010. The bailout imposed severe restrictions on public expenditure – the annual average growth rate in per capita health expenditure dropped by over 3.5% in real terms between 2009 and 2012 (the corresponding growth for the period 2000-2009 was 6.3%) (OECD 2014). Ireland began to emerge from its recession in 2012 and exited the bailout programme in December 2013. In 2014 Ireland achieved an annual GDP growth rate of 4.8% (CSO 2015a).

Recent trends in Irish mortality

Using data on deaths and population from EUROSTAT (2015) two year rates of mortality improvement were calculated from 2000 to 2013 using the formula:

$$m_{x,t} = \frac{\text{Deaths for age group } x \text{ in year } t}{\text{Population for age group } x \text{ in year } t}$$

$$\text{Improvement Rate}_{x,t} = 1 - \left(\frac{\text{average}(m_{x,t}, m_{x,t-1})}{\text{average}(m_{x,t-2}, m_{x,t-3})} \right)^{0.5}$$

Figures 1 and 2 present the two year rates of improvement for males and females respectively. From Figures 1 and 2 it can be seen that rates of improvement were generally higher in the early parts of the century prior to the financial crisis. With the exception of the oldest age group (75+), the remaining age groups for both males and females experienced a sizeable drop in the rate of improvement post the financial crisis between 2007/2008 and 2010/2011. The impact was greatest for young males in the age group 25-44 who actually experienced a dis-improvement in the underlying mortality rates during this period.

Figure 1: 2-year mortality improvement rates for Irish males 2000-2013.

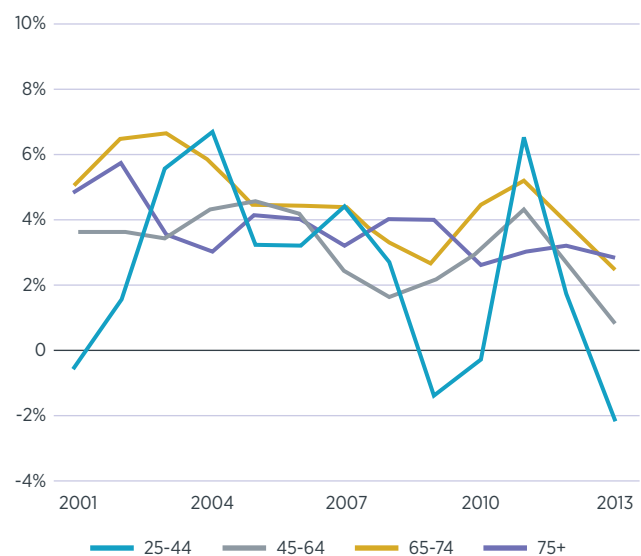
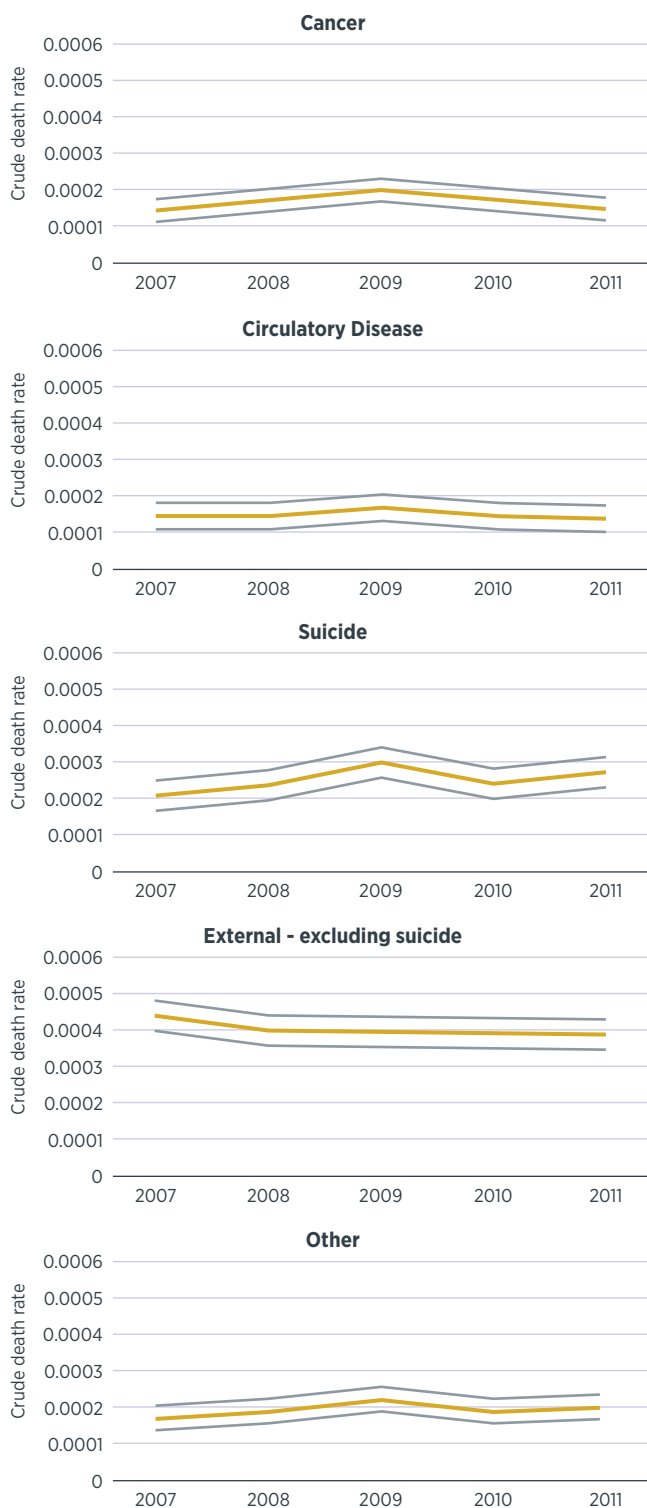


Figure 2: 2-year mortality improvement rates for Irish females 2000-2013



Figure 3: Crude death rates and corresponding 95% confidence intervals for the principal causes of death for Irish males aged 25-44 for the period 2007-2011.



Suicide rates post the financial crisis

In order to understand the significant deterioration in mortality for younger males between 2007/2008 and 2010/2011, it is necessary to look at the underlying causes of death. Using data from the CSO in Ireland (CSO 2015b), on number of deaths

by age, gender and cause and population estimates by age and gender, Figure 3 presents the crude death rate from the principal causes of death (Cancer, Circulatory Diseases, External - Excluding Suicide, Suicide and Other) for the period 2007 to 2011 for Irish males aged 25-44. From Figure 3 it can be seen that deaths from cancer and circulatory diseases increased slightly over the period 2007-2009 before dropping back to 2007 levels by 2011. In contrast, deaths from suicides rose significantly over the same period, peaking in 2009. Deaths from suicide account for over 20% of all male deaths in this age group. Therefore, the increase in the overall death rate for this age group in 2009 can largely be attributed to the increase in the suicide rate. Consistent with our results, several other studies have shown a link between the recent economic crisis and increased suicides in various European countries (Barr et al. 2012, Branas et al. 2015, Corcoran et al. 2015). Male suicides at older ages (45-64) also unfortunately increased over the same period. As suicides form a smaller proportion of deaths at these ages the impact on the overall mortality rate was not as visible.

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6. Can ageing be treated?

