

Speech to Institute of Actuaries Sessional Meeting on 24<sup>th</sup> October 2005.

Thank you all for coming along this evening. I would like to begin by expressing thanks on behalf of all three authors of tonight's paper for the opportunity to present the results of our work to you. We would also like to thank the Government Actuary's Department for the data on which that work is based.

The paper (Richards, Kirkby and Currie, 2005) explores the role of year of birth in explaining mortality patterns in the population of England & Wales. There are strong grounds in believing that year of birth should be significant: lifetime cigarette consumption, pre-natal exposure to infectious disease, and general standards of living during pregnancy and childhood all changed substantially from one generation to the next in the first half of the last century.

The dependence of mortality rates on year of birth is known as the cohort effect. One particularly relevant question is whether the cohort effect is driven mainly by changes in the incidence of smoking. If this were the case, then the cohort effect might be a one-off step change in mortality rates — albeit spread over a period of time — and therefore unlikely to be repeated. Changes in smoking habits are undoubtedly an important part of the cohort effect, as Figure 2 demonstrates. Indeed, the vertical axis in Figures 2(a) and 2(b) show why cohort-related mortality improvements in males are expected to be larger than those for females: males smoked much more, and therefore had more excess mortality to lose. However, changes in smoking incidence cannot be the sole driver of the cohort effect, and possibly not even the primary one. Figure 2(c) shows the survival curves for life-long non-smokers born during successive decades of the early 20th Century. The cohort-related differences in survival are clear, and, since life-long non-smokers cannot benefit from giving up something they never did in the first place, one wonders what has driven their survival improvements if it cannot be changes in their non-existent smoking habits?

One possible answer lies in the major body of modern research into the foetal origins of adult disease. Figure 1 shows that the 1920s and 1930s were a period of particularly rapid falls in exposure to infectious and respiratory disease. This reduction in exposure to pathogens in

early life is mirrored sixty years later by a sustained and dramatic fall in mortality due to circulatory disease. It is perhaps more than coincidence that this steep fall in heart-disease mortality started when the 1920s generation reached an age when they would be expected to show increasing age-related mortality. What could be the causal link between early-life exposure to pathogens and late-life adult mortality? Increasingly it is thought that exposure to these pathogens can disrupt normal physical development, especially if exposure takes place at sensitive times during pregnancy. If true, then the cohort effect is about successive generations being less damaged than earlier ones.

The paper (Richards, Kirkby and Currie, 2005) uses various methods to confirm what is already known or suspected: that mortality has been falling steadily for the past forty years, and that the pace of improvement is fastest for certain birth cohorts. The paper also shows something that is not quite as widely appreciated, namely that the pace of mortality improvement has steadily accelerated. Figure 16 shows a clear trend for mortality improvements to accelerate over the forty-year period shown. The 1931 birth cohort started with mortality improvements of around 1–2% per annum, then 2–3%, then 3–4%, and, most recently, over 4% per annum. What grounds do we have to believe that these improvements won't continue to accelerate? On the basis of a forty-year trend, why would we believe that improvements would stabilise soon, let alone slow down? Figure 1 also shows a near-linear downward trend in mortality due to circulatory disease for nearly forty years. What grounds do we have to believe that this downward trend won't simply continue?

As actuaries, we are not just interested in demographic change for its own sake, but we are tasked with assessing its financial impact. We often advise clients with substantial longevity liabilities, be they insurance-company annuity portfolios or defined-benefit pension schemes. We need to make our clients aware of the likelihood that longevity will continue increasing for the foreseeable future, and that the improvements to come might be just as dramatic as the improvements already seen to-date. I personally see the biggest implications of improving longevity as lying with defined-benefit pension schemes, and I would like to explain why.

Last year, Gavin Jones and I presented a paper on the financial aspects of longevity risk to

SIAS (Richards and Jones, 2004). In it, we claimed:

*“Some of the greatest future ‘surprises’ in longevity risk may come from companies with large defined-benefit pension schemes.”*

Barely four months later, British Aerospace (2005) obliged us by announcing an overnight 17% increase in pension-scheme liabilities. I happen to think that an extra £2.1 billion of liabilities qualifies as a surprise for most people.

The pace, however, is quickening fast. The very same day this paper was published, Delphi, a large US manufacturer of car parts, filed for bankruptcy under Chapter 11 of the US code. *The Economist* (2005) cited Delphi’s future pension obligations as being valued at US\$8.5 billion, of which US\$4.3 billion is unfunded. In a statement to the Financial Times (2005), Delphi’s chief executive, Steve Miller, was characteristically direct about the role played by the pension scheme in his company’s woes:

*“Defined-benefit programmes are an anachronism, and we are witnessing the slow, agonising death of defined benefits as industrial compensation policy.”*

Pension-scheme deficits are no rarity in the United Kingdom either — 92 of the FTSE 100 companies have combined FRS17 liabilities of £364 billion, of which £53 billion is unfunded (Richards, 2005). But is this definitive? What mortality bases were used to calculate this? Are these bases realistic? How many of these schemes contain ‘surprises’ like British Aerospace’s? The question is not academic:

*“Under IFRS the [pension-scheme] deficit will appear on the balance sheet next year and will have a significant adverse impact on reserves, in particular distributable reserves.”*

These aren’t my words, but British Airways’ own comment (2005) on the ability of a company to pay dividends to shareholders when pension-scheme deficits are brought onto the balance sheet.

We need a debate on longevity risk, and an informed debate needs information. However, too few defined-benefit pension schemes publish their mortality basis. Until they do, it is

impossible to comment on whether stated pension-scheme liabilities are realistic. Shareholders should press for this information as longevity risk is now something which directly affects the health of their investment. Pension-scheme members and their representatives should do so, too. Indeed, so large are these liabilities that they are now a public-policy issue of considerable macro-economic importance. If shareholders and pension-scheme members are too sanguine to call for disclosure, then the public interest demands it be made mandatory.

It is now a fact that the largest defined-benefit pension schemes have liabilities greater than the largest annuity portfolio of any insurer. British Airways longevity liability is as large as Standard Life's. British Telecom's longevity liability dwarfs Prudential's (Richards, 2005). Some FTSE-100 companies now have pension liabilities which look rather large relative to their normal business activity. Indeed, some of them look less like ordinary businesses and much more like insurance companies. Perhaps it is time they were regulated as such?

## References

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