Demographic Research Monographs

Elisabetta Barbi
John Bongaarts
James W. Vaupel
Editors

How Long Do We Live?

Demographic Models and Reflections on Tempo Effects

Springer

Demographic Research Monographs

A Series of the Max Planck Institute
for Demographic Research, Rostock, Germany

Editor-in-chief: James W. Vaupel

Demographic Research Monographs

E. Barbi, S. Bertino, E. Sonnino (Eds.)
Inverse Projection Techniques
2004. XI, 137 pages. 978-3-540-20931-7

G. Doblhammer
The Late Life Legacy of Very Early Life
2004. X, 204 pages. 978-3-540-22105-0

R. Rau
Seasonality in Human Mortality
2007. XV, 214 pages. 978-3-540-44900-3

A. Baudisch
Inevitable Aging?
2008. XXI, 170 pages. 978-3-540-76655-1

Elisabetta Barbi · John Bongaarts
James W. Vaupel
Editors

How Long Do We Live?

Demographic Models and Reflections on Tempo Effects

Springer

Professor Elisabetta Barbi
University of Messina
Department of Economics,
Statistics, Mathematics
and Sociology "W. Pareto"
Via Tommaso Cannizzaro 278
98100 Messina
Italy
ebarbi@unime.it

Professor James W. Vaupel
Max Planck Institute
for Demographic Research
Konrad-Zuse-Straße 1
18057 Rostock
Germany
jwv@demogr.mpg.de

Professor John Bongaarts
Population Council
1 Dag Hammarskjold Plaza
New York, NY 10017
USA
jbongaarts@popcouncil.org

Printed with the financial support of the Max Planck Society

ISBN 978-3-540-78519-4 e-ISBN 978-3-540-78520-0

DOI 10.1007/978-3-540-78520-0

Demographic Research Monographs ISSN 1613-5520

Library of Congress Control Number: 2008923550

Production: le-tex Jelonek, Schmidt & Vöckler GbR, Leipzig
Cover design: WMX Design GmbH, Heidelberg
The front cover photo – top right hand corner – includes a photograph by Harald Wenzel-Orf (www.wenzel-orf.de) and has been reproduced with his permission.

Printed on acid-free paper

9 8 7 6 5 4 3 2 1

springer.com

Foreword

How should life expectancy be calculated? More generally, how should life tables be estimated? Since John Graunt's pioneering contribution, read before the Royal Society of London at 6 p.m. on the 27th of February 1661, demographers have developed better and better methods. Some concerns were raised, including concerns about how to deal with heterogeneous populations published in an article in *Demography* in 1979 that I wrote with Kenneth Manton and Eric Stallard. Yet, a few years ago nearly all demographers believed that as long as the underlying population and death counts were accurate, then lifetables could be reliably estimated.

John Bongaarts and Griff Feeney launched a revolutionary assault on this dogma. Two key contributions by them are reprinted in Part I of this monograph. Some very good demographers agreed, as least in part, with Bongaarts' and Feeney's radical argument that when death rates are changing, then tempo effects distort conventional calculations of life expectancy. Other very good demographers disagreed. So John Bongaarts and I brought some leading demographers together in a research meeting, co-sponsored by the Max Planck Institute for Demographic Research and the Population Council and held in New York City on November 18 and 19, 2004. Many of the papers discussed at the workshop, generally after considerable revision, were published in *Demographic Research* in 2005 and 2006. Nine of these articles, in some cases somewhat revised, are published in this monograph: they are the first seven chapters in Part II and the two chapters in Part III. Some provide support for the importance of tempo effects; others raise doubts, either about the general concept or about methods proposed to remove the alleged distortions.

Five additional contributions are published in this monograph. The Introduction, by Elisabetta Barbi, provides an overview of the monograph-and thinking about tempo effects on mortality. The final chapter in Part II, by Marc Luy, is a considerably expanded version of a shorter article published in *Demographic Research*; it applies tempo methods to study the convergence of death rates in East vs. West Germany following unification in 1989. The two

chapters in Part IV are both new. In the first, John Bongaarts and Griff Feeney share their afterthoughts. In the second, I present four simple examples that demonstrate how and why mortality change can roil lifetable calculations. Finally, an Appendix by Jutta Gampe and Anatoli Yashin provides two proofs of a formula developed by Griff Feeney; this material was previously published in *Demographic Research*.

The chapters in this monograph are competently written, but nearly all of them are difficult to read. The material is complicated, controversial, and difficult to explain. I finally began to understand tempo effects, and the more general concept of turbulence in demographic rates, when I worked through some stylized examples. Some readers of this monograph might, therefore, want to start with my concluding chapter. Others may find it more satisfying to experience the developments of ideas by reading the book through from the beginning.

The series of Demographic Research Monographs is under the editorial supervision of the Max Planck Institute for Demographic Research. I am Editor-in-Chief. I am advised by an Editorial Board that currently consists of Prof. Elisabetta Barbi (Messina University, Italy), Prof. Gabriele Doblhammer (Rostock University, Germany), Dr. Jutta Gampe (Max Planck Institute), Prof. Joshua Goldstein (Max Planck Institute), and Prof. Bernard Jeune (University of Southern Denmark). Additional members of the Editorial Board will be appointed as needed to review manuscripts submitted for possible publication. The current manuscript was reviewed and accepted by James Vaupel, Elisabetta Barbi and Joshua Goldstein.The Editors thank Tobias Strauss for helping prepare the manuscript for publication.

The Demographic Research Monographs series can be considered the successor to the series called Odense Monographs on Population Aging, edited by Bernard Jeune and James Vaupel. The volumes in this now-terminated series were first published as hardcover books by an academic publisher, Odense University Press, and subsequently made available online at www.demogr.mpg.de/books/odense. The nine Odense Monographs on Population Aging include two collections of research articles that focus on specific subjects on the frontier of demographic research, three volumes by senior researchers that present path-breaking findings, a review of research on a topic of emerging interest, a presentation of a new method for analysis of demographic data, an outstanding doctoral dissertation, and a unique collection of important demographic data on non-human species.

The series of Demographic Research Monographs will continue this mix, with books that are often under 200 pages in length, that have a clear focus, and that significantly advance demographic knowledge. Research related to population aging will continue to be a focus on the series, but it will not the only one. The series will embrace all of demography, broadly defined. As indicated by the first volume in the series, an important subject will be historical demography. We also plan to publish research on fertility and family dynamics. Mathematical demography is the core of the population sciences

and we will strive to foster monographs, such as this one, that use mathematics and statistics to further develop the theories and methods of demography. Biodemography is a small but rapidly growing and particularly innovative branch of demography: we will seize opportunities to publish monographs at the intersection of biology and demography, pertaining both to humans and other species, and including demographic research with ties to such fields as epidemiology, genetics, evolutionary biology, life-history biology, experimental demography, and paleodemography. The previous monograph in the series, *Inevitable Aging*? by Annette Baudisch, combines mathematical demography and biodemography.

Each volume in the Demographic Research Monograph series will have a substantial link to the Max Planck Institute for Demographic Research. As well as being published as hardcover books by Springer-Verlag, the volumes of the Max Planck series of Demographic Research Monographs will subsequently be available at www.demogr.mpg.de/books/drm. The online version may include color graphs, supplemental analyses, databases and other ancillary or enhanced material. Parallel publication online and in print is a significant innovation that will make the monograph series particularly useful to scholars and students around the world.

James W. Vaupel
Editor-in-Chief

Contents

How long do we live? Demographic models and reflections on tempo effects: An introduction 1
Elisabetta Barbi
1 Background 1
2 Overview of the monograph 3
References 8

I. Theoretical basis for the mortality tempo effect

Estimating mean lifetime 11
John Bongaarts, Griffith Feeney
1 Methods 12
2 Results 15
3 Conclusion 23
References 24
Appendix A 25
Appendix B 26

The quantum and tempo of life-cycle events 29
John Bongaarts, Griffith Feeney
1 Introduction 29
2 Background: age-specific event rates 30
3 Period quantum and tempo measures of the 2nd kind 32
4 Period quantum and tempo measures of the 1st kind 46
5 Conclusion 56
References 58
Appendix A 62
Appendix B 63

II. Critiques, extensions and applications of the mortality tempo effect

Demographic translation and tempo effects: An accelerated failure time perspective 69
Germán Rodríguez
1 Introduction 69
2 Fertility 71
3 Mortality 79
4 Discussion 89
References 91

Lifesaving, lifetimes and lifetables 93
James W. Vaupel
1 Introduction 93
2 How saving a life alters life expectancy 95
3 Individual lifetimes 97
4 The triangle of turbulence 99
5 How large is the distortion? 100
6 Considerations about true life expectancy 101
7 A model of stretched lifetimes 103
8 Quantum and tempo vs. proportions and increments 103
9 Directions for research 104
References 107

Tempo and its tribulations 109
Kenneth W. Wachter
1 Tempo 109
2 Measures 111
3 Representation of M 113
4 The moving average 116
5 Period counts of deaths 119
6 Current latent conditions 120
7 Total fertility 123
References 127

Tempo effects in mortality: An appraisal 129
Michel Guillot
1 Introduction 129
2 The existence of tempo effects in mortality 131
3 Bongaarts and Feeney's tempo-adjusted life expectancy 134
4 Evaluating Bongaarts and Feeney's "proportionality" assumption . . . 135
5 Bongaarts and Feeney's definition of changes in period mortality conditions 139
6 Assessing indicators of period mortality conditions: $\mathbf{e_0}$ vs. *CAL* 144

7 Conclusion 149
References 150

Increments to life and mortality tempo 153
Griffith Feeney
1 Time-discrete increments to life 153
2 Empirical results: Swedish females, 1751-2002 154
3 Time-continuous cohort-indexed increments to life 155
4 Time-continuous period-indexed increments to life 158
5 Relation between cohort and period increments to life 159
6 Robustness of the Bongaarts-Feeney tempo adjustment formula 162
7 Increments to life and mortality tempo: mixed models 163
8 Conclusion 164
References 165

Mortality tempo versus removal of causes of mortality: Opposite views leading to different estimations of life expectancy 167
Hervé Le Bras
1 Introduction 167
2 Decreasing mortality as a sign of delay in deaths 168
3 Decreasing mortality as a change in the causes of death 170
4 Removing one cause of death: deeper insights 171
5 A numerical example of the two methods 172
6 Which life table is the reference table? 174
7 Unifying the two views: the repartition function of the delays by age and duration 176
8 Which is the best model? A discussion of the two methods 178
References 182
Appendix A 182
Appendix B 184
Appendix C 187

Tempo effect on age-specific death rates 191
Shiro Horiuchi
1 Introduction 191
2 Intuitive visual explanation 192
3 Mathematical presentation 195
4 Discussion 197
References 199
Appendix 200

Mortality tempo-adjustment: Theoretical considerations and an empirical application 203
Marc Luy
1 Introduction 203

2 How mortality tempo affects period life expectancy 206
3 Why life expectancy differences between western and eastern Germany call for tempo-adjustment 210
4 Trends in tempo-adjusted life expectancy in western and eastern Germany 215
5 Discussion 218
References 224
Appendix 229

III. Comparison of period and cohort measures of longevity

Five period measures of longevity 237
John Bongaarts
1 Introduction 237
2 Definitions of period longevity measures 238
3 Results 239
4 Discussion 241
5 Conclusion 244
References 244

Found in translation? A cohort perspective on tempo-adjusted life expectancy 247
Joshua R. Goldstein
1 Introduction 247
2 Proof of exact cohort translation 249
3 Discussion 251
4 Conclusion 257
References 258

IV. Conclusions

Afterthoughts on the mortality tempo effect 263
John Bongaarts, Griffith Feeney
1 Do tempo adjusted period longevity measures reflect current mortality conditions? 263
2 Conclusion 268
References 269

Turbulence in lifetables: Demonstration by four simple examples 271
James W. Vaupel
1 Introduction 271
2 Saving infant lives for one year 272
3 Saving infant lives for three years 274

4 Saving infant lives for two years on average 274
5 Saving everyone's life for one year 275
6 Discussion 278

Appendix

Two proofs of a recent formula by Griffith Feeney 283
Jutta Gampe, Anatoli Yashin
1 Introduction 283
2 Proof by Jutta Gampe 283
3 Proof by Anatoli Yashin 284
References 284

How long do we live? Demographic models and reflections on tempo effects: An introduction

Elisabetta Barbi

Department of Economics, Statistics, Mathematics and Sociology "W. Pareto", University of Messina, Via Tommaso Cannizzaro 278, 98100 Messina, Italy.
Email: ebarbi@unime.it

1 Background

The measurement of human longevity is one of the oldest topics in demography. The most widely used measure of longevity is the period life expectancy at birth which is calculated from age specific death rates by life table methods that originated with Graunt (1661) and have been standard in the field for well over a century. Period life expectancy equals the mean age at death in a synthetic cohort and it should be distinguished from the actual cohort life expectancies calculated for a group of individuals observed over long time periods.

A *tempo effect* is defined as an inflation or deflation of the period incidence of a demographic event (e.g., births, marriages, deaths) resulting from a rise or fall in the mean age at which the event occurs (Bongaarts and Feeney, in this volume p.11 and p.29). The existence of tempo effects has been well established in measures of fertility and nuptiality but the idea that mortality measures may be also affected is new and controversial.