Professor Richard Faragher, University of Brighton

Introduction

Ageing is a slippery word, and asking ‘can ageing be treated?’ is fraught with the potential for overstatement and misunderstanding. In this article ageing is not used in its simple sense of ‘growing older’. Instead, ageing is used in its narrow sense to describe the operation of processes within whole organisms or populations of such organisms that result in an exponential increase in the chance of both death (mortality) and sickness (morbidity) over time. Some species, notably *Arctica islandica* (the quahog) and some species of the *cnidarian hydra*, do not show this trend. They are ‘non ageing’ and form a minority, albeit a fascinating one, among the many species inhabiting our planet (Yoshida et al, 2006; Ridgway et al 2011). Ageing is not a universal trait of living things, but it is an exceptionally common one, for good evolutionary reasons (Williams, 1957).

Fundamental discoveries in biogerontology (the science of the biology of ageing) have revolutionised our understanding of both why ageing exists and how organisms age (Kenesary et al. 2013). The result is that enough is now known about the ageing process to intervene in it by many different routes. The medical, financial, conceptual and legal implications of this revolution are profound and the chances of broad spectrum ‘treatments’ are better now than they have ever been. This article explains why.

What causes ageing?

From the 1980s onwards, the isolation and study of single-gene mutants in a variety of species showing both greatly extended lifespans and accelerated ageing has deepened our understanding of the processes which cause organisms to age. In essence, ageing occurs because the mechanisms which act to keep organisms in good health begin to fail over time. These mechanisms may be broken down into two categories in mammals, (1) nutrient sensing mechanisms converging on the Target of Rapamycin (TOR) protein, which lead to the accumulation of damaged cellular components under conditions of high nutrient availability (McElwee et al. 2007); and (2) anti-cancer mechanisms which lead to the progressive accumulation of ‘senescent cells’ within tissue (Baker et al 2011). There is considerable interplay between these two processes and both can be targeted effectively by a plethora of small molecules. Importantly, some of these small molecules are drugs which have already been licenced for clinical use. The best known of these is probably rapamycin (sirolimus) an antibiotic which produces robust improvements in health and

increased lifespan in mice of both sexes. However a range of compounds including acarbose, metformin and α -estradiol have also been shown to increase lifespan typically in a sex-dependent manner. Lifespan extension with these compounds varies from 10% to 22% (Warner, 2015; Wilkinson et al 2012).

The relationship between ageing and age-related disease

For almost two millennia, ageing was conceptualised within the western medical tradition as a ‘natural process’ distinct from ‘unnatural’ disease (Cockayne, 2003). However, this distinction did not result from a detailed mechanistic understanding of the pathologies in question but instead from simple logic. Everyone grows old but not everyone gets a particular age-associated disease went the argument. Therefore ageing and disease are distinct. This chain of reasoning is superficially convincing but amounts to little more than “everyone catches colds but not everyone gets a runny nose each time, therefore runny noses and colds are distinct”.

However, it is now clear that common mechanisms can cause both age-related diseases, and ‘natural ageing changes’ (e.g. the accumulation of senescent cells in the skin contributes to both wrinkling, a ‘natural change’ and to cardiovascular disease, an ‘age-related disease’). As a result, the conceptual basis for maintaining a distinction between ‘natural ageing’ and ‘disease’ is difficult to maintain. Practically, causal unity between the mechanisms of ‘ageing’ and the mechanisms of ‘age-related disease’ opens up the possibility of broad spectrum preventative medicine, which targets multiple age-related diseases and impairments simultaneously.

Ageing as a treatable condition: the TAME study

As a ‘natural process’ ageing has historically not been recognised in law as a treatable condition (i.e. a ‘disease’). This mode of thinking has impeded the design of clinical trials which seek to extend healthspan. However a new clinical trial design is now being considered by the US Food and Drug Administration (FDA) the Targeting Ageing with Metformin (TAME) protocol. A typical clinical trial, in essence, targets a single disease but the essence of the TAME concept is that although the time to first age-related pathology is very variable in humans, the time to development of a second age-associated pathology is much tighter and within the time scale of a typical clinical trial. Designed by a consortium of clinicians and gerontologists led by Dr. Nir Barzilai of Albert Einstein

College of Medicine, TAME proposes to look at duration from any of a range of initial pathologies to any second pathology. A compound which improves healthspan would be predicted to lengthen the time taken to develop a second pathology, keeping subjects healthier for longer.

The anti-diabetic drug metformin was selected as the ageing of choice in the trial design rather than (for example) rapamycin because it is cheap, has an excellent safety record and, above all, because a wealth of epidemiological data already indicates that diabetics taking metformin are at lower risk of cancer than diabetics taking other medicines. Thus there is a high likelihood that the trial, if approved by the FDA would generate positive results on health. Ethically, should this occur then metformin would probably become the standard against which other compounds would be compared (see <http://bit.ly/1P7ZGqO>).

Potential impact on mortality and future developments

Regardless of the progress of TAME or related trials it is important to note that the new knowledge emerging from gerontology is already showing its potential significantly to impact mortality. A case in point is a recent study with a rapamycin related compound everolimus (RAD001). Pretreatment with everolimus is well tolerated and has been shown to significantly improve the immune response to flu vaccination in the older population. The potential impact of this on public health remains to be modelled, at least publically but given the scale of the problem of winter flu is likely to be profound (Mannick et al, 2014).

Typically when discussing the state of the art, gerontologists are asked by those outside the field to describe potential developments over the next thirty years. I am uncertain of the wisdom of this because, as a rule, predicting the future leads to only two types of forecast, the boring, accurate and trite, which attract no public attention, and the exciting, inaccurate and wrong, which attract considerable notoriety. When long term predictions turn out to be accurate this seems to result from serendipity rather than far sightedness on the part of the seer.

Accordingly, this author is reluctant to join the long list of excitingly inaccurate prophets either as a pessimist or an optimist. Patent Commissioner Henry Ellsworth comes to mind as a notable pessimist having told the US Congress in the 19th Century that “everything which can be invented has been invented”. Gordon Rattray Taylor author of *The Biological Time Bomb* (1968) was somewhat optimistic when he opined that the human race would have ‘total control over death’ by the year 2000. With such examples before me I hope my suggestions for what the future holds over the next three decades are boring, trite and possibly even accurate.

It is clear that the ageing process is malleable and no mechanistic barrier separates disease from normal functional decline. This is an important point since it removes both ethical and practical barriers to intervention in the later life phenotype (Faragher, 2015).

It is also clear that drugs already available can lengthen lifespan and improve health. Thus the design of trials specifically intended to measure healthspan rather than single disease progression is feasible and indeed overdue. The results will allow compounds already in the clinical arsenal to achieve their maximum effectiveness in the population as a whole. Researchers are already attempting to develop compounds specifically intended to (for example) target senescent cells. However, any bench to bedside journey for these entities could be of the order of three decades.

Perhaps the most important near-term development is an increased emphasis and understanding of what portion of extended lifespan is spent in good health, as opposed to frailty. The limited evidence available shows that it is possible, depending on the lifespan extending mutant studied, either to extend the healthy period alone or to extend both the healthy and morbid phases of the life course, at least in nematodes (Bansal et al. 2015). The former is clearly beneficial. The effects of the latter could prove more nettlesome.

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7. Assessing high age mortality

Steve Bale, CMI High Age Mortality Working Party

The Office for National Statistics (ONS) 2012-based population projections forecast the proportion of deaths at ages 90 and above to grow from 20% of all deaths in 2013 to 36% of all deaths in 2040 (ONS, 2012). As a result, better estimates of mortality rates at these old ages will become increasingly important. However, for many insurance companies and pension schemes, mortality experience and data coverage is focused on younger ages, so there is considerable uncertainty around mortality experience at older ages.

