LIVING TO 100 SYMPOSIUM*

Lake Buena Vista, FL
January 7-9, 2008

Session 6A: Projection and Statistical Modeling of Mortality at Late Age Q&A

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JOSEPH LU: I want to say that I'm not an expert in Lee-Carter, so forgive me if this question is too obvious. I have a question on the changes in KT. What is the advantage of having a broken KT compared with my just choosing the data after 1975 and doing a Lee-Carter from then on? Is there any advantage of taking data from 1950 and then as it goes through the hassle of observing all these breaks and do them? If I just say okay forget all of these things, let's start from 1975 and do a Lee-Carter from there. Is there a difference in the approach? Do I get different results? Thank you.

JOHNNY LI: Can I answer the question? Well, that is actually another research question. Looking for an optimal period for fitting the Lee-Carter model is another research question that has been studied by some other researchers. But in my opinion, using data after 1970s, and using data from 1950 to now is somewhat different because in the model there are three parameter vectors. KT is only one of the three parameter vectors. If we can use more data, we can say, for example, given more reliable estimate of the variances of other parameters like AX and BX, which is not quite related to the timeframe of KT.

TIM HARRIS: What exactly happened in 1974 to 1975 to cause the shift in the projections of this model? It's like there is something happened biologically, or within society to cause a shift in the mortality patterns. Any thoughts on that?

JOHNNY LI: Yes, there must be something behind it. From the literature that I read, the reasons for this abrupt acceleration of mortality were not discussed but I suspect it may be due, for example, to cohort effects which we have not taken into account in this

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model. So I don't have a precise answer for it now, but cohort effect is one of the many possibilities.

TIM HARRIS: Ward, you had an answer?

WARD KINGKADE: In order to answer that, we have to introduce the factors that drive the reduction in mortality. Basically, there was a period in which mortality decline was sort of stagnating in many western countries and that's the point where you had a shift from death occurring due to infectious diseases and death occurring due to degenerative diseases. It took the medical community a long time to catch up to that, and to get the populous, the human community, to come along with them with some of their ideas on how to deal with cancer which was killing people like stopping smoking and changing your diet, changing your lifestyle and so on. Also it took pharmaceutical companies time to come up with things like ACE Inhibitors that leave your blood vessels supple, so that they don't increase hypertension and so forth. So it took some time for these changes, to the knowledge of what is taking place to yield its benefits. Ultimately, this sort of slowing down in the reduction of mortality ceased and mortality went on to decline at an increasing pace.

TIM HARRIS: Steven, you have a comment?

STEVEN HABERMAN: I wonder if I could just comment. Picking up what Johnny said. When we've looked at fitting Lee-Carter in England and Wales, we certainly did see a structural break in 1974-5 for males but not for females when we looked at the fitted KT values. But then when we fit a cohort model, we get a linear progression for KT for males and for females suggesting that there is a cohort component to that structural change. I mean that's not necessary to contradict what Ward said but I think in the U.K. there is a strong cohort effect that does come through in the 1970s.

WARD KINGKADE: And that might be cohort behavior in terms of things like smoking.

STEVEN MAKIN: What data did you use in your forecasting, Steven? Was it the CMI pensioner data set?

STEVEN HABERMAN: Yeah.

STEVEN MAKIN: I guess I was particularly struck by one comment on slide 19 when you looked the residuals by cohort or the residuals by period or by year of birth. I think you made a comment that there were no terribly significant cohort effects by looking at the residuals by year of birth. But it struck me when you made that comment that the shape of the residuals is almost like a rugby ball or a football. Part of it I suppose has to do with the volumes of data so you've not got many very old people in the data and not got many very young people. But that notwithstanding, it looks to me that thin at either end, thick in the middle and then thin at the other end, and I just wondered whether or not you know there really was a cohort effect in the U.K. in this data?

STEVEN HABERMAN: There may be. We'll go back and have another look. We interpreted it as being a feature of the dataset itself, but there is sparsity at the two ends. When we've compared these residual patterns to those where there is a strong cohort effect, one sees a cycling in the actual patterns of residuals. There's a very strong feature and that isn't present here or in any of the corresponding residual patterns that we've got. This was only one set. I mean there are obviously many of them. But we'll go back and have a look.

STEVEN MAKIN: The forecasting is based then probably on something like 20 years worth of data. Do you think that's adequate for 40, 50, 60 years forecasting?

STEVEN HABERMAN: No. I mean I think if you have 20 years data, you should really only go forward 10 years. So we've broken that rule and managed to project forward about 40 or 50 years, based on 20 years data.

JASON GOLDSTEIN: In the life insurance business we already have different splits of populations. We split smokers from non-smokers, and more detail we have we split the non-smokers into preferred, plus preferred and standard. Can you say anything to the trend in mortality reduction if you already split those groups out? Because obviously in aggregate if you put the smokers in there, then it seems that there will be more of a mortality reduction. But if you split them out, is there as much of a mortality reduction if you're just talking about the more healthy cohorts?

STEVEN HABERMAN: I mean that's difficult to answer. So, if I answer a parallel question which is if you compare different types of population, for example, if you take the national population of the U.K. and then you take those people who have life insurance policies, those who have annuities or those who have pensions and compare Lee-Carter for each of those, you find the KT values for annuitants and life insurance policyholders and people with pension plans, those KTs are falling much faster than for the national population. So one could infer from that, that the more select your group is, perhaps the more self-select it is, the more adverse selection there is. Perhaps the stronger the KT is, the stronger the downward trend. To actually answer your specific question, one would need to have the data on preferred lives.

JASON GOLDSTEIN: Is that something that's going to be looked into in the future?

STEVEN HABERMAN: We don't have enough data in the U.K. to do that. You can see from just the pension plan data, there is only 20 years worth of reliable data to use and so its use is questionable for long term projections.