Tempo effects were first discovered and analyzed in the study of fertility. If women shift the ages at which they bear children upward without changing their completed fertility, annual numbers of births will be less than they would have been because the same number of births will be spread out over a longer time period. Similarly, if women begin to have children at younger ages, annual numbers of births will be larger than they would have been because the same number of births occurs over a shorter time period. These changes in annual number of births induced by changes in the timing of childbearing are tempo effects. The post-war "baby boom" in the United States, for example, was due in part to a decline in the mean age at childbearing during the late 1940s and the 1950s (Ryder, 1964,1980) and in much of Europe recent period fertility levels are depressed by tempo effects resulting from the postponement of childbearing (Sobotka, 2004). Tempo effects complicate the study of

levels and trends of fertility because they produce changes in period fertility rates that depend on the rate at which the mean age at childbearing changes, independently of changes in completed fertility of cohorts. Ryder (1956) introduced the term "timing distortion" to refer to tempo effects in the total fertility rate because they are undesirable in most analyses of fertility levels and trends.

Ryder's pioneering work established the existence of tempo distortions in the total fertility rate, but he did not propose quantitative adjustments to remove tempo distortions. This may be explained in part by his strong emphasis on the conceptual priority of cohort fertility measures. Bongaarts and Feeney (1998) first proposed to remove tempo distortion from the period total fertility rate. Their tempo adjustment is obtained by dividing the observed total fertility rate by $1 - r$, where r equals the annual change in the period mean age at birth. Recent applications of this method to obtain tempo adjusted fertility levels by birth order in many European countries are presented in Sobotka (2003, 2004). The same method can be used to remove tempo distortions in period nuptiality measures as demonstrated by Winkler-Dworak and Engelhardt (2004).

Bongaarts and Feeney (2002, in this volume p.11 and p.29) noted that tempo effects affect the numerators of all period event rates. As a result, tempo effects inflate and deflate not only incidence rates such as conventional age specific birth or marriage rates (rates of the second kind) but also occurrence/exposure rates (rates of the first kind). Kohler and Ortega (2002,a,b) propose procedures for calculating period fertility measures based on tempo adjusted occurrence/exposure rates and applications of this method are found in Sobotka (2003) and Winkler-Dworak and Engelhardt (2004).

The possibility that period life expectancy contains tempo effects was proposed and studied by Bongaarts and Feeney (2002, in this volume p.11 and p.29). They reasoned that if occurrence/exposure rates for births and marriages contain tempo effects then the same should be true for occurrence/exposure rates of other events such as deaths. Period life-expectancy derived from these rates therefore should contain tempo effects as well when the mean age at death changes. Their studies also propose an adjustment to remove the tempo effect which is conceptually similar to the adjustments made in fertility and nuptiality measures.

Tempo adjusted period measures of fertility, nuptiality and mortality should be interpreted as variants of their conventional counterparts. The life expectancy at birth, for example, is defined as the average age at death of a newborn subjected throughout life to the age-specific death rates observed in a given year. This is a hypothetical lifespan because no actual cohort will experience these observed period death rates. According to Bongaarts and Feeney, the tempo adjusted life expectancy is a similar hypothetical measure, but one that corrects for distortions caused by year to year tempo changes. Neither the observed nor adjusted life expectancy attempts to estimate the mean age at death of any actual cohort, nor do they attempt any prediction

of future mortality. The goal of the tempo adjustment is simply to provide period quantum and tempo measures that are free of the tempo distortions.

This volume examines the question whether period life expectancy as calculated by a conventional life table is affected by tempo effects. The interest in the mortality tempo effect has grown recently and has generated an animate debate among scientists involved in mortality research. Some scholars remain unpersuaded about the existence of the effect in mortality but some skeptics have begun to revise their views about the significance of the effect.

In order to promote further research on this important but controversial issue, the Max Planck Institute for Demographic Research (Rostock, Germany), in collaboration with the Population Council (New York, USA), organized an international workshop on 18-19 November 2004 in New York.

The workshop produced a number of high-quality papers. Many of these papers were then revised and submitted for publication to the journal *Demographic Research* and underwent the usual process of peer review. The present volume collects a selection of these articles which have been already published in *Demographic Research* during 2005 and 2006. Furthermore, the volume includes other important studies on the mortality tempo effect which were not presented at the workshop in New York.

2 Overview of the monograph

This collection includes fourteen chapters grouped into four sections, plus an Appendix. Two chapters by John Bongaarts and Griffith Feeney in the first group present the background and the theoretical framework for the mortality tempo effect. The first one, a pioneering work in the field, was published in the *Proceedings of the National Academy of Sciences* in 2003. It shows that observed death rates and period life expectancy as conventionally estimated are distorted whenever mortality is changing and lead to a misleading indication of current mortality conditions. The authors propose an alternative period measure of longevity adjusted for tempo changes. The new measure is based on the assumption that the observed force of mortality at a given time t is proportional to an age intensity defined as the rate at which the proportion of cohort survivors in a population at time t varies from one age to the next. It is shown that this assumption implies uniform delays of death to older (younger) ages as mortality declines (increases). The authors demonstrate that the so-called proportionality assumption is realistic in populations with high life expectancy and when ignoring child and young adult mortality. In the second chapter, published in the *Vienna Yearbook of Population Research* in 2006, the authors extend their previous studies and demonstrate the existence of tempo distortions in period quantum and tempo measures of a wide range of life cycle events. This chapter makes the connection between the fertility and mortality tempo effects and gives a more general framework

for the analysis of the tempo effect in demographic events, with empirical examples for fertility, marriage and mortality.

The second section of the volume, consisting of eight chapters, is devoted to critiques, extensions and applications of the mortality tempo effect. The leitmotif of the first four chapters is the definition and the interpretation of longevity measures for a better understanding of the complexity of mortality dynamics.

German Rodriguez analytically reviews the concept of tempo effect in demography. He analyzes the cohort implications of the Bongaarts-Feeney delay-death model and shows that this is closely linked to accelerated failure time models used in survival analysis. The author emphasizes important similarities as well as fundamental differences between the analysis of fertility and mortality. He argues that in the case of fertility, the adjustments help to distinguish changes in the quantum or tempo. This is not the case for mortality which is a pure tempo phenomenon. When cohorts start delaying death, observed mortality rates decline. Conventional life expectancy reacts instantly, whereas the Bongaarts-Feeney tempo adjusted life expectancy reacts more slowly. According to the author, there is no bias or distortion in the observed force of mortality. The two indicators - the conventional and the tempo-adjusted life expectancy - simply measure different things. Conventional life expectancy depends only on the force of mortality, whereas, the adjusted measure is affected by the age composition of cohort survivors and, thus, reflects past rather than current mortality. So, the conventional life expectancy tells us how long today's newborns will live under the current rates, whereas the tempo-adjusted life expectancy tells us how long those dying today have lived under the proportionality assumption.

James Vaupel argues that life expectancy under current rates and life expectancy under current conditions are different under a broad variety of circumstances. In particular, when mortality is changing, calculations of period life expectancy do not, except in special circumstances, measure the life expectancy of a cohort of newborns that hypothetically live all their lives under the current mortality regime, as argued by Bongaarts and Feeney. The Bongaarts-Feeney delay-death model as well as various models accounting for population heterogeneity in individual frailty are considered special cases. The author then presents a model of stretched lifetimes based on the idea that deaths that would have occurred over some period of time occur over a longer period of time after a mortality improvement. Neither the conventional life table approach nor the delay-death model nor the stretched lifetimes model account for population heterogeneity. Vaupel concludes that the tempo-quantum metaphor may not be optimal and considers the issue of selective survival a better starting point as mortality changes may affect individuals differently. Instead of the narrower term "tempo distortion in mortality" he suggests to use a broader "theory of mortality turbulence" to allude to the general phenomenon that when mortality is changing conventional lifetables do not describe the cohort mortality experience under current conditions.

In the following chapter, Kenneth Wachter focuses on the understanding of what the mortality tempo adjusted measures do measure. He shows that, under the proportionality assumption, the Bongaarts-Feeney measure is an exponentially weighted moving average of period life expectancies from recent past. This dependence on the past is the fundamental property of the mortality adjusted measure. In contrast, the conventional life expectancy is an indicator of current observed mortality. The fact that it is sensitive to sudden mortality changes is, according to Wachter, an advantage, not a drawback. The author concludes that adjustments for tempo do not make obvious sense in mortality but only when there is a distinction between quantum and tempo in individual experience as in the case of fertility.

The existence of tempo effect in mortality is critically examined by Michel Guillot. He concludes that the Bongaarts-Feeney indicator can indeed be considered a period measure under specific assumptions. But he argues that the proportionality assumption is met only if one disregards mortality under age 30 and that this additional assumption may not be appropriate even in contemporary populations. Furthermore, the author asserts that Bongaarts and Feeney's index relies on a particular definition of changes in period mortality conditions which implies, as a result of mortality changes, delays in future cohort deaths that are cohort-constant. Thus, for instance, the fact that the amount of benefits (delays in age at death) of a medical innovation depends on how long before the innovation appeared is not considered in Bongaarts and Feeney's approach. Guillot concludes that until our knowledge of mortality dynamics is better developed, it is preferable to use the conventional life expectancy as an indicator of current mortality conditions.

One of the assumptions in the Bongaarts-Feeney's approach is that delays of death are age-independent. This issue is addressed in the following chapter by Griffith Feeney who introduces the idea of "increments to life" as a complementary perspective to the force of mortality in the study of changing mortality and length of life. The author develops a general mathematical representation of life gains allowing for continuous variation in age and time which provide a method for assessing the robustness of the Bongaarts-Feeney mortality tempo adjustment formula. Furthermore, the formulation of age-variable increments to life is useful to avoid a restrictive assumption of the Bongaarts-Feeney approach, that is the assumption of ignoring mortality changes in infancy, childhood and young adult ages. Linked to this chapter is an appendix by Jutta Gampe and Anatoli Yashin who provide two different proofs for the first formula appearing in Feeney's chapter. This formula, for which the author did not give a mathematical proof, decomposes the difference between the expectations of life at birth for two cohorts in terms of the increments to life values.

An alternative way of dealing with mortality tempo is devised by Hervé Le Bras. The author proposes a model where mortality changes take place with the removal of a given cause of death. This may produce a delay in death but, contrary to Bongaarts-Feeney delay-death model, one that de-

pends strongly on age. In the removal model, there is no discrepancy between cross-sectional and longitudinal indexes, period measures of longevity are not distorted and, thus, no correction is needed. The author claims that the proposed method is more general and better suited to the true nature of mortality processes. He concludes that the removal method should be preferred to the delay method.

The following two chapters support enthusiastically the idea of tempo effect in mortality. Shiro Horiuchi investigates the effects of changes in the age distribution of cohort deaths on the age-specific number of period deaths and, in turn, on the age-specific period death rate, under the assumption that the age-specific number of deaths is constant among cohorts but allowing for non parallel shifts in the age distribution. He first gives an intuitive and visually oriented demonstration that a tempo distortion can in effect occur in age-specific mortality. Then he provides a mathematical representation of the mechanism. However, Horiuchi recognises that the study presented clarifies the logical mechanism of only one of all possible pathways through which mortality changes can affect period measures.

Finally, Marc Luy also has no doubt about the existence of tempo effects in period life expectancy and the distortions they may cause (this chapter does not spring from the workshop in New York). A shorter version of this chapter was recently published in *Demographic Research*. Here we reproduce an extended version including an extension of an example provided by Feeney (2003) in his unpublished paper "Mortality tempo: a guide for the skeptic". Luy presents an application of the Bongaarts-Feeney method to the analysis of mortality differences between western and eastern Germany. The results from tempo-adjusted life expectancy provide a better fit than those from the conventional life expectancy to the expected trends of changing mortality in Germany. As a consequence, the author claims that the adjusted life expectancy is a more realistic indicator of the level and changes in current mortality conditions than the conventional life expectancy. Luy concludes that, although the Bongaarts-Feeney adjusted measure can be improved since it is based on strong assumptions, their approach should be preferred as long as there are no better solutions.

The third part of the volume includes two chapters focusing on the comparison of period and cohort measures of longevity. John Bongaarts summarizes five recently proposed period measures of longevity and shows that three of the five measures are identical to one another under the assumption that mortality follows a Gompertz model with a constant rate of improvement over time. These measures, however, differ substantially from the conventional period life expectancy when mortality changes over time. The author notes that these empirical findings are consistent with the theoretical analysis by Bongaarts and Feeney which showed that the deviation of conventional life expectancy from the other longevity measures is caused by a tempo effect whose size varies with the rate of change in mortality.

The following chapter by Joshua Goldstein shows that, under the Bongaarts - Feeney's assumption of uniform postponement of death across all ages, the additional assumption of linear mortality shift, and ignoring mortality below age 30, the tempo-adjusted life expectancy for a given year t, $e_0^*(t)$, is equal to the life expectancy of the cohort dying in that year t, that is the cohort born $e_0^*(t)$ years earlier. Accordingly, Bongaarts-Feeney period longevity measure corrected for tempo distortion may be seen also as a measure of cohort life expectancy. The author concludes that, in case of sudden mortality change, the tempo adjustment is useful for understanding the implications of mortality rates during shocks. However, in recent years, almost all the developed countries have experienced a steady mortality decline, a situation in which the cohort interpretation gives more valuable sense.

Two chapters in the final section of the volume summarize the discussion about the existence and the meaning of the mortality tempo effect. In their concluding note Bongaarts and Feeney comment briefly on the main question that has been raised by some chapters in the volume about their analysis of the tempo effect and their proposal to remove this effect by adjusting the conventionally calculated life expectancy. This question is whether the tempo adjusted life expectancy is a current measure of mortality conditions as they and Vaupel and Guillot believe, or a measure of the past mortality as suggested by Rodriguez and Wachter. The authors also discuss the assumptions underlying their tempo adjustment and argue that these assumptions hold for senescent mortality which dominates in contemporary low mortality countries.