In response to this, the Continuous Mortality Investigation (CMI) established the High Age Mortality Working Party in 2014 with the following remit:

- to provide a broad indication of the potential financial impact of misestimating high age mortality;
- to investigate and summarise published research on high age mortality;
- to identify potential issues with existing CMI data sources (self-administered pension schemes, insurers and ONS); and
- to consider methodologies specific to estimating high age mortality.

Our work to date is summarised in CMI Working Paper 85 which is available on the IFoA website (CMI, 2015).

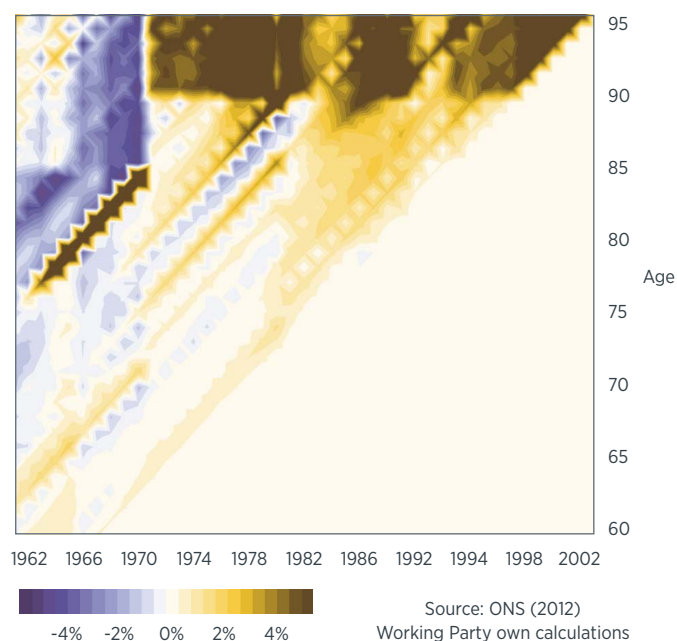
This article sets out some of our key findings.

Mortality of closed cohorts

We have analysed the mortality from closed cohorts (defined here as cohorts by year of birth where everyone is assumed to have died or reached at least their 110th birthday) for the England & Wales population above age 90 as published by the ONS. We have used the deaths registered at each age and calendar year to estimate the population in prior years, and hence, exposure for calculating death rates. We have then compared the implied estimates for mortality against those produced by the ONS which are based on the decennial Census. Figure 1 illustrates the relative difference in the two sets of mortality rates. Warm (red/orange) or cold (blue) colours denote differences between the two sets of rates.

Overall, for males at ages 90 and above, the mortality rates generated by our study of closed cohorts are about 5% higher than the mortality reported by the ONS. For females, the difference is lower at about 1-2%. We understand the underestimation of mortality to be driven by overestimation of population exposures at very high ages.

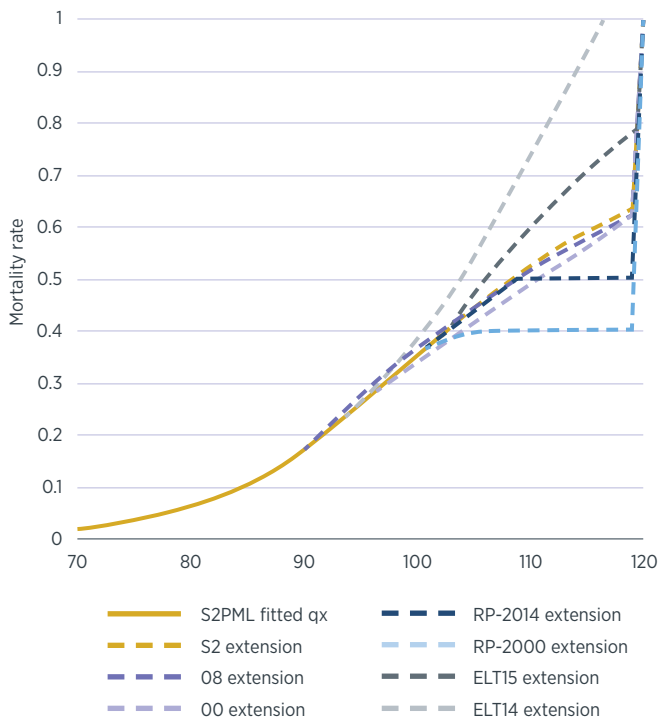
Figure 1: Comparison of closed cohort mortality to reported



Level and shape of high age mortality

We reviewed mortality tables published by the CMI, the ONS and North American actuarial associations and found wide variation in the relative level and shape of mortality assumed at high ages. We have applied methods used in these tables for extrapolating mortality rates at high ages to CMI data on the mortality experience of male pensioners in UK self-administered occupational pension schemes. Figure 2 summarises the results. Relative to the S2PML table – produced by the CMI for this dataset – there is significant variation of -4.1% to +0.4% in cohort life expectancy for a male aged 90, compared to variation of only -0.4% to +0.1% at age 65.

Figure 2: Comparison of different extension methodologies applied to S2PML graduated table at high ages



The debate within the literature we reviewed on whether the percentage increases in mortality rates by age at high ages accelerate, decelerate or remain broadly constant is inconclusive. The S2PML tables effectively assume mortality deceleration occurs, i.e. that the percentage increase in mortality rates at successive ages decreases. If instead it is assumed that mortality rates increase at a broadly constant percentage rate the impact on cohort life expectancy for a male aged 90 at high ages is to reduce them by 2.5% to 0.5%. Again, the impacts are smaller at age 65, being reductions of 0.4% to 0.1% on cohort life expectancy.

Data and modelling issues

We have identified a number of issues with data quality across the variety of sources from which high age mortality is available and have modelled the potential impact of two of these – late reporting of deaths and age mis-statement – on the mortality curve. Our analysis shows that both of these issues can lead to apparent deceleration of the curve at the very old ages, due to the sensitivity of mortality rates to small changes in deaths and exposures, whereas they do not materially impact mortality rates at younger ages.

We welcome feedback on our findings – please email us at: HighAgeMortality@cmilimited.co.uk.

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8. Mortality projections by using extrapolative models

Professor Andrew Cairns, Heriot-Watt University

Actuaries, demographers and other professionals have always been aware of the changing nature of mortality rates and, for many decades, have been projecting future improvements to address questions of fundamental importance in their respective fields of work. However, over the last 20 years or so, increasing attention has been paid to the inaccuracies of past forecasts and, consequently, the level of uncertainty around current projections. Alongside this, the importance of risk measurement and management in insurance has risen considerably, again pointing to a need for assessments of uncertainty around central projections.

In this article, we will discuss the types of model that might be used to project mortality, with a focus on extrapolative models: what are their pros and cons relative to other philosophical approaches. We will also discuss some of the numerous criteria (see Table 1) that an extrapolative model should ideally meet in order to be fit for the intended purpose.

Table 1: Desirable criteria for a stochastic mortality model.

For further discussions and further criteria, see Cairns et al. (2009) and Haberman et al. (2014).

1	Model should be relatively parsimonious, but also fit for the complexity of the intended application.
2	Model mechanics should be transparent to the users.
3	Incorporation of a cohort effect should be possible.
4	Model plus calibration should be compatible with available data.
5	For all key metrics (e.g. mortality and survival rates, life expectancies etc.) the model should produce biologically and demographically plausible projections.
6	For relevant applications, the age and term structure of correlation should be plausible and non-trivial: <ul style="list-style-type: none">• between different ages within a population;• between two populations in a multi-population model;• at different time horizons.
7	Model calibrations and projections of key metrics should be robust.

