features@theactuary.com

Jon Palin outlines a new structural model of mortality that takes into account the underlying drivers such as lifestyle trends

LIVE LONG AND PROSPER

The CMI Mortality Projections Model provides a useful framework for considering and communicating the cost of improving longevity. Under this approach, projected mortality improvements move smoothly over time from current observed rates to a long-term rate. But it leaves unanswered the question of what long-term rate of mortality improvement should be assumed.

The CMI suggests that "the long-term rate is better informed by 'expert opinion' and analysis of long-term patterns of change and the causes driving them". Several different approaches could be taken.

One option is to assume that mortality improvements revert to historical average rates. But it is not obvious why this should be appropriate, when past periods have seen large differences in the predominant cause of death, medical treatments and lifestyle practices such as smoking.

Another option is to use a statistical model such as Lee Carter. As a stochastic model, it provides a valuable indication of uncertainty in projections. But the central projection is just extrapolating recent trends seen in the calibration period. Other similar statistical models have greater complexity, but still consist of splitting past trends into component parts and assuming that the historical trend for each is repeated.

Another possible approach is to break down mortality into its constituent causes, extrapolate these causes independently, and then recombine them. This raises similar concerns to other extrapolative methods and also has concerns of its own. In particular, it is difficult to model correlated causes of death such as cancer and cardiovascular disease (CVD), which are both linked to smoking.

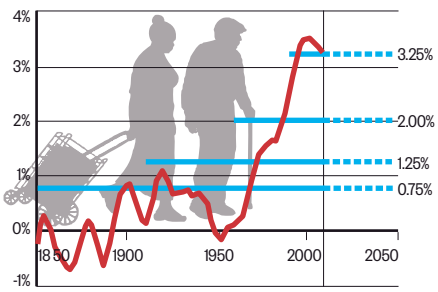
All three approaches can seem appealingly objective. But all contain the assumption that the past is a good guide to the future. Since improvements have varied over time, the choice over which period to calibrate to is subjective and material (*see figure 1*).

A structural model

The alternative approach is a 'structural model'. We still base current improvements on current observed data, but take a different approach to future improvements. Informed by work on natural catastrophe and pandemic mortality models where historical data can be sparse, we take a bottom-up approach and look at the underlying drivers of mortality.

We base our model on what we call 'vitagions': the underlying drivers of mortality. The five vitagions are lifestyle (smoking, diet, exercise, stress); medical interventions (drugs, screening, surgery); the health environment (sanitation, pollution, housing); regenerative medicine (stem cell therapy, nanomedicine); and age retardation (telomerase activation). Key factors in our choice of vitagions are that they are distinct, few, and can be treated as being independent. This avoids the complexities of traditional

Figure 1: Mortality improvements
and averages over 20, 50, 100 and 150 years for males aged 65 (England & Wales)



"We base our model on drivers of mortality including lifestyle (smoking, diet, exercise, stress)"

GETTY / CHARTS: J PALIN

cause-of-death models, where it is difficult to manage large numbers of correlations.

Our model focuses not on the long-term annual mortality improvement rates, but on long-term limits to how much cumulative improvement can come from a single cause. We want to limit mortality rates to what is biologically and socially plausible. We use the term 'Vmax' for the maximum progress that can be made from a single vitagion.

We know that mortality for smokers at retirement is two-and-a-half times as high as for non-smokers. So we can calculate the improvement if everyone stopped smoking: at age 65 we would see mortality rates fall by about 20%. Add in improved blood pressure, BMI, cholesterol, stress levels and others, allow a margin for unknowns, and we get a maximum possible fall in mortality of over 40% from the lifestyle vitagion.

Similarly, we can investigate the limits to mortality improvements from treatments such as stem cell therapy. The effectiveness will vary by cause of death: it seems promising for replacing damaged tissue, but will not stop you getting run down by a bus. So we consider each treatment and cause of death in turn, and combine them to get a maximum possible fall in mortality of around one-third from the regenerative medicine vitagion.

We also need to consider constraints on when mortality improvements might be realised. It has taken 20-30 years for smoking prevalence (*see figure 2*) to fall from 50% of

Jon Palin is a director at RMS, specialising in longevity modelling and risk management

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the population to 25%. So we do not expect rates to fall to zero overnight. Even if they did, recently stopped smokers would not have the same longevity as those who have never smoked. Drugs require around a decade for development, clinical trials, and wide adoption. Stem cell therapy is in its infancy and, while experimental treatments show promise, they have huge costs. We expect it will take around 50 years until improvements from regenerative medicine peak.

The research needed to calibrate the model is large, so we collate data from a variety of publications, 'crowd-sourcing' and cross-checking multiple views where possible. To do so requires a multi-disciplinary team.

Putting all of this together, our central view for a pensioner portfolio is equivalent to the CMI model with a long-term rate of around 2%. This is above typical pension scheme practice, but below an extrapolative model such as Lee Carter. We also find that our uncertainty around the central estimate is lower than most stochastic models, since we have constrained the model to behave in biologically and socially realistic ways.

The main aim is to understand both the expectation and uncertainty of future mortality rates. But this approach also has fringe benefits. It helps to explain potential future changes in terms that are accessible to lay people such as trustees.

Existing actuarial models for longevity improvement either extrapolate the past or rely on pure judgement. Bottom-up cause-of-death models are intractably complex. We suggest a hybrid approach. A

Figure 2: Smoking prevalence

among 40-year-old males (UK)