The issue of distortion in period death rates and life expectancy occurring whenever mortality is changing is complicated and difficult to explain. For this reason, James Vaupel presents four simple examples which clearly show how lifesaving can roil lifetable statistics. He concludes that the question about the existence of tempo effects in mortality is open but there is no doubt that mortality change produces turbulence in lifetables. However, how much life is extended when a death is averted is a question that needs further research.

What is then the "true life expectancy"? There is no doubt that the conventional period life expectancy is not an accurate measure of longevity of people born in or living in a given year. However, the debate on how best to measure period longevity, the existence of tempo distortion in mortality and the need of adjustments in longevity measures is still open. Nevertheless, I believe that this set of insightful studies makes an important step toward a deeper understanding of the population dynamics and the tune of valuable longevity measures, and hope that it will stimulate further extensive research in the field.

References

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. The quantum and tempo of life cycle events. *In this volume, also published in Vienna Yearbook of Population Research*, pages 115–151. 2006.

Bongaarts, J. and Feeney, G. (1998). On the quantum and tempo of fertility. *Population and Development Review*, 24(2):271–291.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Feeney, G. (2003). Mortality tempo: a guide for the skeptic. to be downloaded from http://www.gfeeney.com. unpublished manuscript.

Kohler, H. P. and Ortega, J. A. (2002a). Tempo-adjusted period parity progression measures, fertility postponement and completed cohort fertility. *Demographic Research*, 6:92–144. www.demographic-research.org.

Kohler, H. P. and Ortega, J. A. (2002b). Tempo-adjusted period parity progression measures: Assessing the implications of delayed childbearing for cohort fertility in Sweden, the Netherlands and Spain. *Demographic Research*, 6:146–190. www.demographic-research.org.

Ryder, N. B. (1956). Problems of trend determination during a transition in fertility. *Milbank Memorial Fund Quarterly*, 34:5–21.

Ryder, N. B. (1959). An appraisal of fertility trends in the United States. In *Thirty Years of Research in Human Fertility: Retrospect and Prospect*. 38-49. Milbank Memorial Fund, New York.

Ryder, N. B. (1964). The process of demographic translation. *Demography*, 1:74–82.

Ryder, N. B. (1980). Components of temporal variations in American fertility. In Hiorns, R. W., editor, *Demographic Patterns in Developed Societies*, pages 15–54. Taylor & Francis, London.

Sobotka, T. (2003). Tempo-quantum and period-cohort interplay in fertility changes in Europe. Evidence from the Chech Republic, Italy, the Netherlands and Sweden. *Demographic Research*, 8:152–214.

Sobotka, T. (2004). Is Lowest-Low Fertility in Europe Explained by the Postponement of Childbearing? *Population and Development Review*, 30(2):195–220.

Winkler-Dworak, M. and Engelhardt, H. (2004). On the tempo and quantum of first marriages in Austria, Germany, and Switzerland. *Demographic Research*, 10(9):231–263.

I. Theoretical basis for the mortality tempo effect

Estimating mean lifetime*

John Bongaarts[1] and Griffith Feeney[2]

[1] Population Council, 1 Dag Hammarskjold Plaza, New York, NY 10017, USA. E-Mail: jbongaarts@popcouncil.org
[2] 9 Fairview Road, Scarsdale, NY 10583, USA. E-Mail: feeney@gfeeney.com

Summary. The life expectancy implied by current age-specific mortality rates is calculated with life table methods that are among the oldest and most fundamental tools of demography. We demonstrate that these conventional estimates of period life expectancy are affected by an undesirable "tempo effect." The tempo effect is positive when the mean age at death is rising and negative when the mean is declining. Estimates of the effect for females in three countries with high and rising life expectancy range from 1.6 yr in the U.S. and Sweden to 2.4 yr in France for the period 1980-1995.

When a group of persons is observed from birth to death, mean lifetime may be calculated simply and directly as mean age at death. This statistic is problematic, however, for studying trends in mean lifetime. Mean lifetime for Swedish females born in 1850, for example, reflects mortality conditions from the mid-19th to the mid-20th centuries, a period of historically unprecedented increases in human survival. The study of these changes requires a different approach.

Period life expectancy at birth calculated by life table methods has been the standard solution to this problem since the mid-19th century (Preston, Heuveline and Guillot, 2001). This chapter argues that it is an imperfect solution, because life expectancy at birth calculated in this way is distorted whenever it is changing.

Conventional life expectancy depends solely on the force of mortality function for time t. We propose an alternative measure that depends both on the force of mortality function and on the rate of change in the standardized mean age at death. Our alternative is based on the assumption that the observed force of mortality function at any given time has the same shape as the force of mortality function inherent in the standardized population age distribution at time t, which reflects the history of mortality in the population. We

demonstrate that this assumption is realistic in contemporary societies with high life expectancy and also that the proposed measure is consistent with well-established measures used in other demographic contexts.

1 Methods

1.1 Cohort mean lifetime

The distribution of lifetimes for a group of persons born during any given time period (a "birth cohort") may be described in three different ways. The survival function,

$$l(a)\,, \quad a \geq 0\,, \tag{1a}$$

gives the proportion of individuals who survive to exact age a. It is nonincreasing, with $l(0) = 1.0$ and $l(\omega) = 0$ for some advanced age ω. The death density function,

$$d(a) \equiv \frac{-\partial l(a)}{\partial a}\,, \tag{1b}$$

gives the distribution of deaths by age. The force of mortality function,

$$\mu(a) = \frac{d(a)}{l(a)} = \frac{-\partial l(a)/\partial a}{l(a)} \tag{1c}$$

gives the risk of dying at each age. These functions are formally equivalent in the sense that any two may be derived from the third. The force of mortality function $\mu(a)$ may be derived from $d(a)$ or $l(a)$ by using Eq. (1c), for example, and $l(a)$ may be derived from $\mu(a)$ or $d(a)$ by using

$$l(a) = \int_a^\omega d(x)\,dx = \exp\left[-\int_0^a \mu(x)\,dx\right]\,. \tag{1d}$$

Fig. 1 plots $l(a)$, $d(a)$, and $\mu(a)$ for the cohort of females born in Sweden in 1850. The survival function declines to zero at around age 100 yr. The density function is broadly bimodal with peaks at age 0 and $\approx$ 80 yr. The force of mortality exhibits a U-shaped pattern with a minimum at about age 10. Note the use of the log scale to accommodate the large differences in magnitude at different ages. These patterns are broadly typical, although levels of mortality vary widely between populations and over time.

Mean lifetime for a birth cohort, M, may be calculated from $l(a)$ as

$$\int_0^\infty l(a)\,d(a)\,, \tag{2a}$$

from $d(a)$ as

$$\int_0^\infty a d(a)\,da\,, \tag{2b}$$

or from $\mu(a)$ as

$$\int_0^\infty \left\{ \exp\left[-\int_0^a \mu(x)\, dx \right] \right\} da \tag{2c}$$

These formulas give identical results. For the 1850 cohort of Swedish females, for example, we calculate $M = 48.1yr$ from each.

1.2 Period mean lifetime

Let

$$l(a,t) \equiv l_{t-a}(a)\,, \tag{3a}$$
$$d(a,t) \equiv d_{t-a}(a)\,, \text{ and} \tag{3b}$$
$$\mu(a,t) \equiv \mu_{t-a}(a)\,, \tag{3c}$$

where the subscripts at right indicate time of birth. Thus $l(a,t)$ denotes the proportion of persons born at time $t-a$ who are surviving at time t; $d(a,t)$ denotes the density of deaths for this cohort at age a and time t; and $\mu(a,t)$ denotes the corresponding force of mortality. Note that $l(a,t)$ and $d(a,t)$ differ from the survival and density functions for synthetic cohorts obtained from conventional period life tables, and that their calculation requires data on either past births and migrations or on past deaths.

We refer to $l(a,t)$ as the standardized population age distribution at time t and to $d(a,t)$ as the standardized age distribution of deaths at time t. The standardized population age distribution and age distribution of deaths are the same as their unstandardized counterparts in any population that experiences constant numbers of births over time.

By analogy with Eq. (2), mean lifetime at time t may be calculated as

$$M_1(t) = \int_0^\infty l(a,t)\, da\,, \text{ as} \tag{4a}$$
$$M_2(t) = \frac{\int_0^\infty a d(a,t)\, da}{\int_0^\infty d(a,t)\, da}\,, \text{ or as} \tag{4b}$$
$$M_1(t) = \int_0^\infty \exp\left[-\int_0^a \mu(x,t)\, dx \right] da\,. \tag{4c}$$

Each of these formulas has been used in demography to calculate period mean age for some demographic event. Mean age at first marriage is often calculated as a variant of $M_1(t)$ that allows for persons not marrying. This is the singulate mean age at marriage introduced by Hajnal (1953), with $l(a,t)$ taken as the proportion of single persons at age a at time t (see, for example, ref. United Nations (1990)). Mean age at childbearing is generally calculated as $M_2(t)$, with agespecific or age-order-specific birth rates substituted for $d(a,t)$ (see,

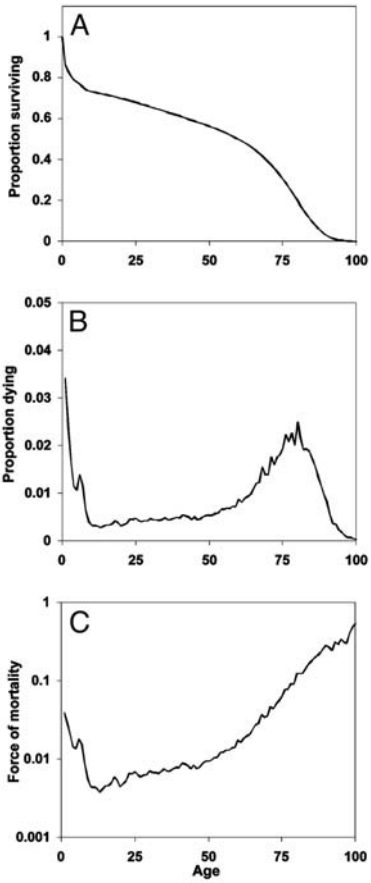


Fig. 1. Mortality experience of the cohort of Swedish females born in 1850, as summarized by the survival function, $l(a)$ (A), the death density function $d(a)$ (B), and the force of mortality function $\mu(a)$ (C).

for example, ref. Council of Europe (2001)). Life expectancy at birth, denoted $e_0(t)$, is conventionally calculated as $M_3(t)$.

We refer to $M_2(t)$ as the standardized mean age at death. The unstandardized mean age at death is unacceptable as a measure of mean lifetime, because it may be heavily distorted by the population age distribution. This objection does not apply to the standardized mean age at death, which might be a widely used measure of period mean lifetime if it were more easily calculated.

If $l(a,t)$ is constant with respect to t, the three means defined by Eq. (4) are identical. When length of life changes, the three means diverge. The following sections develop relationships among them.

2 Results

2.1 Relation between M_1 and M_2

To establish a simple relationship between $M_1(t)$ and $M_2(t)$, let

$$d_s(a,t) = \frac{-\partial l(a,t)}{\partial a} \quad \text{and} \quad \mu_s(a,t)\frac{d_s(a,t)}{l(a,t)} \,. \tag{5a,b}$$

The age schedules $d_s(a,t)$ and $\mu_s(a,t)$ are inherent in the standardized population age distribution at time t. They may be interpreted as the age distribution of deaths and the force of mortality function in the stationary population whose age distribution is given by $l(a,t)$, with $l(0,t) = 1$ for all t. This interpretation is, of course, valid only if the mortality history of the population is such that $l(a,t)$ is a nonincreasing function of a $(dl(a,t)/da \leq 0)$.

Assume now that for t in the time interval $[0, \Delta]$, there exists a function $p(t)$ independent of age, such that

$$\mu(a,t) = p(t)\mu_s(a,t) \text{ or, equivalently,} \tag{6a}$$

$$d(a,t) = p(t)d_s(a,t) \,, \tag{6b}$$

and that the function $p(t)$ is a real valued integrable function bounded below by 0. We refer to this as the proportionality assumption.

The proportionality assumption implies that the age schedules of $\mu(a,t)$ and $d(a,t)$ are the same in shape (but not necessarily level) as the age schedules of $\mu_s(a,t)$ and $d_s(a,t)$. As will be shown below, this assumption provides a good approximation for patterns of adult mortality in contemporary countries with high life expectancy.

From Eqs. (4a) and (5a),

$$M_1 = \int_0^\infty l(a,t)\, da = \frac{\int_0^\infty a d_s(a,t)\, da}{\int_0^\infty d_s(a,t)\, da} \tag{7a}$$

and from Eq. (4b) and (6b),

$$M_2 = \frac{\int_0^\infty a p(t) d_s(a,t)\, da}{\int_0^\infty p(t) d_s(a,t)\, da} \tag{7a}$$

On cancellation of the proportionality factor p(t), Eq. (7b) becomes Eq. (7a), thus proving that $M_1(t) = M_2(t)$.