Mortality projections can be constructed broadly using two approaches. The first contains causal/explanatory models, and the second, extrapolative models. Causal models drill down into the details of mortality, looking at potential causes of change (the inputs) ranging from medical and lifestyle, through to macro-economic factors, through to outputs such as death rates by individual causes of death. Such models allow us to develop a better understanding of the historical drivers of change in aggregate mortality as well as develop a feel for what might happen in the future. Causal models can also often be characterised by their requirement for expert input. Extrapolative models, in contrast, are purely data driven, normally look at all-cause mortality rates, and usually build around the assumption that past trends are the best guide to the future.

Neither approach is perfect and a good user of mortality models should be prepared to combine elements of both before drawing conclusions about the future. Causal models are good for developing individual future scenarios which can be useful for stress and scenario tests in insurance, and might be good also for constructing central forecasts. However, in some cases, causal-based scenarios are limited by the imagination of the user. Extrapolative modellers, in contrast, are modest about their expertise. Instead, they take the view that the pattern (trend plus volatility) in the future will be similar to what we have observed in the past. The frequency, timing and impact of future medical advances and lifestyle changes will be similar, but we don't know what any of these changes will be. A final advantage of extrapolative models is that they give a good idea of how much uncertainty there is around the central forecast: a feature that is very important in an insurance context as well as other applications.

The difference between the two approaches is exemplified by the classic paper by Oeppen and Vaupel (2002) in which they demonstrate that simple extrapolation of life expectancy has repeatedly outperformed causal-based projections.

Early work on extrapolative stochastic modelling focused on single populations starting with the Lee-Carter model (1992) and, the last 20 years have seen the development of many alternatives. Philosophically different approaches were proposed by Cairns et al. (2006, 2009) (CBD) and Currie et

al. (2004), and, between them, the three have spawned many variants, several drawing on the best features of more than one parent model. The development and relationship between models is illustrated in Figure 1. At the right hand side there is an emphasis on multi-population models.¹ In a single population context, the Lee-Carter model and variants of the CBD prevail amongst users.

In a multipopulation setting, progress has been slow, hindered by the lack of good quality sub-population mortality data. As a starting point, a useful and extensive review has been published by Haberman et al. (2014) but the range of models is growing, in parallel with the development of new test datasets. The Li and Lee (2005) model has been an early pace setter. However, as Haberman et al. (2014) point out, and as we will discuss below, there are serious drawbacks to the Li and Lee model: it is a model that is appropriate for some applications but which also has significant limitations that are regularly ignored.² The model can be written as:

$$\log m_i(t,x) = \alpha_p(x) + \beta_p(x)K(t) + \alpha_i(x) + \beta_i(x)\kappa_i(t) \quad (1)$$

where $m_i(t,x)$ is the underlying death rate in population i in calendar year t at age x . $\alpha_p(x) + \beta_p(x)K(t)$ describes the global trend, covering all populations, while $\alpha_i(x)$ describes the structural difference between the global population and population i , and $\beta_i(x)\kappa_i(t)$ describes the stochastic part of the deviation from the global trend. Typically it is assumed that $K(t)$ and the vector $\kappa(t)$ are independent.

At a high level there are two limitations to use of the model that are frequently ignored:

- The model should be calibrated over a limited range of ages (e.g. 50 to 89, rather than, say, 0 to 99) that is linked to the underlying application.
- The model should be used only for constructing central forecasts (i.e. not used for an assessment of uncertainty).

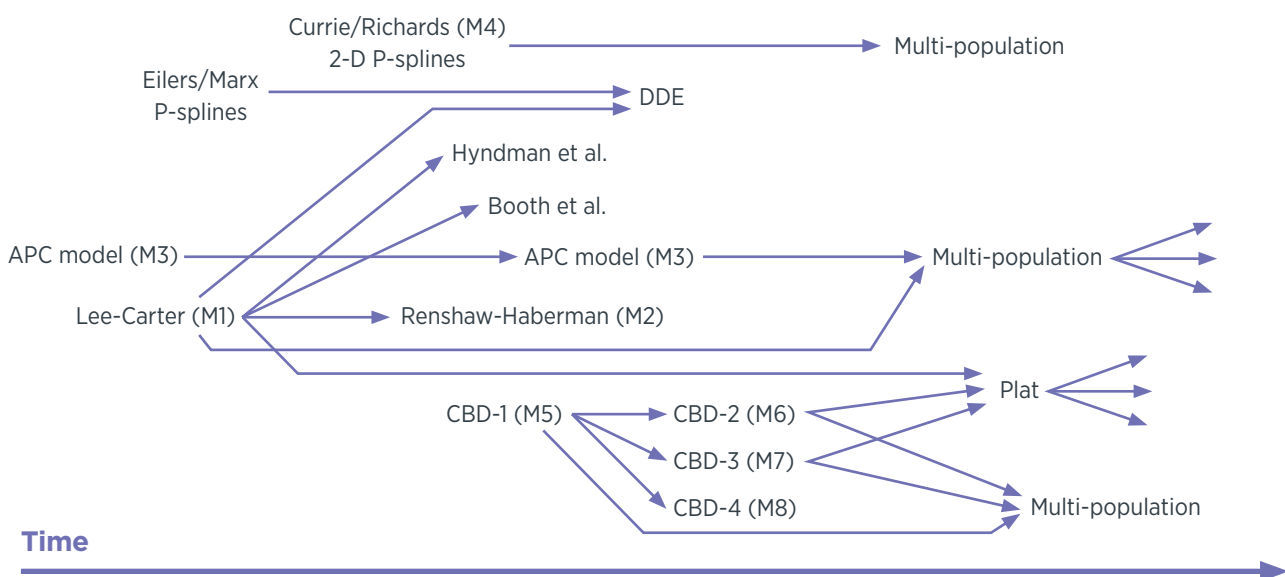
Why should the age range be restricted? Historically, we have seen different rates of improvement over different periods of time and affecting different age groups in very different ways. A single global period effect cannot possibly explain these diverse historical changes across all ages, whereas it has much greater explanatory power if the model is applied to a limited range of ages.

Why restrict to central forecasts only? For a multi-population model we are potentially concerned with the following quantities:

- For each population, i , and age, x :
 - the central projection for $m_i(t,x)$ (and other derived quantities); and
 - the uncertainty around that central projection.
- Correlations between:
 - different ages within the same population;
 - different populations at potentially different ages.

Figure 1: Timeline for the development of stochastic mortality models.

Arrows indicate the influence that individual models have had on the development of later generations. (Adapted from Cairns, 2014.)



¹ To follow up on individual models, see Cairns, 2014, and Haberman et al, 2014, and references therein.

² We need to keep in mind that many financial disasters involved misuse of models, including users who were unaware of, or ignored, the limitations of the models they were using: both good examples of operational risk.

