

IUSSP Workshop on “The b-hypothesis and the modal age at death”

Rostock, Germany, 24-25 October 2011

Organized by the IUSSP Scientific Panel on Social and Biological Determinants of Longevity

Workshop Report

The IUSSP Scientific Panel on Social and Biological Determinants of Longevity organized a research workshop on “The b-hypothesis and the modal age at death”, with financial support from the Max Planck Institute for Demographic Research (MPIDR). The meeting took place on 24-25 October 2011 in Rostock, Germany, at MPIDR.

A group of 19 researchers, including demographers, bio-demographers, mathematical demographers, and biologists, were invited to discuss new ideas and findings and deep knowledge on the rate of ageing, the selective survival and compression of mortality. Death rates at all ages have been dramatically reduced over time, resulting in a remarkable rise in human life expectancy. The evidence suggests that this lifespan revolution has not been caused by a slowing of the ageing process. Demographers have shown that the rate of ageing has increased considerably over time. Furthermore, it has been shown that the transition from high to low mortality has been accompanied by rapid and massive compression. However, for recent years, empirical findings are mixed. Changes in the shape of the distribution of deaths reflect changes in the age pattern of mortality improvement, that is in the rate of ageing. Reduction in mortality rates at younger ages lead to mortality compression whereas those at older ages lead to mortality expansion. In a paper published by *Nature* in 2010, James Vaupel has hypothesized that all individuals share the rate of increase in the chance of death due to senescence. Thus, when controlling for selection effects and health improvements, the rate of ageing would be constant over time and across countries.

Eleven research papers were presented at the workshop. These papers included empirical studies on historical and contemporary populations as well as methodological papers. The first day of the workshop was devoted to research on the “b-hypothesis”, that is on the rate of ageing of the human population (b is usually used as symbol for the slope parameter of a Gompertz curve). Analyses of Swedish and Italian mortality data highlighted that the individual mortality curve is consistent with the Gompertz model only after age 80 and that women are generally more heterogeneous than men. Furthermore, it was shown that it is relevant to consider heterogeneity in individual frailty as a function changing over cohort. The “paradox” of an increasing rate of ageing while mortality declines is more evident for period data, less obvious for cohort data. However, when properly accounting for selection effects, differences in the life-table ageing rate between countries and over time are reduced for women but for men the picture is less clear. Significant attention was also given to important methodological issues arising from the most common assumptions of statistical models for the analysis of mortality. Besides the need to develop and apply methods to examine the “b hypothesis”, discussion highlighted the need to develop biological hypotheses to try to understand why the b-hypothesis could or couldn't be true. A biological hypothetical mechanism has been proposed by which the physiology actively regulated the rate of decline over time, without reference to age. The first day of the workshop ended with a presentation focusing on the impact of sudden changes in external conditions on the rate of ageing. Using events like internment in war camps or famine as examples of human shocks, some evidence

was found that the rate of ageing is unaffected by physical hardship and that the convergence of the mortality curve at old ages could be caused by the selection process.

The second day of the workshop, devoted to the modal age at death and mortality compression, opened with a discussion on the cause-specific life table ageing rate and the relations at the modal age at death. Mechanisms underlying the compression were investigated by decomposing the increase of b into effects of major causes of deaths, using cause-specific mortality data in France. The rises in all-cause b during 1979-1994 are attributable mainly to rises in cause-specific b 's for cardiovascular diseases as well as those for some pulmonary and digestive diseases, mental disorders and some cancers. These rises in cause-specific b occurred for both diseases at relatively high b levels (e.g. heart failure) and diseases at relatively low b levels (acute myocardial infarction). This seems to suggest that logit-mortality from many major diseases declined more at younger old ages (e.g. around 70) than at older old ages (e.g. around 90). The workshop continued with two methodological papers. The first presentation was about modelling changes in the distributions of deaths. Compositional data methods were applied to data from the Human Mortality Database and showed how deaths are reallocated by age as mortality falls, leading to changes in the mode and other statistics of the density function. The second paper focused on sensitivity analysis of longevity disparities. Several indices of inequality in longevity revealed similar patterns, suggesting that not only are the indices correlated, but so are their responses to changes in mortality schedules. The workshop ended with two empirical studies. An analysis of data from the Canadian Human Mortality Database revealed that provincial disparities in adult mortality in general and among the elderly population in particular are substantial in Canada. Moreover, based on the modal age at death and the standard deviation of ages at death above the mode, provincial disparities at older ages have barely reduced over time, despite the great mortality improvements in all provinces since the early 20th century. Lastly, a study was presented that investigated the lifespan variation by occupational class in Finland. This study demonstrates that continued mortality compression can be compatible with increases in life expectancy. This requires tackling the high early adult mortality of the lower socioeconomic groups, especially the high mortality from external causes.

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Max Planck Institute for Demographic Research
Rostock, October 24-25 2011

Programme

Monday, October 24

9.15-9.30 James Vaupel
Welcome address

9.30-13.00 Session 1 - Chair: Roland Rau

9.30-10.15 Elisabetta Barbi and Carlo G. Camarda
Rates of aging in human populations

10.15-11.00 Zhang Zhen
Cross-cohort differences in the heterogeneity of mortality

11.00-11.30 *Coffee break*

11.30-12.15 Trifon Missov
Modelling unobserved heterogeneity given observed mortality patterns at oldest-old ages

12.15-13.00 Daniel Levitis
Why might b not vary? A speculation

13.00 *Lunch*

14.30-17.00 Session 2 - Chair: Jean-Marie Robine

14.30-15.15 Virginia Zarulli
Mortality shocks and the human rate of aging

15.15-16.30 Discussion of research ideas and directions for further studies

19.00 *Dinner*

Tuesday, October 25

9.00-12.30 Session 3 - Chair: Troel Steenstrup

- 9.00-9.45 Vladimir Canudas-Romo
The cause-specific life table aging rate, LAR_i , and the relations at the modal age at death
- 9.45-10.30 Shiro Horiuchi, Karen Siu Lan Cheung, Jean-Marie Robine
Cause-of-death decomposition of old-age mortality compression
- 10.30-11.00 *Coffee break*
- 11.00-11.45 Jim Oeppen
Modelling and forecasting changes in the density of deaths in period life table
- 11.45-12.30 Hal Caswell
Sensitivity analysis of disparity in longevity
- 12.30 *Lunch*

13.45-16.30 Session 4 - Chair: James Vaupel

- 13.45-14.30 Robert Bourbeau and Nadine Ouellette
Regional disparities in Canadian adult and old-age mortality: A comparative study based on smoothed mortality ratio surfaces and age-at-death distributions
- 14.30-15.15 Alyson van Raalte
Lifespan variation by occupational class in Finland: Compression or stagnation over time?
- 15.15-16.15 Discussion of research ideas and directions for further studies
- 16.15-16.30 James Vaupel**
Concluding remarks

List of Participants:

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