2.2 Other implications of the proportionality assumption

It is shown in Appendix A that if the proportionality assumption holds, then

$$p(t) = 1 - \frac{\partial M_1(t)}{\partial t} \,. \tag{8a}$$

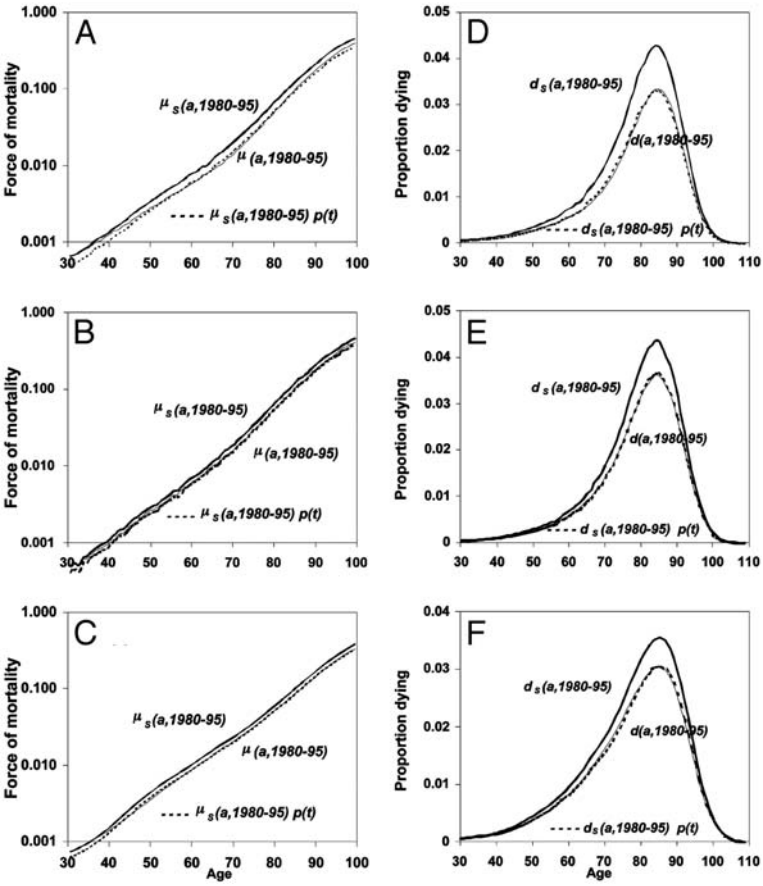


Fig. 2. Average force of mortality for 1980-1995, observed as $\mu(a,t)$, estimated from $l(a,t)$ as $\mu_s(a,t)$, and estimated as the product $\mu_s(a, 1980-1995)p(t)$ for France (A), Sweden (B), and the U.S. (C). Also shown is the average death density function for 1980-1995, observed as $d(a,t)$, estimated from $l(a,t)$ as $d_s(a,t)$, and estimated as the product $d_s(a, 1980-1995)p(t)$ for France (D), Sweden (E), and the U.S. (F).

Substituting this in Eq. (6) and noting that $M_1(t) = M2(t)$ yields

$$\mu(a,t) = \left[1 - \frac{\partial M_2(t)}{\partial t}\right] \mu_s(a,t)\,, \tag{8b}$$

$$d(a,t) = \left[1 - \frac{\partial M_2(t)}{\partial t}\right] d_s(a,t)\,. \tag{8c}$$

This shows that $\mu(a,t)$ and $d(a,t)$ are functions of the rate of change in the standardized mean age at death $M_2(t)$, because $\mu_s(a,t)$ and $d_s(a,t)$ are determined by mortality conditions up to time t. When this mean age is rising,

$\mu(a,t) < \mu_s(a,t)$ and $d(a,t) < d_s(a,t)$, but when it is declining, $\mu(a,t) > \mu_s(a,t)$ and $d(a,t) > d_s(a,t)$.

As shown in Appendix B, the proportionality assumption also implies that the age schedule $l(a,t)$ shifts uniformly to older (younger) ages as the mean age at death rises (falls). Uniform shifting between time 0 and time T means that there is a function $F(t) = M_1(t) - M_1(0)$, giving the magnitude of the shift between time 0 and time t, such that, for all $0 \le t \le T$,

$$l(a,t) = l(a - F(t), 0) \quad \forall a \ge F(t)\,, \tag{9}$$

and $l(a,t) = 1$ for $a \le F(t)$. Downward as well as upward shifts are possible, provided that $l(a,t) = 1$ for a less than some number > 0.

It follows from Eq. (5) that uniform shifts in $l(a,t)$ imply uniform shifts in $\mu_s(a,t)$ and $d_s(a,t)$ with the same shift function $F(t)$, with $\mu_s(a,t) = d_s(a,t) = 0$ when $l(a,t) = 1$. The proportionality assumption is therefore equivalent to the shifting assumption made by Bongaarts and Feeney (2002).

Changes over time in the schedules $\mu(a,t)$ and $d(a,t)$ are of two types. First, as the mean age at death rises or falls, $\mu(a,t)$ and $d(a,t)$ shift to higher or lower ages with $l(a,t)$, $\mu_s(a,t)$, and $d_s(a,t)$. Second, $\mu(a,t)$ and $d(a,t)$ are deflated or inflated relative to $\mu_s(a,t)$ and $d_s(a,t)$ by the proportionality factor $p(t)$.

2.3 Mortality change in France, Sweden, and the U.S.

We will now show that observed mortality patterns conform closely to the proportionality assumption (Eq. (6)) if child and young adult mortality is ignored. All quantities in this section, in Figs. 2-6, and in Table 1 are calculated from observed values of $\mu(a,t)$ for ages > 30, but $\mu(a,t)$ is set to zero for ages < 30 years for all t. Our estimates of life expectancy at birth are therefore equal to 30 plus the life expectancy at age 30. For populations with high life expectancy, nearly all deaths (97-98%) occur at ages > 30 yr, and actual life expectancy at birth is therefore close to 30 plus the life expectancy at age 30.

Table 1. Alternative estimates of the period mean age at death (assuming no mortality under age 30).

	Mean age at death, females, 1980 – 1995				
	$M_1(t)$	$M_2(t)$	$M_3(t)$ $(= e_0(t))$	$M_4(t)$	Tempo effect $M_3(t) - M_4(t)$
France	79.0	79.2	81.4	79.0	2.4
Sweden	79.5	79.5	81.1	79.4	1.6
U.S.	78.3	78.3	79.9	78.3	1.6

Fig. 2 A-C shows the age schedules, $\mu(a,t)$, $\mu_s(a,t)$, and $p(t)\mu_s(a,t)$, all calculated as averages of annual values for 1980-1995, for France, Sweden, and

the U.S. Fig. 2 D-F shows the age schedules $d(a,t)$, $d_s(a,t)$, and $p(t)d_s(a,t)$ calculated in the same way with $p(t)$ estimated with Eq. (8a). The near coincidence of $\mu(a,t)$ and $p(t)\mu_s(a,t)$ and of $d(a,t)$ and $p(t)d_s(a,t)$ shows that the proportionality assumption is a good approximation for all three countries. Note that the logarithmic scale used in Fig. 2 A-C means that perfect proportionality corresponds to constant differences between the plotted values of $\mu(a,t)$ and $\mu_s(a,t)$.

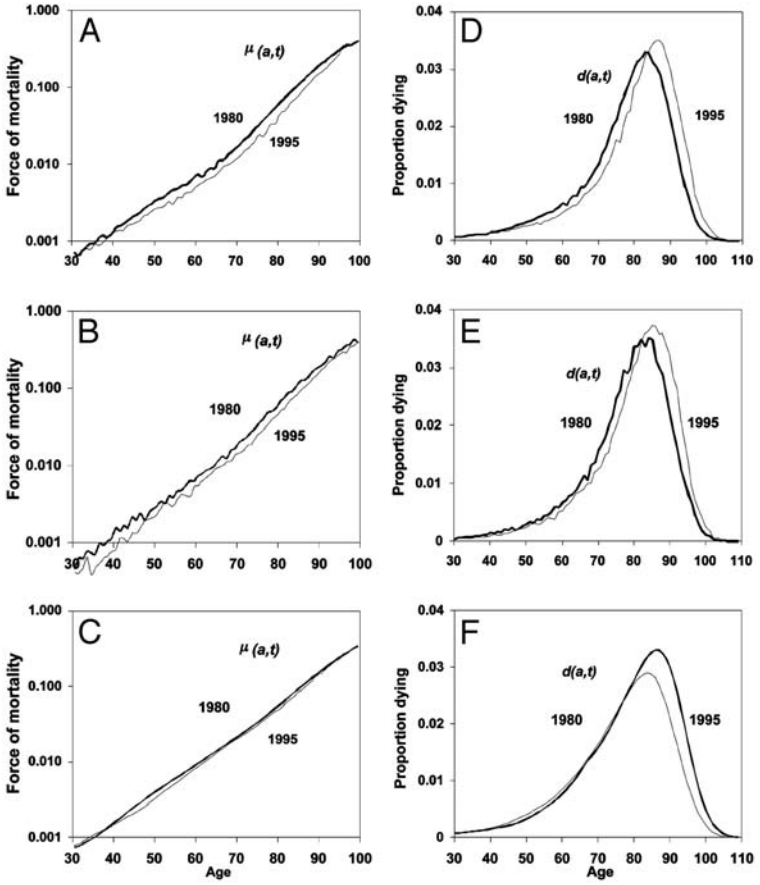


Fig. 3. Observed period force of mortality $\mu(a,t)$ in 1980 and 1995 for France (A), Sweden (B), and the U.S. (C). Also shown is the observed period death density function $d(a,t)$ in 1980 and 1995 for France (D), Sweden (E), and the U.S. (F).

Fig. 3 A-C shows $\mu(a,t)$ for 1980 and 1995 for the same three countries. Fig. 3 D-F shows corresponding values for $d(a,t)$. The pattern of change in

these schedules is consistent with the pattern of shifting and inflation/deflation noted above.

Fig. 4 plots the age schedule $l(a,t)$ for 1980 and 1995 for the three countries. The shape of $l(a,t)$ changes very little, but there is a shift to higher ages as life expectancy rises. The magnitude of the shift was 3.4 yr for France, 2.4 yr for Sweden, and 2.1 yr for the U.S.

The first three columns of Table 1 present averages of annual estimates of $M_1(t)$, $M_2(t)$, and $M_3(t)$ for the years 1980-1995. The values for $M_1(t)$ and $M_2(t)$ are nearly identical, as expected, but the $M_3(t)$ values are substantially higher. The reason for the higher value of $M_3(t)$ is discussed below.

2.4 Tempo effects in demographic analysis

Tempo effects were first discovered and analyzed in the study of fertility. If women shift the ages at which they bear children upward without changing their completed fertility, annual numbers of births will be less than they would have been, because the same number of births will be spread out over a longer time period. Similarly, if women begin to have children at younger ages, annual numbers of births will be larger than they would have been, because the same number of births occurs over a shorter time period. These changes in annual number of births induced by changes in the timing of childbearing are tempo effects.

Fertility tempo effects have been extensively documented. The postwar "baby boom" in the U.S., for example, was due in part to a decline in the mean age at childbearing during the late 1940s and the 1950s (Hajnal, 1947, Ryder, 1964, 1980, Bongaarts and Feeney, 1998).

Tempo effects complicate the study of levels and trends of fertility, because they produce changes in period fertility rates that depend on the rate at which the mean age at childbearing changes, independently of changes in completed fertility of cohorts. Ryder (1956) introduced the term "timing distortion" to refer to tempo effects, because they are undesirable in most analyses of fertility levels and trends.

Tempo effects influence demographic processes other than fertility. A tempo effect can be defined in general as an inflation or deflation of the period incidence of a demographic event (births, marriages, and deaths) resulting from a rise or fall in the mean age at which the event occurs.

Tempo effects influence demographic processes other than fertility. A tempo effect can be defined in general as an inflation or deflation of the period incidence of a demographic event (births, marriages, and deaths) resulting from a rise or fall in the mean age at which the event occurs.

2.5 Tempo effects in mortality

A simple example will demonstrate how mortality tempo effects operate. Consider a stationary population with a life expectancy at birth of 70 yr. Suppose

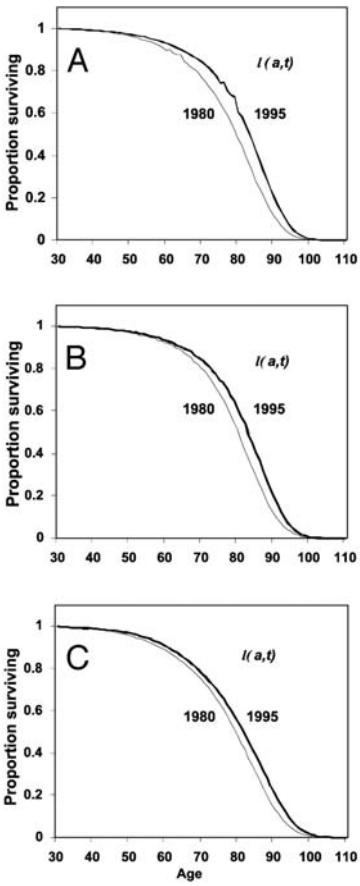


Fig. 4. Observed period survival function $l(a,t)$ in 1980 and 1995 for France (A), Sweden (B), and the U.S. (C).

the exact age of death of each individual is predetermined until the invention of a "life extension" pill that adds 3 mo to the life of any person who consumes it.

If everyone in the population takes this pill on January 1 of year T, there will be no deaths during the first 3 mo of the year. The number of deaths in year T will fall by 25%, and the mean age at death will rise from 70 to 70.25 yr. Because the pill's effect is the same at all ages, the level of the force of mortality function is also reduced by 25%, and the age to which each value of the function is attached increases by 0.25 yr. This fall in values of the force of mortality function, together with the shift to older ages, causes life expectancy at birth as conventionally calculated to rise to ≈ 73 yr for year T.