The Li and Lee model suffers from the same problem as the original Lee-Carter model that the age effects $\beta_P(x)$ and $\beta_i(x)$ serve multiple conflicting purposes. Importantly, calibration of these age effects (relative to each other) are driven primarily by historical long term trends rather than historical volatility. So it is reasonable to conclude that on the extrapolative model hypothesis that past trends will continue, central forecasts will be plausible. But then the same $\beta_P(x)$ and $\beta_i(x)$ age effects also dictate relative levels of uncertainty and levels of correlation, almost completely ignoring historical evidence. Two extreme cases stand out. In the Lee-Carter model ($\beta_i(x) = 0$ for all x) if $\beta_P(x) = 0$ then there is no uncertainty in future death rates. In the Li and Lee model, if $\beta_i(x) = 0$ in one population, i (this is quite likely for at least some ages x) and $\beta_j(y) = 0$ in a different population, j , for a different age y (also quite likely) then:

$$\text{cor}(\log m_i(t,x), \log m_j(t,y)) = 1.$$

This, of course, is an absurd conclusion, but these multi-population models are increasingly being used in risk management applications in insurance where correlation is a vital factor. The takeaway, therefore, is that you should think carefully about the requirements of your application before you choose a model.

The way ahead in both single and multi-population settings seems to be to use multiple period effects at the global and sub-population levels with fully (e.g. Cairns et al., 2015, or the M7-M5 model proposed by Haberman et al., 2014) or partially (e.g. the common-age-effect model of Kleinow, 2014) fixed age effects. These allow, to varying extents, decoupling on the central projections from the variance-covariance structure.

Within sample, there are a range of graphical diagnostics that need to be carried out to ensure that the model fits the data adequately. This includes heat plots of residuals (e.g. Cairns et al., 2009, Figure 2) and scatterplots of residuals (e.g. Haberman et al., 2014, Figure 6.4). Other graphical diagnostics are designed to test robustness (e.g. Cairns et al., 2009, Figures 3 to 9). Out of sample projections can also provide a range of graphics to help communicate to users both the central projections and the uncertainty around them. Examples are shown in Figures 2 and 3 based on affluence based analysis of Danish mortality (Cairns et al., 2015). Fan charts introduced in a mortality setting by Dowd et al. (2010) provide an effective way of achieving this including the overlays that can be seen in Figure 2. Figure 3 and variants give a simple way to examine the age structure of correlations within and between groups.

Figure 2: Specimen fan charts for Danish males for two affluence determined decile subgroups.

Left: fitted and projected mortality rates at age 75 with historical rates (dots and crosses). Up to 2012, fan widths for fitted rates reflect small population sampling risk (volatility in the dots and crosses). Middle: partial period life expectancy from age 65. Right: projected ex post survival probabilities from age 65.

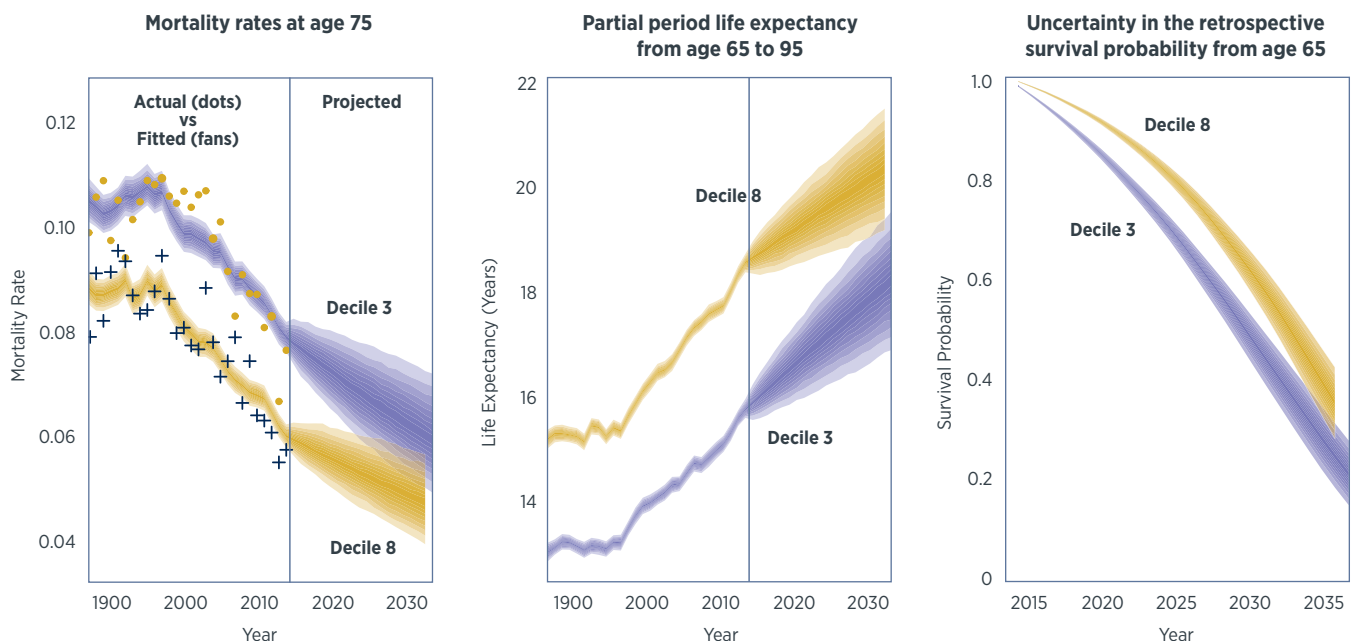
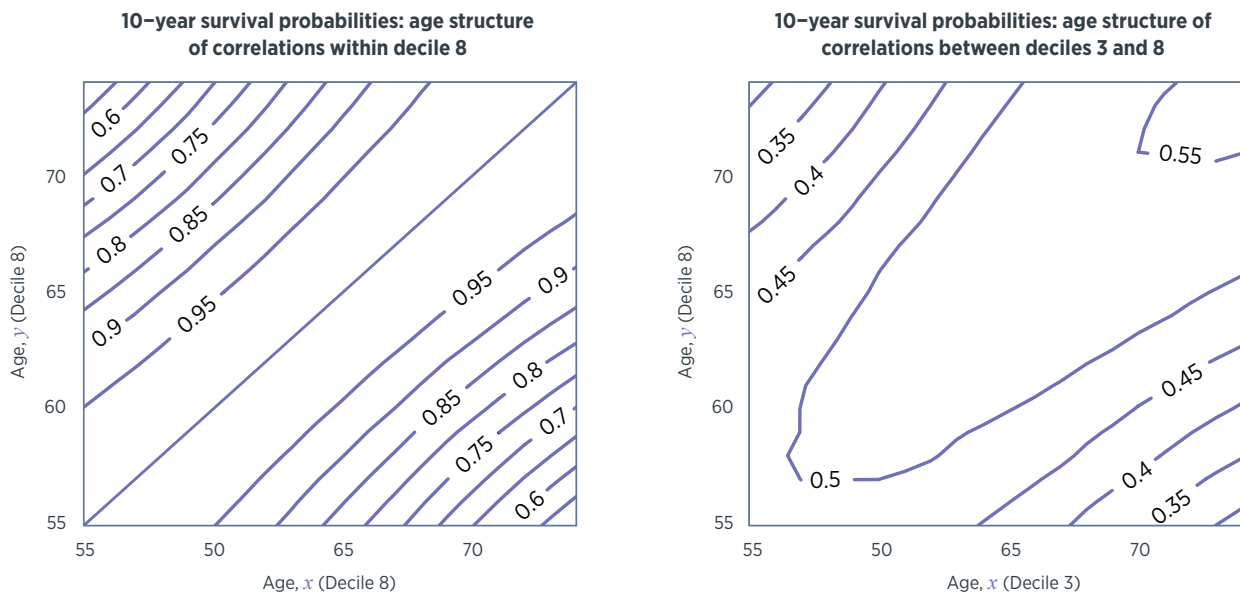


Figure 3: Contour plots of correlations between cohort survival probabilities over 10 years for different cohorts.
 Left: different cohorts within the same decile sub-population 8. Right: different cohorts in decile sub-populations 3 and 8.