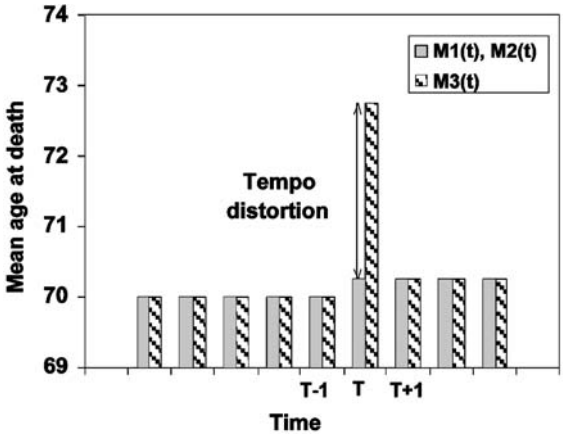


Fig. 5. Hypothetical illustration of effect of increase in mean age at death by 0.25 yr (from 70.0 to 70.25) during year T on conventional life expectancy. Before and after T, $M_1(t) = M_2(t) = M_3(t)$. During T, a tempo distortion of -25% in the number of deaths results in an upward distortion of ≈ 2.5 yr in $M_3(t)$.

In the next year, the number of deaths and the force of mortality function rise to the level observed before year T, but with values shifted forward to older ages by 0.25 yr. Life expectancy at birth as conventionally calculated, having risen from 70 yr prior to year T to ≈ 73 yr during year T, falls back to 70.25 yr (Fig. 5). We contend that this temporary rise in life expectancy at birth as conventionally calculated is a tempo distortion, because it is at variance with the known trend in the mean length of life. Distortion of this kind occurs whenever the standardized mean age at death changes.

2.6 Removing tempo effects

The tempo effect deflates (inflates) $d(a,t)$ and $\mu(a,t)$ when the standardized mean age at death rises (falls). Formulas (8b,c) show this deflation or inflation is estimated by the multiplicative factor $1-\partial M_2(t)/\partial t$ when the proportionality assumption holds. The tempo effect may therefore be removed by dividing $d(a,t)$ and $\mu(a,t)$ by $1 - \partial M_2(t)/\partial t$. Because $M_1(t) = M2(t)$, division by $1 - \partial M_1(t)/\partial t$ gives the same result. The latter approach is preferred, because it gives more stable results when applied to observed mortality rates. We define

$$\mu^*(a,t) = \frac{\mu(a,t)}{1 - \partial M_1(t)/\partial t} \quad \text{and} \tag{10a}$$

$$d^*(a,t) = \frac{d(a,t)}{1 - \partial M_1(t)/\partial t} \tag{10b}$$

and refer to the expressions on the left as the tempo-adjusted death density and force of mortality. It follows from Eq. (8) that $\mu^*(a,t) = \mu_s(a,t)$ and $d^*(a,t) = d_s(a,t)$ when the proportionality assumption holds.

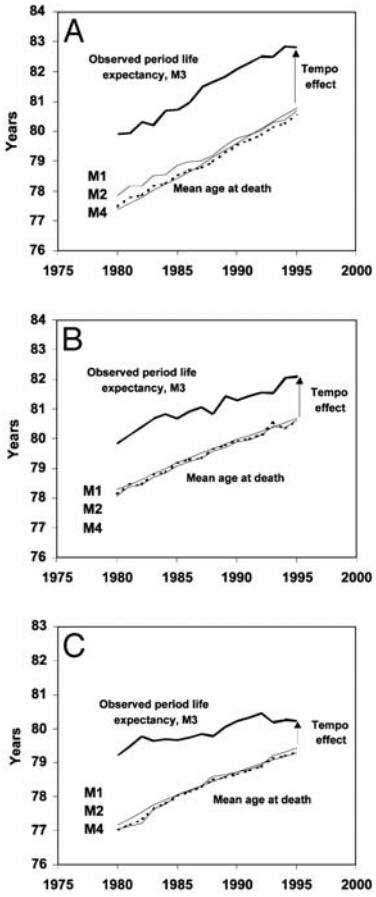


Fig. 6. Trends from 1980 to 1995 for alternative estimates of mean age at death M_1, M_2, M_3, and M_4 for France (A), Sweden (B), and the U.S. (C). The difference $M_3 - M4$ equals the tempo effect.

To calculate life expectancy at birth corrected for the tempo effect, the defining formula (4c) is used with $\mu^*(a,t)$ substituted for $\mu(a,t)$, giving

$$M_4(t) = \int_0^\infty \exp\left\{ - \int_0^a \left[\frac{\mu(x,t)}{1 - \partial M_1(t)/\partial t} \right] dx \right\} da$$
$$= \int_0^\infty \exp\left\{ - \int_0^a \mu_s(a,t)\, dx \right\} da = \int_0^\infty l(a,t)\, da = M_1(t)\,, \quad (11)$$

where $M_4(t)$ denotes life expectancy at birth without the tempo effect. Removing the tempo effect from $M_3(t)$ gives the same result as $M_1(t)$ or $M_2(t)$. The undistorted life expectancy at birth can be estimated as $M_1(t)$, $M_2(t)$, or $M_4(t)$.

Table 1 shows average annual values of $M_4(t)$ as well as $M_1(t)$, $M_2(t)$, and $M_3(t)$ for females in France, Sweden, and the U.S. for the period 1980-1995. The corresponding annual trends are plotted in Fig. 6. These results confirm that $M_1(t)$, $M_2(t)$, and $M_4(t)$ are nearly identical, but $M_3(t)$, the life expectancy at birth calculated by conventional life table methods, is substantially higher than the other three means. The tempo effect, $M_3(t)$ minus $M_4(t)$, averages 2.4 yr for France and 1.6 yr for Sweden and the U.S.

This analysis of tempo effects is based on trends in adult mortality only. We ignore any tempo effects in mortality under age 30, because they are probably small and difficult to quantify. In the absence of tempo effects under age 30, the tempo effect in life expectancy at birth is only 2% or 3% smaller than the tempo effect above age 30 measured here. This is because the probability of survival from birth to age 30 is typically 0.98-0.97 in contemporary societies with high life expectancy.

3 Conclusion

Life expectancy at birth as conventionally calculated is distorted whenever it is changing. We have provided formulas to adjust for this distortion. The formulas are applicable to populations with high life expectancy. The adjustments for France, Sweden, and the U.S. in recent decades reduce conventionally calculated life expectancy at birth by 1.6 to 2.4 yr. These results confirm and extend those given in Bongaarts and Feeney (2002).

The essential argument is as follows. Empirical observation indicates that the proportionality assumption is closely approximated when life expectancy at birth is high and child and young adult mortality are ignored. When the proportionality assumption holds, increases (decreases) in length of life are realized by a uniform translation of the standardized population age distribution and the force of mortality function inherent in this age distribution to higher (lower) ages. Neither the shape nor the level of the standardized age distribution or the inherent force of mortality function changes; only their location on the age scale changes.

The force of mortality function is likewise translated to higher (or lower) ages without any change in shape, but its level changes with the rate of change

in the standardized mean age at death, as shown by Eq. (8b). When the standardized mean age at death rises (falls), the force of mortality function falls and shifts to the right (rises and shifts to the left). This fall (rise) in the force of mortality represents the tempo effect and produces an undesirable rise (fall) in life expectancy at birth as conventionally calculated. In our hypothetical example (Fig. 5), increasing the standardized mean age at death from 70 to 70.25 yr over 1 yr results in a temporary decline of 25% in the force of mortality function and a temporary rise of nearly 3 yr in conventionally calculated life expectancy at birth. The tempo effect in life expectancy in this case is ≈ 10 times the net change in mean lifetime.

In interpreting these findings, it is important to distinguish between current observed death rates and current mortality conditions Ryder(1956). We do not question the conventional life table calculation of period life expectancy from observed age-specific death rates. We argue rather that tempo effects distort both the observed death rates and the corresponding life expectancy, so that their values give a misleading indication of current mortality conditions.

Our empirical focus has been on human survival, but life table methods are widely applied to survival data of all kinds. Examples include age at marriage (the interval between birth and marriage), birth interval analysis (intervals between successive births), length of schooling (interval between entering and leaving school), and postoperative survival (interval between operation and death). It is therefore likely that tempo effects are pertinent to many other kinds of statistical survival analyses.

Acknowledgements

We are grateful to Joshua Goldstein, Michel Guillot, Shiro Horiuchi, and Kenneth Wachter for comments on an earlier version of this article. This research was supported by the Andrew W. Mellon Foundation and the Hewlett Foundation.

References

Arthur, W. B. and Vaupel, J. W. (1984). Some general relationships in population dynamics. *Population Index*, 50:214–226.

Bennett, N. and Horiuchi, S. (1981). Estimating the completeness of death registration in a closed population. *Population Index*, 47:207–221.

Bongaarts, J. and Feeney, G. (1998). On the quantum and tempo of fertility. *Population and Development Review*, 24:271–291.

Bongaarts, J. and Feeney, G. (2002). How long do we live. *Population and Development Review*, 28:13–29.

Council of Europe (2001). Recent demographic developments in Europe 2001. Council of Europe Publishing, Strasbourg, France.

Hajnal, J. (1947). The analysis of birth statistics in the light of the recent international recovery of the birth-rate. *Population Studies*, 1:137–164.

Hajnal, J. (1953). Age at marriage and proportions marrying. *Population Studies*, 7:111–136.

McKendrick, A. G. (1926). Applications of mathematics to medical problems. *Proceedings of the Edinburgh Mathematical Society*, 40:98–130.

Preston, S. H. and Coale, A. J. (1982). Age structure, growth, attrition, and accession: A new synthesis. *Population Index*, 48:217–259.

Preston, S. H., Heuveline, P., and Guillot, M. (2001). *Demography: Measuring and Modeling Population Processes*. Blackwell, Malden, MA.

Ryder, N. B. (1956). Problems of trend determination during a transition in fertility. *Milbank Memorial Fund Quarterly*, 34:5–21.

Ryder, N. B. (1964). The process of demographic translation. *Demography*, 1:74–82.

Ryder, N. B. (1980). Components of temporal variations in American fertility. In *Demographic Patterns in Developed Societies*, pages 15–54. Taylor & Francis, London.

Trucco, E. (1965a). Mathematical models for cellular systems. The Von Foerster equation. *Bulletin of Mathematical Biophysics*, 27:285–304.

Trucco, E. (1965b). Mathematical models for cellular systems. The Von Foerster equation. *Bulletin of Mathematical Biophysics*, 27:449–470.

United Nations (1990). Patterns of first marriage. United Nations, New York.

Vaupel, J. W. (2002). Life Expectancy at Current Rates vs. Current Conditions: A Reflexion Stimulated by Bongaarts and Feeneys "How Long Do We Live?". *Demographic Research*, 7:365–377.

Von Foerster, H. (1959). Some remarks on changing populations. In Stohlman, F., editor, *The Kinetics of Cellular Proliferation*, pages 382–407. Grune and Stratton, New York.

Appendix A

We have to prove that the proportionality assumption (Eq. (6)) implies Eq. (8a) of the text. Bennett and Horiuchi(1981), Preston and Coale(1982), and Arthur and Vaupel (1984) show that

$$\mu(a,t) = \mu_s(a,t) - r(a,t) \tag{A1}$$

where

$$r(a,t) = \frac{-\partial l(a,t)/\partial t}{l(a,t)} \tag{A2}$$

is the age-specific growth rate for age a at time t for the population whose age distribution at time is t given by $l(a,t)$. Note that Eq. (A1) may be written as

$$\mu(a,t) = -\left[\frac{\partial l(a,t)/\partial a}{l(a,t)} + \frac{\partial l(a,t)/\partial t}{l(a,t)}\right], \tag{A3}$$

which is an equation used in modeling cell population dynamics (McKendrick, 1926; Von Foerster, 1959; Trucco, 1965a,b). Equating the expressions for $\mu(a,t)$ given by the proportionality assumption (Eq. (6a)) and Eq. (A1) and rearranging terms gives

$$r(a,t) = [1 - p(t)]\mu_s(a,t)\,. \tag{A4}$$

Substitution of Eqs. (A2) and (5b) in Eq. (A4) yields

$$\frac{\partial l(a,t)}{\partial t} = [1 - p(t)]\frac{-\partial l(a,t)}{\partial a}\,. \tag{A5}$$

From the definition (Eq. (4a)) of $M_1(t)$, then,

$$\begin{aligned}\frac{\partial M_1(t)}{\partial t} &= \frac{\partial}{\partial t}\int_0^\infty l(a,t)\,da = \int_0^\infty \frac{\partial l(a,t)}{\partial t}\,da \\ &= [1 - p(t)]\int_0^\infty \frac{-\partial l(a,t)}{\partial a}\,da\,.\end{aligned} \tag{A6}$$

Because the last integral on the right equals one, we have established formula 8a of the text.

Integrating the density function $d(a,t)$ over age results in a period mortality measure that may be called the total mortality rate $TMR(t)$. (This measure is equivalent to the total fertility rate widely used in the analysis of fertility levels and trends.)

$$TMR(t) = \int_0^\infty d(a,t)\,da\,. \tag{A7}$$

Substitution of Eq. (8a) gives

$$TMR(t) = \int_0^\infty p(t)d_s(a,t)\,da = p(t)\,. \tag{A8}$$

Appendix B

We have to prove that the proportionality assumption implies uniformly shifting age distributions, i.e., Eq. (9), provided there is no mortality at younger

ages. The first step is to find a characterization of uniformly shifting age distributions that applies to a point in time. The directional derivative provides such a characterization. The directional derivative of the function $l(a,t)$ at the point (a,t) in the direction (b,u) is the rate of change at time t of the function $l(a+bt, t+ut)$, which may be expressed as

$$\frac{1}{\sqrt{b^2+u^2}}\left[b\frac{\partial l(a,t)}{\partial a}+u\frac{\partial l(a,t)}{\partial t}\right]. \tag{A9}$$

Now let $f(a,t)$ be such that the directional derivative of $l(a,t)$ at the point (a,t) in the direction $(f(a,t),1)$ equals zero. Uniform translation corresponds to the condition that $f(a,t)$ be constant with respect to age, $f(a,t)\ f(t)$ for all t, and therefore to the condition

$$f(t)\frac{\partial l(a,t)}{\partial a}+\frac{\partial l(a,t)}{\partial t}=0\,. \tag{A10}$$

If this identity holds, the directional derivative of $l(a,t)$ at the point (a,t) in the direction $(f(t),1)$ is zero.

If the proportionality assumption holds, text formula (8b) holds (as just shown in Appendix A), and this together with Eq. (A1) implies, equating the expressions for $\mu(a,t)$ and rearranging terms,

$$\frac{\partial M_1(t)}{\partial t}\mu_s(a,t)-r(a,t)=0\,. \tag{A11}$$

Multiplying both sides by $-l(a,t)$ gives

$$\frac{\partial M_1(t)}{\partial t}\frac{\partial l(a,t)}{\partial a}+\frac{\partial l(a,t)}{\partial t}=0\,, \tag{A12}$$

which shows that the directional derivative of $l(a,t)$ at (a,t) in the direction $(f(t),t)$ equals 0 for all ages a, with $f(t)=\partial M_1(t)/\partial t$.

To show that this implies uniform shifting of the age distribution, it is necessary only to note that $f(t)$ is the rate of change of the contour line in the age-time plane defined by the points $(x+t,t)$ for which $l(x+t,t)=l(a,0)$. The function $F(t)$ of the uniform shifting formula (Eq. (9)) therefore equals the integral of $f(\cdot)$ from 0 to t.

The quantum and tempo of life-cycle events*

John Bongaarts[1] and Griffith Feeney[2]

[1] Population Council, 1 Dag Hammarskjold Plaza, New York, NY 10017, USA. E-Mail: jbongaarts@popcouncil.org
[2] 9 Fairview Road, Scarsdale, NY 10583, USA. E-Mail: feeney@gfeeney.com

Summary. This study develops and applies a general framework for the analysis of the period quantum and tempo of life-cycle events, extending methods developed previously by the authors. The existence of tempo distortions is demonstrated in selected period *quantum* measures such as the total fertility rate and in period *tempo* measures such as life expectancy. A tempo distortion is defined as an inflation or deflation of a period quantum or tempo indicator of a life-cycle event, such as birth, marriage, or death, that results from a rise or fall in the mean age at which the event occurs. Period measures derived from life tables are also found to be subject to tempo distortions. Methods to remove these tempo distortions are then developed and applied.

1 Introduction

Questions about human life-cycle events are central to demographic analysis and to social and health policies. How many children do we have? How long do we live? What proportion of men and women ever marry? When do we retire? How much time in old age is spent in good health?

To answer such questions standard demographic methods have been developed to measure key dimensions of the distribution of events over the life cycle. Attention usually focuses on the two primary components of these distributions, the level or *quantum* component and the timing or *tempo* component. Quantum is measured as the average number of events over the course of the life cycle and tempo as the mean age at the event[3]. The total fertility rate is a quantum measure of fertility, for example, and life expectancy at birth is a tempo measure of mortality.

The quantum and tempo of events can be measured either for *cohorts*, to summarise the actual experience of a group of persons born in the same year,

* ©2006 Vienna Yearbook of Population Research, pp. 115-151. http://www.oeaw.ac.at/vid/yearbook/. Reprinted with permission
[3] On the standard hypothetical condition that the cohort experiences no deaths.

or for *periods*, to describe the experience of a hypothetical cohort subject to the conditions observed in a given time period. Cohort measures of quantum and tempo are easily and unambiguously obtained by following a cohort over time until it reaches an age at which the risk of the event equals zero.

Period measures of quantum and tempo, though conceptually more difficult, are far more widely used for two main reasons. First, because cohort indicators measure ongoing changes in demographic processes after a lag, they cannot adequately describe year-to-year changes. Second, period measures require less historical data than cohort measures and may therefore be calculated for many more countries and more times.

This study continues our work on tempo effects in demographic analysis. Bongaarts and Feeney (1988a) defines period tempo distortions for total fertility rates and provides a method for correcting these distortions. Bongaarts and Feeney (2002, in this volume p. 11) gives analogous results for life expectancy. This chapter develops and applies a general framework for the analysis of the period quantum and tempo of life-cycle events of all kinds, with empirical examples for fertility, marriage and mortality. We begin with a brief presentation of the two main types of age-specific rates from which period quantum and tempo measures are calculated. The remainder of the study is divided into two parts corresponding to the two types of rates. Both of these parts demonstrate the existence of tempo distortions in selected period quantum and tempo measures. A tempo distortion is defined as an inflation or deflation of a period quantum or tempo indicator of a life-cycle event, such as birth, marriage, or death, that results from a rise or fall in the mean age at which the event occurs. We then develop and apply methods to remove these tempo effects.

2 Background: age-specific event rates

Two types of age-specific rates are used in demographic analysis: rates *of the 1st kind*, or *hazard* rates; and rates *of the 2nd kind*, or *incidence* rates (Henry 1972; Sobotka 2003, 2004a; Kohler and Ortega 2002a).

Rates of the 1st kind (hazard rates) are illustrated by standard age-specific death rates. They are quotients in which the numerator counts events occurring to persons at age a and time t and the denominator counts persons *exposed to risk* of the event in question at age a and time t. These rates are also called risks, intensities, conditional rates and occurrence/exposure rates. For the life–cycle events considered in this chapter–first birth, first marriage, and death-persons exposed to risk are those who have not already experienced the event.

Rates of the 2nd kind (incidence rates) are illustrated by standard age–specific birth and marriage rates. They are quotients in which the numerator counts events occurring to persons at age a and time t and the denominator counts *all persons* at age a and time t, including those who have already

experienced the event. Incidence rates are also called densities, unconditional rates, reduced rates, and frequencies.

The relation between rates of the 1st kind and rates of the 2nd kind is straightforward for first births, first marriages, and death. The denominators of hazard rates exclude persons who have already experienced the event, whereas the denominators of rates of incidence include these persons. This relationship may be expressed using life table notation as $\mu(a,t) = d(a,t)/p(a,t)$, where $\mu(a,t)$ denotes a hazard rate, $d(a,t)$ a corresponding incidence rate, and $p(a,t)$ denotes the proportion of persons born at time $t-a$ who have not experienced the event by age a.

The relation between the two kinds of rates for events that can occur more than once in a lifetime (recurrent events) is more complicated. The established way of dealing with recurrent events is to number events in order of occurrence to each individual. For example, births are divided into first births, second births, third births, and so on. In this way any recurrent event may be resolved into a series of non-recurrent events, which can be analysed separately.

Table 1 displays and compares the two kinds of rates for first birth, first marriage, and death. The first row shows clearly the distinction between rates of the 1st and 2nd kind. The numerators of the two rates are the same (first births), but the denominators of rates of the 1st kind exclude women who have already had a first birth, whereas the denominators of rates of the 2nd kind include these women. Summing these rates of the 2nd kind for all birth orders gives the standard age-specific birth rates from which the total fertility rate is calculated.

The second row of Table 1 shows the two kinds of rates for first marriages. As in the case of first births, the numerators of the two rates are the same. The denominators of rates of the 1st kind exclude women who have already married, whereas the denominators of rates of the 2nd kind include these women.

The last row of the table shows the two kinds of rates for death. The rates of the 1st kind are standard age-specific death rates or, given the continuous formulation, the force of mortality. As in the case of the rates for first birth and first marriage, the numerators of the two kinds of death rates are the same, but the denominators of the rates of the 1st kind exclude persons who have already experienced the event–i.e., persons who have died–whereas the denominators of rates of the 2nd kind include these persons. Thus the denominators of the death rates of the 2nd kind include persons in the cohort who have already died as well as those who are living.

Death rates of the 2nd kind are obviously unconventional, for although the quotient shown is a standard demographic statistic–the value of $d(x)$ in the cohort life table for persons born at time $t-a$–this statistic has not generally been regarded as comparable to the other frequencies shown in the table. Indeed, it is only regarded in this way in a very few studies, e.g., by Sardon (1993, 1994) and Bongaarts and Feeney (2002, in this volume p. 11).

Table 1. Rates of the 1st and 2nd kind for first birth, first marriage, and death.

Event	Rates of the 1st kind (occurrence-exposure rates)	Rates of the 2nd kind (frequencies)
First birth	1st births at age a and time t Childless women age a at time t	1st births at age a and time t All women age a at time t
First marriage	1st marriages at age a and time t Never-married women at age a and time t	1st marriages at age a and time t All women age a at time t
Death	Deaths at age a and time t Persons living at age a and time t	Deaths at age a and time t All persons born at time t a

Death rates of the 2nd kind are strictly analogous to first birth rates and first marriage rates of the 2nd kind. For all three events, the denominator includes persons who have not yet experienced the event as well as persons who have already experienced the event. The characterisation of the denominator for death rates of the 2nd kind appears exceptional only because "persons" usually connotes "living persons", though of course it may refer to deceased persons as well.

An important general property of rates of the 2nd kind for non-repeatable life cycle events is that the sum (integral) of these rates over all ages for a birth cohort gives the proportion of the cohort that experiences the event. Thus summing age-specific first birth rates gives the proportion of women ever having a (first) child, and summing age-specific first marriage rates gives the proportion ever marrying. The sum of the death rates of the 2nd kind over all ages for a birth cohort will equal one because everyone dies.

The interpretation of the sum of rates of the 2nd kind over all ages as the proportion experiencing the event is straightforward and unambiguous for *cohorts*. Calculating the sum of first birth, first marriage or death rates for *periods* is equally straightforward, but the interpretation of these sums as the proportions ever experiencing the event in synthetic cohorts is problematic due to tempo effects in period measures, as will be demonstrated next.

3 Period quantum and tempo measures of the 2nd kind

3.1 Standard equations for quantum and tempo measures

Table 2 presents general equations for calculating period quantum and tempo from rates of the 2nd kind and specific results for first birth, first marriage, and death. The total event rate $TER(t)$ and the mean age at event $MAE(t)$ are defined by the formulas in the first row of the table, with $d(a,t)$ denoting the age-specific rate of the 2nd kind for any of the events shown. The total event rate equals the average number of events over the life cycle for a hypothetical cohort subjected to the rates at time t (in the absence of competing events).

For events that occur only once, the total event rate equals the proportion of persons in the hypothetical cohort who ever experience the event.

The quantum and tempo measures of first birth and first marriage in Table 2 are standard tools in demographic analysis and estimates are available for many countries. The total mortality rate and the mean age at death (birth cohort normalised), though defined in precise analogy with the fertility and mortality measures, were introduced for the first time by Sardon (1993, 1994) and further analysed in Bongaarts and Feeney (2002, in this volume p. 11; note that $MAD(t)$ is not the mean age of deaths occurring at time t because the effects of variations in cohort size are removed). The table therefore illustrates that measures that are standard for some demographic processes may be unknown in the study of other processes.

Table 2. Period measures of quantum and tempo based on rates of the 2nd kind for first birth, first marriage, and death.

	Period quantum
General formula	Total event rate, $TER(t)$
	$TER(t) = \int_0^\infty d(a,t)da$
First birth	Total fertility rate, order 1, $TFR_1(t)$
First marriage	Total 1st marriage rate, $TNR_1(t)$
Death	Total mortality rate, $TMR(t)$
	Period tempo
General formula	Mean age at event, $MAE(t)$
	$MAE(t) = \frac{1}{TER(t)} \int_0^\infty ad(a,t)da$
First birth	Mean age at 1st birth, $MAB_1(t)$
First marriage	Mean age at 1st marriage, $MAM_1(t)$
Death	Mean age at death (birth cohort normalised), $MAD(t)$

Note: "TNR" (N for "nuptiality") is written for 1st marriage so that "TMR" may be used for Total mortality rate.

Figures 1 to 3 present empirical results for the quantum and tempo measures summarised in Table 2 for selected populations. Figure 1 shows total fertility rates for birth order one, $TFR_1(t)$, and the mean age at first birth, $MAB_1(t)$, for the United States from 1950 to 2000. Values of $TFR_1(t)$ exceeded one for most of the 1950s, an obvious anomaly since no woman can have more than one first birth. This period of elevated fertility coincided with the decline in the age at first birth during the baby boom years of the 1950s.

Figure 2 shows total first marriage rates, $TNR_1(t)$, and the mean age at first marriage, $MAM_1(t)$, for France from 1960 through 2001. The above-one rates for France in the early 1960s are anomalous because a woman can experience at most one first marriage. The apparent explanation, by analogy with that for first births, is the declining mean age at first marriage. First marriage rates for France decline over the period shown, with values around 0.5 toward the end of the period. Similar trends are observed in many other

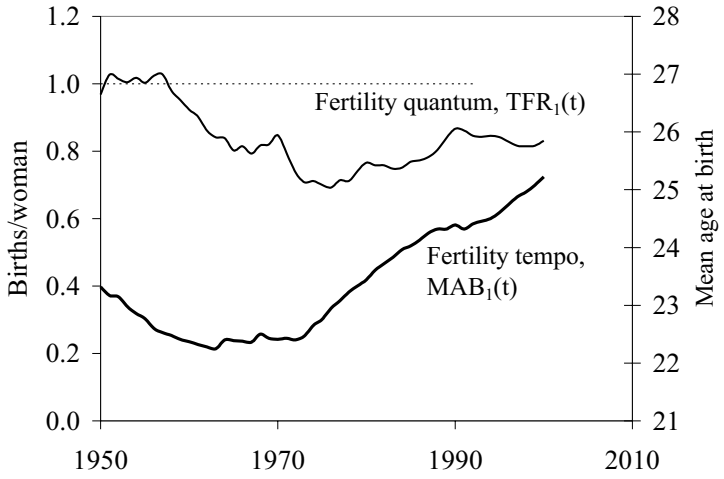
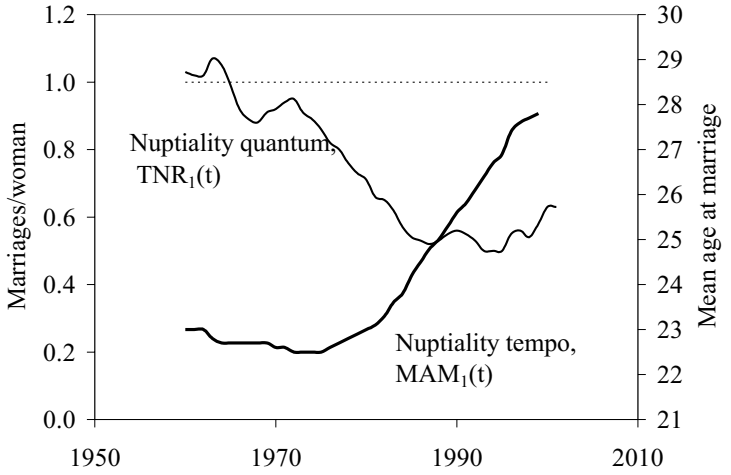


Fig. 1. Total fertility rate, order one, and mean age at first birth in the USA.