A further key issue in the use of extrapolative models is robustness. How robust are key quantities (e.g. fitted and projected mortality rates, estimated period and cohort life expectancies, and derived financial quantities) to small changes in the data used for calibrating the model? Typically, we might think about adding an extra year of data or modifying the age range (e.g. Cairns et al., 2009), or we might wish to test for sensitivity to perceived outliers in the data. Cairns et al. (2009) demonstrate that some models are robust, while others are clearly not. If any elements lack robustness, then end users will not have sufficient trust in what is being recommended, with the fear that a significantly suboptimal decision might be taken. Alternatively, a lack of robustness will cause an end user to back off the use of that model and take a decision based on other potentially flawed methods.

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9. Mortality projections by cause of death and risk factors

Dr Chris Martin, Managing Director, Crystallise

There is increasing interest in analysing mortality rates by cause of death, and the risk factors influencing them, as a means of potentially improving the prediction of future rates of mortality improvement. In 2013, the Institute and Faculty of Actuaries responded to the request from Harry Burns, the then Chief Medical Officer for Scotland, to use the collective wisdom and experience of mortality modelling in the actuarial profession to explore the reasons for the inequalities in life-expectancy both within Scotland and between countries in the UK. This led to the IFoA establishing the “Cause of Death Modelling” working party (Macleod et al., 2014). Here this article will outline the motivations for using cause of death modelling, the advantages and disadvantages of taking that approach and what the current trends are in the medical domain that may be transferrable to the actuarial experience.

Motivation for cause of death and risk factor based modelling

Extrapolative methods of statistical and demographic projection of all-cause mortality rates have become increasingly sophisticated, but are hampered by the need to make two assumptions. Firstly, for many extrapolative models, a long-term mortality improvement target rate needs to be set as a convergence point, and the uncertainty in parameter

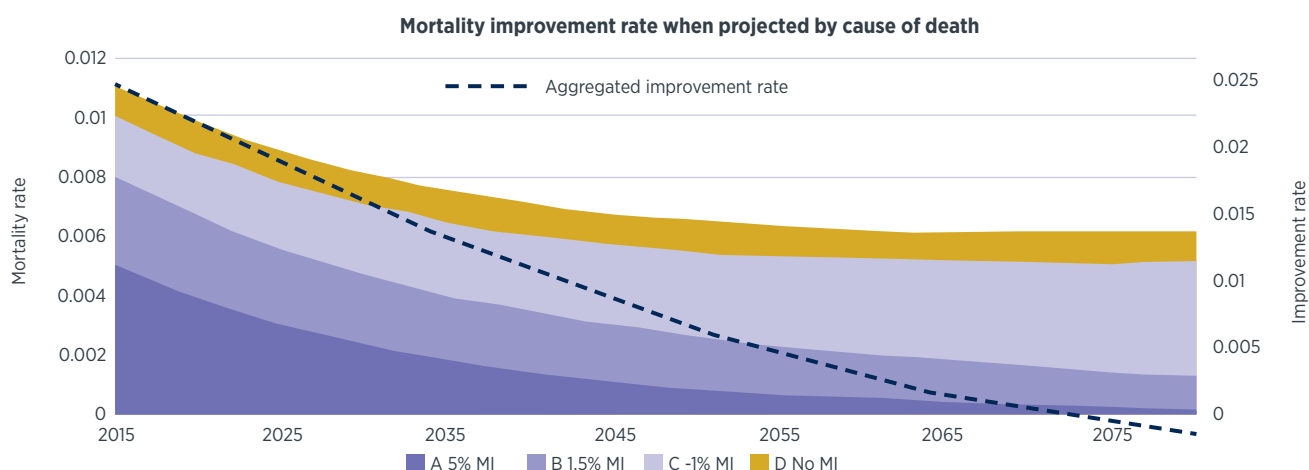
assumption increases with length of time over which it is projected. Secondly, there is a necessary assumption that the aggregate effect of improvement rates in the recent past will continue unchanged into the future (Rosener et al., 2013). This is unlikely to be the case, as there is variation in the rates of change in the environmental factors, medical technological progress and the lifestyle factors that influence mortality rates.

Understanding mortality projection in terms of the underlying cause of death can aid transparency. Bold assumptions of mortality improvement based on all-cause projections alone may be better understood, and be more acceptable, if the underlying trends by cause of death can be demonstrated as plausible, or rejected if they are not. Explanatory models allow the evaluation of the potential impact of emerging medical innovations, bringing some perspective from the clamour of ‘breakthroughs’ reported in the press and the significance they have for pensions or insurance.

Cause of death process modelling

Process models, such as the cause of death extrapolative models, are being explored as a means of utilising additional information (cause-specific mortality improvement rates) that capture some of the changing underlying dynamics of

Figure 1: Chart showing the impact of projecting mortality improvements by cause of death. Four causes of death A, B, C, D each have an improvement rate of 5%, 1.5%, -1% and 0% respectively. This remains constant throughout the projection. The aggregated all-cause rate slowly converges on the lowest improvement rate (-1%). (Own calculations - illustrative figures only).



mortality improvement by cause of death to gain insights into how mortality improvement rates may evolve over time. The absolute impact of relatively high improvement rates in one cause of death will diminish over time as the proportion of all death attributable to that cause diminishes. Where there is any inequality in the initial improvement rates between causes, deterministic models inevitably predict a decline in the all-cause improvement rate which ultimately converges on the worst improvement rate - which can even be negative. By breaking down the modelling by cause, the assumption of the continuation of historical rates into the future is hardened. Flagger mortality improvements in one cause cannot be replaced by accelerations in improvement in other causes in deterministic models.

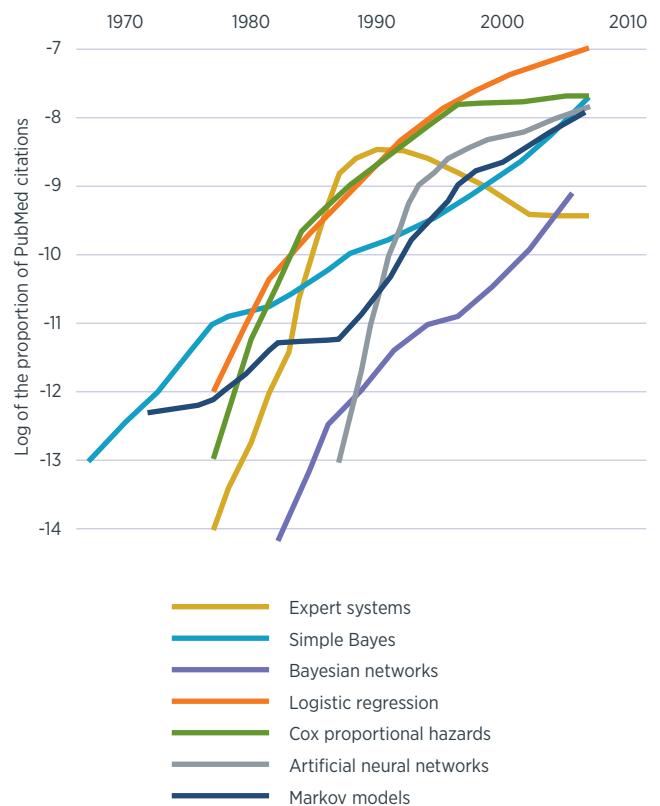
Stochastic modelling of improvement rates by cause can mitigate this particular problem. However, with stochastic cause of death extrapolative models, additional complexity is introduced by the need to accommodate correlations between causes of death. Such correlations are well recognised. For example, mortality rates from coronary artery disease, chronic obstructive pulmonary disease and lung cancer are all strongly linked to smoking, so changes in smoking rates affect the different causes in a correlated fashion. However, this is only a problem in stochastic cause of death models, and does not affect deterministic models using projection of existing rates.