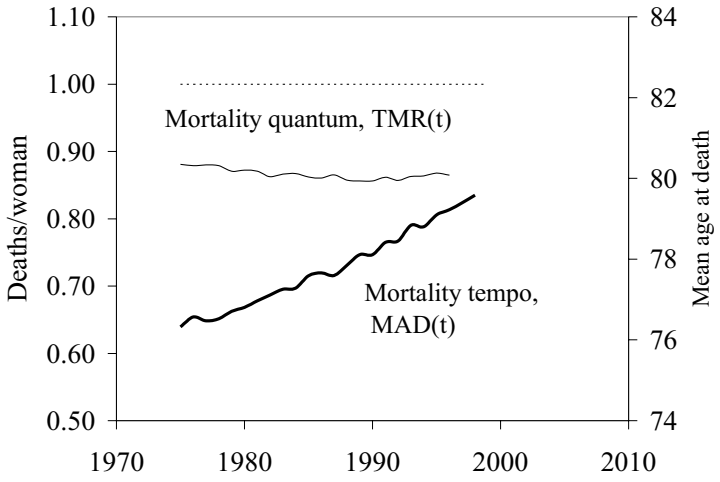
European countries, but proportions ever-married for cohorts born in the late 1960s are much higher than 0.5 (Council of Europe 2002). This suggests that the low first marriage rates are distorted.



Source: Council of Europe 2002

Fig. 2. Total first marriage rate and mean age at first marriage, females in France.

Figure 3 shows total mortality rates, $TMR(t)$, and the mean age at death, $MAD(t)$, for England and Wales from 1975 to 1998. (For reasons given below all mortality measures in this study include adult mortality above age 30 only.) The total mortality rate is well below one (0.85-0.90). Since every person dies once, any total mortality rate other than one is anomalous. Mortality tempo (MAD) rose sharply throughout the period, and the analogy for first birth and first marriage therefore suggests again that this is the reason for the TMR values different from one.



Source: Estimated from data in Human Mortality Database (2005)

Fig. 3. Total mortality rate and mean ages at death, females in England and Wales (adult mortality only).

3.2 Tempo effects

We will now demonstrate that the various anomalies evident in Figures 1, 2, and 3 are largely attributable to tempo effects. A tempo effect is defined as an inflation or deflation of the number of events observed in a period when the period (cohort size adjusted) mean age changes. Tempo effects in event numbers lead to tempo effects in event rates (of the first and second kind) and these in turn lead to tempo effects in most period tempo and quantum measures. Tempo effects in these aggregate measures (but not in rates) will also be referred to as distortions, following terminology introduced by Ryder (1956) in his analysis of the fertility tempo effect. This section presents

the theoretical basis for this effect and offers additional empirical evidence supporting the theory.

Theoretical basis for tempo effects

Norman B. Ryder (1956, 1959, 1964, 1980, 1983) made a series of fundamental contributions to the study of quantum and tempo measures. His paradigmatic contribution was a simple model that showed that the period total fertility rate (TFR) does not, in general, equal the cohort completed fertility rate (CFR) even if fertility has been constant for a long period of time. His "translation" formula

$$TFR = CFR(1 - r_c) \tag{1}$$

shows that the TFR in a constant fertility population tends to be lower than the CFR when the cohort mean age at childbearing is rising (i.e., the rate of change in this mean, r_c, is positive and hence $(1 - r_c) < 1$) and higher than the CFR when the mean age at childbearing is falling (r_c is negative, $(1-r_c) > 1$). This equation assumes linearity in time trends of the age-specific fertility rates. Ryder refers to $(1 - r_c)$ as an "index of fertility distortion" and he considered the TFR to be a distorted measure when the fertility tempo changes.

Ryder's analyses of period fertility trends in the United States (1980, 1983) showed how changes in the timing of childbearing among cohorts of women influenced annual age-specific birth rates and total fertility rates. When women shift upward the ages at which they bear children, annual numbers of births tend to be deflated because the same number of births will be spread out over a longer time period (e.g., during the 1970s and 1980s). Similarly, when age at childbearing shifts to younger ages, total fertility rates tend to be inflated because the same number of births are compressed into a shorter time period (e.g., during the late 1940s and 1950s).

Zeng and Land (2002) extend Ryder's analysis by deriving the following translation formula,

$$TFR = CFR(1 - r_p)\,, \tag{2}$$

where r_p denotes the rate of change in the period mean age at childbearing and TFR, CFR, r_p, and the shape of the schedule of age-specific fertility rates are assumed constant. They consider this alternative version of the translation equation preferable to (1) because their constant shape assumption is more realistic than Ryder's linearity assumption.[4] The conditions under which (2) holds (i.e., constant quantum, fixed rate of increase in the period mean, and an invariant shape) will collectively be referred to as the "translation assumptions".

[4] Zeng and Land (2002) prove this with their assumptions $TFR = CFR/(1 + r_c)$ and $r_c = r_p/(1 - r_p)$, where r_p is the rate of change of the period mean age of childbearing. Cf. formula (2).

These translation equations were developed for the analysis of fertility trends, but analogous equations apply to other life-cycle processes provided that the same translation assumptions apply. For mortality, for example, we have

$$TMR = CMR(1 - r_p)\,, \tag{3}$$

where *TMR* is the total mortality rate, *CMR* denotes the cohort completed mortality rate, and r_p is the rate of change in the period mean age at death, *MAD* (see Table 2). Because everyone dies once, the *CMR* equals (1) and (3) simplifies to

$$TMR = 1 - r_p \tag{4}$$

This result shows the operation of the tempo distortion in its most basic form: the *TMR* simply equals the distortion index. The undistorted value of $TMR = 1$ is obtained only if the mean age at death is constant (i.e., $r_p = 0$). Any change in the mean age at death, whether up or down, results in a tempo effect in the total mortality rate and in the mortality rates of the 2nd kind from which it is calculated. The effect is evident in Figure 3, which shows that estimates of *TMR* for England and Wales is about 0.86. This is more or less consistent with the rate of change in the mean age at death shown in Figure 3, about 0.14 years per year.

The period-cohort translation formulas of Ryder (1) and Zeng and Land (2) may be applied when fertility is changing slowly by comparing the *TFR* for any given year with the *CFR* for the cohort that reaches its mean age at childbearing in this year (Ryder 1956; Sobotka 2003). If the 1960 birth cohort has a mean age at first birth of 25 years, for example, the *CFR* for this cohort is compared with the *TFR* for 1985. To attenuate year-to-year fluctuations, *TFR*s may be averaged over a series of years.

To illustrate, Table 3 presents evidence for tempo distortion in the *TFR* of France during the last quarter of the 20th century. The *TFR* was relatively stable during this period, with an average value of 1.80 children per woman. The completed fertility rate (*CFR*) for the cohorts that were at prime childbearing ages during these periods was also nearly stable, but with an average value of 2.08 children per woman, 0.28 children per woman higher than the average total fertility rate.

This disparity between period and cohort fertility is explained largely by a tempo distortion on the *TFR* resulting from the change in the period mean age at childbearing. This mean rose at an average annual rate of 0.125 years per year between 1975-80 and 1995-99 ($r_p = 0.125$). Since the constant fertility assumption is approximately valid, the *TFR* implied by (2) is $CFR(1 - r_p) = 2.08(1 - 0.125) = 1.82$ births per woman. This is very close to the observed average total fertility rate for the period, 1.80 children per woman. In this example, the translation formula (2) quite accurately estimates the tempo distortion due to rising mean age at childbearing.

In this illustration, the translation formula has been applied to births of all orders. In general, however, it is recommended that the translation formula

Table 3. Analysis of tempo distortion of the period total fertility rate (*TFR*) in France, 1975-1999.

Period	*TFR* **(births per woman)**	*CFR* **(births per woman)**[a]	**Mean age at childbearing (years)**
1975-79	1.86	2.11 (1950)[a]	26.6
1980-84	1.88	2.13 (1955)	27.1
1985-89	1.81	2.10 (1960)	27.9
1990-94	1.72	1.99 (1965)	28.5
1995-99	1.74		29.1
Average	1.80	2.08	

Source: Council of Europe 2002.
Note: a Year of birth of cohort in parentheses.

be applied separately for births of each order, as illustrated in Bongaarts and Feeney (1998a).

Empirical evidence supporting the theory: first births, first marriages, and deaths

This section systematically applies and tests the Zeng-Land translation formula (2) using empirical data for first birth, first marriage, and death.

-First births. Figure 4 compares completed first birth cohort fertility for women born in 1960 (CFR_1) and period first birth total fertility for 1980-89 (TFR_1) for 15 European countries, the USA, and Japan. In most countries, the cohort level exceeds the period level. To show that this difference is due largely to tempo distortions, the translation equation (2) is rearranged as follows:

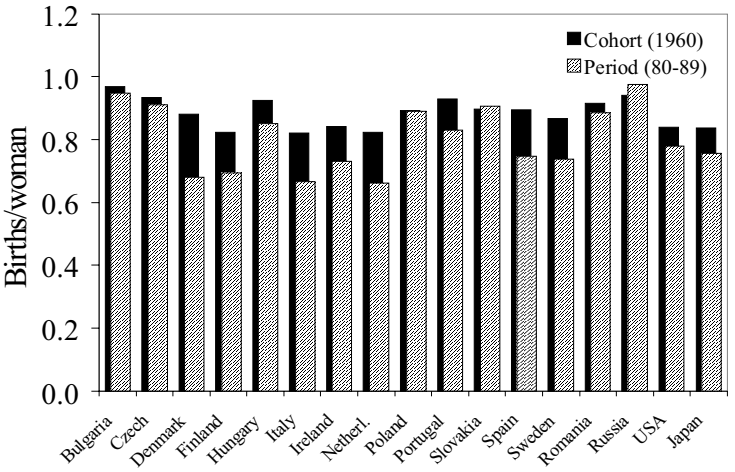
$$\frac{TFR}{CFR} = 1 - r_p \, . \tag{5}$$

This shows that, in a constant fertility population, there is a simple linear relationship between $TFRCFR$ and $(1 - r_p)$, so that if values of these two quantities for different countries are scatter plotted, the points will lie on a straight line with slope one that passes through the origin. We refer to $(1-r_p)$ as the *period distortion index*.

To test the validity of this translation equation for first births, Figure 5 plots TFR_1/CFR_1 ratios (vertical axis) against the corresponding values (horizontal axis)[5] for the 17 countries represented in Figure 4. There is a close correspondence between the data points for the 17 countries and the linear relation predicted by the translation equation. This is consistent with tempo distortions of the TFR_1 being the main explanation for the difference between the TFR_1 and the CFR_1. We do not expect the observations for the different

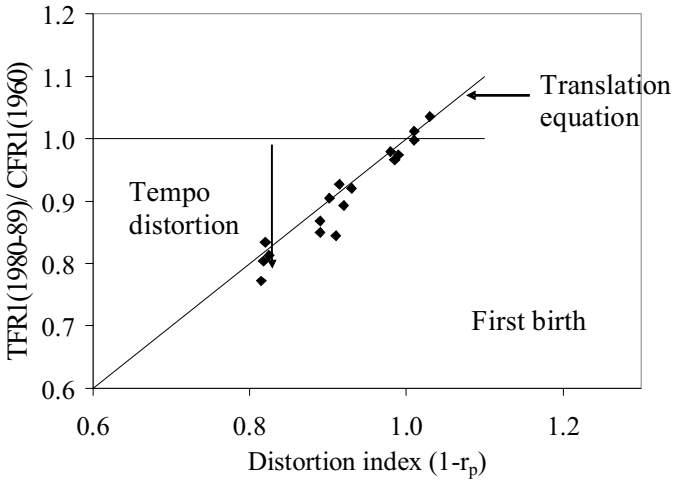
[5] The *TFR* value is the average TFR_1 for 1980-89. The *CFR* value is CFR_1 for the 1960 birth cohort. Values of r_p are estimated as one tenth of the difference between the period mean ages at first birth in 1980 and 1990.

countries to fall exactly on the diagonal because the translation assumptions hold only approximately.



Source: Bongaarts 2002

Fig. 4. Completed cohort fertility (1960) and period total fertility(1980-89), first births.