Risk factor-based explanatory modelling

Explanatory models that link the impact of risk factors to mortality or morbidity rates are widely used in medicine for a variety of purposes. For example, decisions to prescribe statin drugs to reduce cholesterol levels, or anti-hypertensive drugs to reduce blood pressure are, in part, routinely based on assessments of the 10-year risk of cardiovascular events like death, heart attacks and strokes in those who are fit and well at the time of assessment. Historically a range of modelling methods have been applied, including simple Bayes, Bayesian networks, expert systems, artificial neural networks and Markov state transition models. However the field is now dominated by regression models such as logistic regression or Cox proportional hazards models (Figure 2)(Martin, 2013).

A variety of regression equations have been used, including the Framingham risk equations (Anderson et al., 1991), QRisk (Hippisley-Cox et al., 2008) and the European SCORE equation (Conroy et al., 2003). Regression models have become dominant in medicine as they tend to perform well in comparison with other methods in terms of predicting risk, and they can be very flexible and versatile in how they are applied to different subjects. They necessarily require large, detailed data sets to allow model fitting, and this data is not always accessible, if it exists at all. Also, they usually relate to a conditional outcome at a specific time horizon and it is not always clear how this can be applied in the time-series modelling necessary for projecting future mortality improvement rates.

Figure 2: A graph showing the proportion of all publications in the US National Library of Medicine's PubMed database that mentioned different modelling methods between 1966 and 2008. (log scale)(Martin 2013).

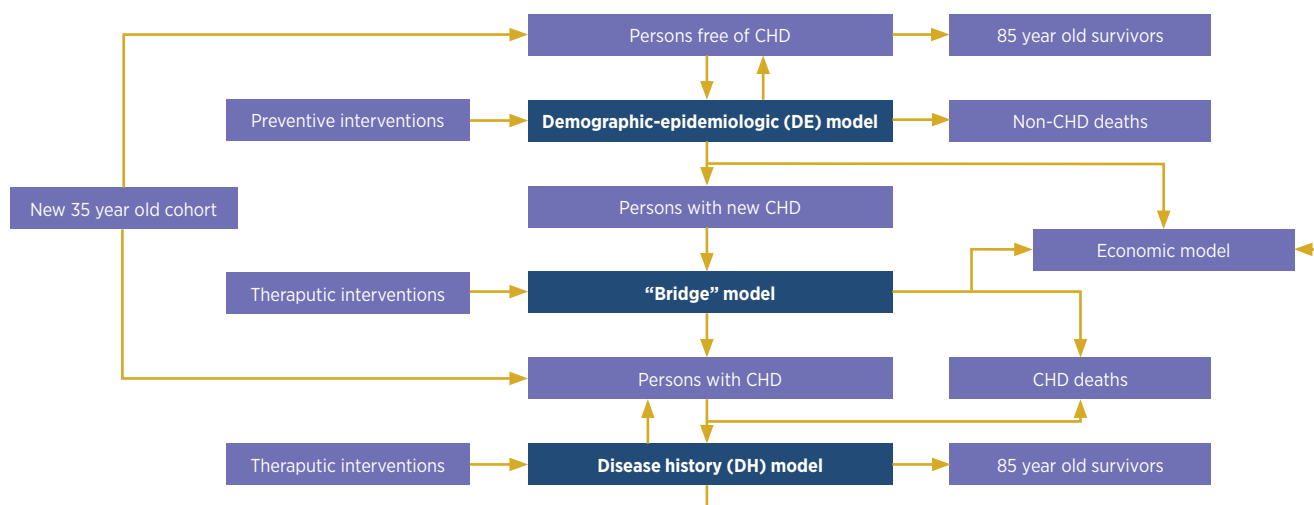


Combined cause of death and risk factor models

Models that combine risk factor explanatory modelling and cause of death process modelling can address some of the weaknesses already mentioned, including the correlations between causes of death.

Markov simulation models have been widely used for generating time series risk estimates in a variety of applications. An actuarial example is the Chatterjee model of heart disease, stroke and death, based on the Framingham data set (Chatterjee, 2008), and a prominent medical example is the Coronary Heart Disease policy model from the USA (Figure 3) (Weinstein et al., 1987)(Unal et al., 2006). These models maintain a set of states such as 'alive', 'sick' and 'dead', or 'smoker' and 'non-smoker', with the probability of transition between the states being calculated at each time cycle based solely on the current states in the model. These are complex models that can be computationally inefficient, and an alternative methodology focussed on the timing of events rather than the states at each time cycle is gaining increasing traction in the medical domain.

Figure 3: The overall structure of the Coronary Heart Disease Policy Model as described in Weinstein 1987. There are three sub-models: the Demographic-epidemiologic (DE) sub-model, which allocates the risks of developing CHD, dying from non-CHD cause, or remaining well; the Bridge sub-model, which covers the first 30 days after diagnosis of CHD; and the disease history sub-model, which allocates those with CHD to one of 12 categories according to type of CHD and interventions like CABG.



Discrete event simulation calculates the future probability density function for a number of events in parallel, based on risk factors that influence the probability of those events. For example, probability density curves for the events ‘heart attack’, ‘stroke’, ‘quitting smoking’, ‘developing diabetes’ or ‘developing hypertension’ could be calculated based on factors like age, gender, smoking status, current blood pressure and cholesterol levels, diabetes state and blood sugar levels. A random number would be generated for each event to determine which would occur first. The modelling then leaps to that point in time, and fresh probability densities are calculated. They are necessarily stochastic models, and the projected risks are based on large numbers of simulations, but can efficiently incorporate complex chains of events. They can be a more practical solution than cohort Markov micro-simulation, which progress in time-cycle steps, when the models are very complex. Medical examples of the discrete event simulation in medicine includes the ‘Patient Orientated Simulation Technique’ (POST), which has been used to model the impact of different coronary heart disease treatment strategies and prospective screening methods (Cooper et al., 2002). Another is the modelling of sudden cardiac death and the impact of interventions to prevent it (Andreev et al., 2013).

As a worked example, a stochastic Markov chain Monte Carlo micro-simulation of how projected changes in obesity rates might impact on future mortality improvement rates in women from different socio-economic groups was conducted. Details of the methodology can be found in the author’s PhD thesis (Martin, 2013). Three socio-economic groups were examined, NSSEC 1.1 ‘Large employers and higher managerial occupations’, NSSEC 2 ‘Lower managerial and professional occupations’ and NSSEC 7 – ‘Routine occupations’. A forward projection of the current trend in obesity rates was taken as the ‘expected’ scenario, and this was compared to a symmetrical reversal of

trends back to their 1990 rates, with higher reduction rates in the higher socio-economic groups (Figure 4). The impact of obesity on mortality was mediated via diabetes, cardiovascular disease and cancers including breast, kidney and endometrial cancer.

Figure 4: Showing the trend in the rise in obesity in men in the UK with a forwardly projecting ‘expected’ scenario and improvement scenarios segmented by socio-economic group encompassing a symmetrical reversal in the trend over the next six decades.

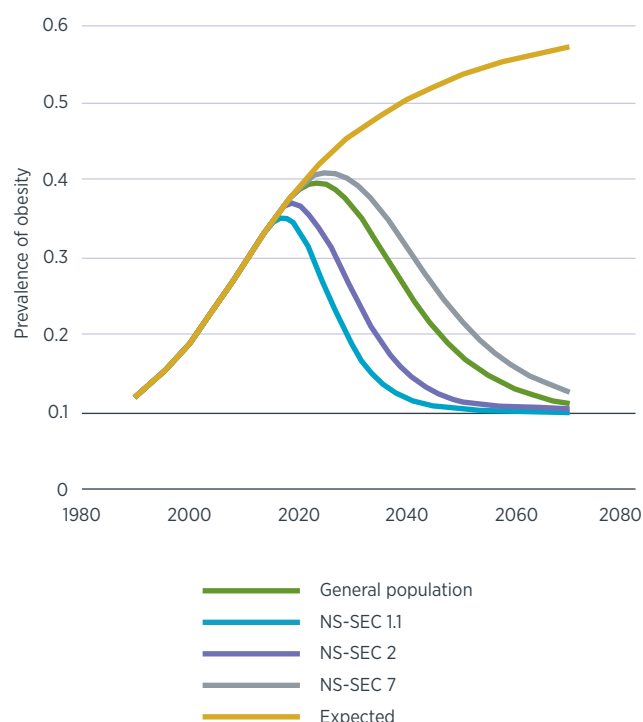


Figure 5: The mortality improvement rates generated by the expected and improvement scenarios for a 60 year old woman.



The impact of the improvement trends on mortality improvement are surprisingly modest, with peaks in additional improvement of about 0.1% per annum (Figure 5).

Summary

Markov and discrete event simulation techniques incorporating both cause of death and risk-factor elements can modify estimates of future mortality rates based on current developments that can be projected forwards. However, they are not able to accommodate the ‘unknown unknowns’ that will inevitably occur: those future events that will impact mortality that we cannot anticipate or imagine in the present. However, they can be useful in testing the plausible boundaries of future mortality improvement rates.

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10. Recent developments and events

News from the IFoA

Save the date: IFoA International Mortality and Longevity Symposium, 7- 9 September 2016

This international symposium will bring together thought leaders across all the relevant disciplines to discuss the latest thinking on the drivers and future of mortality trends in populations. Whether you are an actuary, modeller, medical scientist, epidemiologist, researcher or other professional, this symposium will provide opportunities to gain new insights and learn new techniques, as well as networking to enhance your professional activity.

The symposium will cover the following topics:

- How will longevity in the population develop in the future?
- How to project future trends by incorporating research from wider fields including statistics, medical sciences, epidemiology and demography.
- Differences in mortality and mortality improvement rates in sub-populations such as gender, socio-economic circumstances and health status.
- Causal processes of morbidity or mortality trends.
- Issues relating to morbidity and mortality trends including evidence, modelling and cost of care.
- What would disrupt current mortality trends?
- How will Big Data contribute to mortality and longevity trends and analyses?
- New techniques for mortality and longevity analyses and forecasting.

Venue: Royal Holloway, University of London in Egham, Surrey, TX20 0EX.

Call for Speakers: To submit a proposal for a presentation or a poster please complete the call for speakers survey by Friday 26 February 2016: www.surveymonkey.com/r/Symp2016

Putting Life on the Table: The Institute and Faculty of Actuaries publishes Mortality Data Directory

The Institute and Faculty of Actuaries has compiled a directory of datasets which cover the UK and Ireland as well as those that provide an overview of European and world data. The directory not only lists the datasets and provides links to each, but also provides some details on data points of interest to actuaries and the timeframe over which the data was collected.

It is hoped that greater access to and awareness of these datasets will enable more accurate modelling, allowing actuaries to make informed decisions regarding longevity and mortality in relation to life assurance, pensions and long term care products.

To access this free resource please visit <http://bit.ly/20x5emd>

IFoA Thought Leadership Lectures

The IFoA's 2015 Autumn lecture took place in Edinburgh on 9 November and was delivered by Lady Susan Rice. In this lecture she drew on her unparalleled experience to explore the vital role of banking and business has to play in society. This event was live streamed and can be watched on the IFoA website <http://bit.ly/1N7hynU>

SAVE THE DATE! Our 2016 Spring Lecture will be delivered by Sir David Spiegelhalter, Winton Professor of the Public Understanding of Risk at the University of Cambridge, on 4 May 2016 in London. With a background in medical statistics, particularly the use of Bayesian methods in clinical trials, health technology assessment and drug safety, we expect this lecture to appeal to a wide audience

British Actuarial Journal 20th Anniversary

In 2015 the British Actuarial Journal celebrated its 20th anniversary. To mark the occasion the IFoA published a special introduction piece written by three members, David Wilkie, David Hare and Andrew Smith. The reflection piece is available at <http://bit.ly/1Sb9m5Z> and readers can also view an online collection of BAJ articles drawn from this piece here <http://bit.ly/1GyyBhn>

News from the CMI

The Continuous Mortality Investigation (CMI) carries out research into mortality and morbidity experience, providing outputs that are widely used by UK life insurance companies and pension funds. The following is a summary of the CMI's latest outputs.

The CMI Mortality Projections Model and forthcoming consultation

The latest version of the CMI Model, CMI_2015, was published in September 2015. The Model uses ONS England & Wales population mortality experience covering the period 1 January 1975 to 31 July 2015 to model a smoothed fit of past experience, which converges over time into a single long-term rate of mortality improvement, which must be input by the user. The Model and supporting documentation are available alongside Working Paper 84 for subscribers to the CMI. <http://bit.ly/1NCQafK>

The Mortality Projections Committee is also currently reviewing the responsiveness of the CMI Model to new data and developing alternative methods for projecting mortality. The Committee hosted public meetings during October 2015 to discuss issues with the current Model, to make public their current thinking and to give others the opportunity to influence the Committee's views. Slides from the events can be viewed by all on the IFoA's website: <http://bit.ly/1OqlzoQ>. A recording of the London meeting will be made available on the IFoA's website during November 2015. The Committee intends to hold a formal consultation on possible revisions to the Model during 2016, with the next version of the Model scheduled for release in March 2017.

Recent mortality in England & Wales

Mortality experience in the early months of 2015 was exceptionally high and follows a period of low mortality improvements that began in 2011. This is examined further in Working Paper 83. <http://bit.ly/1QiTGhl>

The CMI format for heatmaps of mortality improvements

The CMI has adopted a new format for heatmaps of mortality improvements, in particular to allow them to be more easily interpreted by colourblind readers. Working Paper 82 was released with an accompanying Excel macro to allow others to adopt the CMI's format. <http://bit.ly/1HdDQOG>

Final "08" Series annuities tables

The final "08" Series annuitant mortality tables based on insurance companies' data covering the period 2007-2010 were released in June 2015. These update the "00" Series mortality tables, which were based on data covering the period 1999-2002. <http://bit.ly/1WjuWul>

Report of the Graduation and Modelling Working Party

The Graduation and Modelling Working Party was established to review the CMI's approach to mortality tables and projections and make broad recommendations on modelling. Working Paper 77 sets out its findings. <http://bit.ly/1NCQnzt>

If you have any questions about the CMI or are interested in becoming a subscriber to the CMI's full library, please email us at info@cmilimited.co.uk



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