Source: Bongaarts 2002

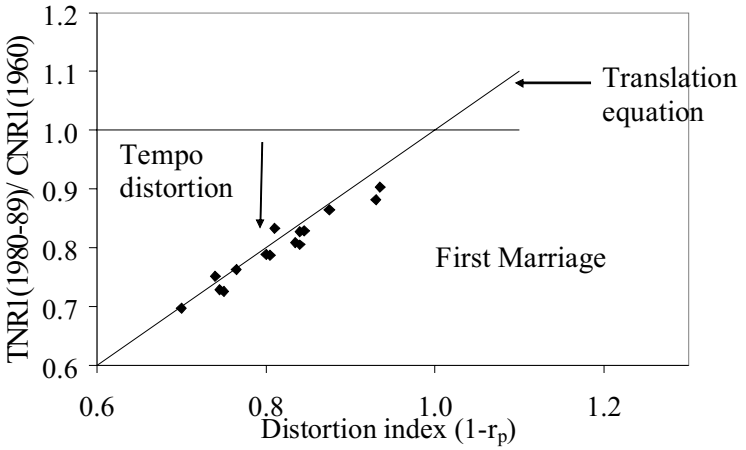
Fig. 5. Ratio of period to cohort fertility rate by tempo distortion index, first births, 17 countries.

-*First marriages*. The same translation formula analysis may be applied to quantum and tempo measures of first marriage. The total first marriage rate is influenced by tempo effects in the same way that the total fertility rate is. When the mean age at marriage is rising (falling), the same number of marriages occur over a longer (shorter) period and annual numbers of marriages are lower (higher) than they would have been in the absence of the change in mean age. Most of the concepts and derivations developed for the analysis of fertility tempo apply to the analysis of "nuptiality tempo" as well. Recent studies by Goldstein (2003) and by Winkler-Dworak and Engelhardt (2004) provide examples of this application.

Figure 6 tests the translation equation for nuptiality. The ratio of the period to cohort quantum for first marriage (TNR_1/CNR_1) is plotted against the distortion index ($1 - r_p$), with r_p representing the rate of change in period mean age at first marriage. The diagonal line represents the relationship predicted by the translation equation. Most countries again fall close to the predicted values, confirming the existence of tempo effects for first marriage rates.

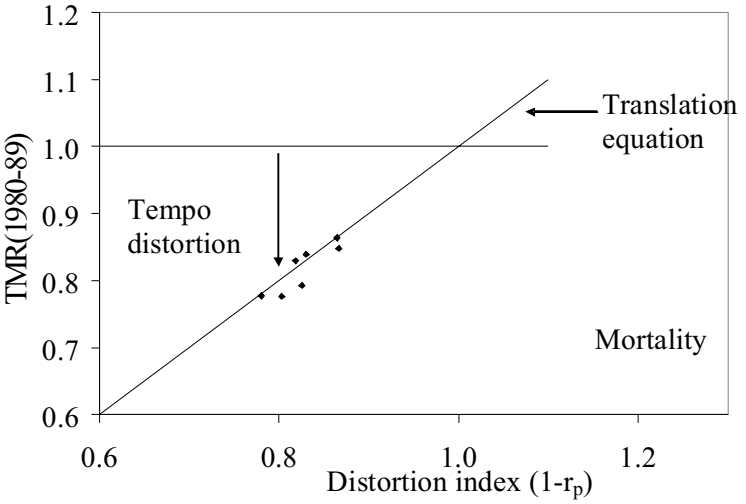
-*Deaths*. We now extend the same translation formula analysis to the quantum and tempo measures of mortality based on rates of the 2nd kind. Since the cohort completed mortality rate necessarily equals one, the period-cohort ratio equals the *TMR*. The relationship predicted by the translation equation (4) is given by the diagonal line in Figure 7. (Following Bongaarts and Feeney (2002, in this volume p. 11) the analysis of the quantum and tempo of mortality is limited to adult mortality above age 30 to ensure consistency with the constant shape assumption.) Figure 7 includes the resulting data points for seven countries (England and Wales, Italy, France, Norway, Switzerland, Sweden, and the US) for which the required historical data from 1900 to the present are available. As in the fertility and nuptiality analyses, the data points fall close to the line predicted by the translation equation, supporting both the validity of the translation equation and the existence of tempo distortions for adult mortality.

These analyses show that the distortions established in the case of fertility apply to nuptiality and (adult) mortality as well when the period quantum measures for each event are calculated from rates of the 2nd kind. They also show that the magnitude of tempo distortions may be substantial. Figures 5-7 show that average distortions of 10 per cent are common during the 1980s and that distortions exceeding 20 per cent occur for some countries for fertility, mortality, and in particular for nuptiality. The distortions are even larger in individual years. Bongaarts and Feeney (1998a), for example, estimate distortions in the *TFR* in the United States ranging from +28% in 1948 to -11 % in 1975.



Source: Council of Europe 2002

Fig. 6. Ratio of period to cohort marriage rate by tempo distortion index, first marriages, 17 countries.



Source: Estimated from data in Human Mortality Database 2005

Fig. 7. Period total mortality rate by tempo distortion index, in 7 countries.

3.3 Correcting tempo distortions in quantum measures of the 2nd kind

Ryder's work established the existence of tempo distortions in the total fertility rate, but he did not propose specific, quantitative adjustments to counteract tempo distortions. This may be explained in part by his strong emphasis on the conceptual priority of cohort fertility. The emphasis on cohorts probably influenced his focus on "translating" period measures to cohort measures as well, which diverted attention from the problem of adjusting period measures for tempo distortions.

Empirical research over the past three decades has demonstrated, however, that period influences on fertility are much more important than cohort influences. Brass (1974) concludes that cohort completed fertility reveals no significant feature that distinguishes it from time averages of period indexes. Pullum (1980) concludes that "temporal variations that cut across cohorts, such as economic cycles, appear to be more important than changes in those variables that distinguish cohorts, such as shared socialising experiences" (see also Page 1977). Foster's (1990) analysis of data for eight countries in Europe and North America arrives at a similar conclusion. In an authoritative review, N Bhrolchin (1992) concludes that "of the two dimensions of calendar time–period and cohort–period is unambiguously the prime source of variation in fertility rates." Bongaarts and Feeney (in this volume p. 11) demonstrate that the same dominance of period effects exists for adult mortality rates in contemporary populations with high life expectancy. These findings provide the basis for the tempo adjustment procedure discussed next.

Correcting tempo distortions in period quantum measures of fertility

Bongaarts and Feeney (1998a) reformulated the issue of tempo distortions by posing the following counterfactual question: What would the total fertility rate have been in a particular year, other things being equal, if the mean age at childbearing had been constant during that year? The purpose is to remove the distortion resulting from a changing mean age of the event to obtain a better measure of current fertility conditions. Subject to a simplifying assumption on the pattern of fertility change, they show that the answer to this question is given by

$$TFR^*(t) = \frac{TFR(t)}{1 - r_p(t)} \tag{6}$$

where r_p denotes the rate of change in the period mean age at childbearing in year t. $TFR^*(t)$ is referred to as the *tempo-adjusted TFR*, and the tempo distortion in the observed TFR equals $TFR^*(t) - TFR(t)$.

Unlike the translation formulas (1) and (2), formula (6) involves only period measures. Another advantage of (6) is that it separates the issue of tempo

distortion from the question of the relationship between period and cohort measures. The tempo-adjusted is not intended to estimate and need not equal the *CFR* for any cohort. (However, as shown in Appendix A, the *CFR* is approximately equal to the weighted average of values observed during the years in which the cohort reproduces.)

Formula (6) depends on the *constant shape assumption*, which may be stated in this way: the age schedule of fertility rates (of the 2nd kind) observed at any time can be transformed into the schedule observed at any other time by inflating or deflating and/or by shifting the schedule to higher or lower ages. This is equivalent to assuming that fertility is determined strictly by period effects. By comparison, formula (2) requires not only the constant shape assumption, but also (page 9) constant quantum and a constant rate of increase of the mean age. The tempo adjustment equation (6) can therefore be applied much more widely. Recent studies by Kohler and Philipov (2001) and Zeng and Land (2001) confirm the mathematical derivation of (6).

Although equation (6) can be applied to births of all orders combined, superior results are obtained by applying the formula separately to each birth order component of the *TFR*, because the constant shape assumption is more valid for the fertility schedule at each order than for all orders combined (Bongaarts and Feeney 1998a). This disaggregation is particularly important in countries in which the overall *TFR* is changing rapidly, for this is likely to result in substantial changes in the weighting of the different birth order components.

Bongaarts and Feeney (1998a) and the follow-up work of Bongaarts (1999a, 1999b, 2002) have stimulated a number of criticisms, extensions, and elaborations. Van Imhoff and Keilman (2000) and Van Imhoff (2001) point out that the constant shape assumption does not hold exactly for the Netherlands and Norway during the second half of the 20th century. This issue is addressed by Zeng and Land (2001), who carried out a sensitivity analysis and concluded that "the Bongaarts-Feeney formula is not sensitive to temporal changes in the shape of the fertility schedules". Kohler and Philipov (2001), on the other hand, find that errors resulting from deviations from the assumption in Sweden were not insignificant and addressed this by proposing a procedure for calculating tempo-adjusted total fertility rates when the variance of the fertility schedule changes over time (see also Kohler and Ortega 2002a and 2002b). A number of past studies have applied the adjustment to fertility (Lesthaeghe and Willems 1999; Smallwood 2002; Sobotka 2003, 2004a, 2004b). Implications of fertility tempo effects for population growth are examined by Goldstein et al. (2003).

Extension of tempo adjustments to nuptiality and mortality

The Bongaarts-Feeney method can be extended to obtain estimates of tempo-adjusted period quantum measures for life-cycle events other than fertility. Table 4 shows formulas for adjusted quantum and tempo for the total first

birth rate, the total first marriage rate, and the total mortality rate. Tempo adjustments are effected by dividing observed event rates of the 2nd kind by the period tempo distortion index, as in formula (6) above. As in Table 2, the formulas in the first row of the table define the tempo-adjusted total event rate $TER^*(t)$ and the tempo-adjusted mean age at event $MAE^*(t)$, with $d(a,t)$ denoting the age-specific rate of the 2nd kind for any of the events shown.

The tempo-adjusted total mortality rate TMR^*

$$TMR^*(t) = \frac{TMR(t)}{1 - r_p} \tag{7}$$

is of particular interest. Because $TMR^*(t)$ must equal one, it follows that

$$TMR(t) = 1 - r_p(t) \tag{8}$$

This is a more general version of (4) because it allows $TMR(t)$ and $r_p(t)$ to vary over time. As noted, the results in Figure 7 confirm this relationship for mortality over age 30.

The right hand column of Table 4 shows that tempo measures based on rates of the 2nd kind are not affected by tempo distortion (assuming the constant shape assumption holds). This is because the distortion index occurs in both the numerator and the denominator of the formula, and so cancels out. Empirical confirmation of this conclusion will be provided in a later section.

Table 4. Adjustments for tempo distortions in period quantum and tempo measures based on rates of the 2nd kind.

Event	**Adjusted quantum** (total event rate)	**Adjusted tempo** (mean age at event)
General	$TER^*(t) = \int_0^\infty \frac{d(a,t)}{1-r_p(t)}\, da$ $= \frac{TER(t)}{1-r_p(t)}$ $r_p(t) = \frac{dMAE(t)}{dt}$	$MAE^*(t) = \frac{1}{TER^*(t)} \cdot$ $\cdot \int_0^\infty \frac{ad(a,t)}{1-r_p(t)}\, da$ $= MAE(t)$
First birth	Tempo-adjusted total fertility rate, order 1, $TFR_1^*(t)$	No adjustment needed: $MAB_1^*(t) = MAB_1(t)$
First marriage	Tempo-adjusted total first marriage rate $TNR_1^*(t)$	No adjustment needed: $MAM_1^*(t) = MAM_1(t)$
Death	Tempo-adjusted total mortality rate $TMR_1^*(t)$	No adjustment needed: $MAD^*(t) = MAD(t)$

Empirical application to first births, first marriages, and deaths

Empirical estimates of tempo-adjusted quantum measures contain seemingly random year-to-year fluctuations. These are caused by sensitivity to small errors in $r_p(t)$ and by deviations from the constant shape assumption. To

minimise these fluctuations, we plot five-year moving averages of $TER^*(t)$ in place of annual values in Figures 8-10.

Figure 8 presents observed and tempo-adjusted total first birth fertility rates for the United States[6]. The adjusted rates are lower than the observed rates during the 1950s and early 1960s, when the mean age at first birth was declining, and higher than the observed rates between 1975 and 1990, when the mean age at first birth was rising.

Figure 9 presents observed and adjusted total first marriage rates for France from 1960 to 1996. The adjusted rates are more plausible than the observed rates, because the adjusted rates are uniformly below one and because the tempo-adjusted total first marriage rate *circa* 1990 is 0.7, which is approximately equal to the proportion ever marrying among cohorts born in the late 1960s.

Figure 10 gives the observed and adjusted total mortality rate (adult mortality only) for England and Wales. The adjusted rate fluctuates around to the level of one, as it should. If the constant shape assumption holds perfectly, the tempo-adjusted TMRs would all equal one exactly, assuming no measurement error.

Figures 8-10 also include the corresponding quantum estimates for successive cohorts. Let $M(c)$ be the mean age at the event for the cohort born in year c. In Figure 8 the cohort quantum (i.e., the proportion ever having a first birth) for the cohort born in year $c = t - M(c)$ is plotted at time t. In Figure 9 this comparison of cohort and period quantum is made for the first marriage quantum and in Figure 10 for the quantum of mortality. In each of these comparisons, the (lagged) cohort quantum is close to the adjusted rate of the 2nd kind, but the fit is not perfect. Perfect agreement between the cohort and adjusted period quantum is expected only when all the translation assumptions hold: the period and cohort quantum are constant, the period mean age is rising linearly, and the shape of the age pattern is constant. Since these assumptions do not hold exactly, the cohort quantum is not exactly equal to the adjusted total event rates, but the correspondence is good and clearly better than for the unadjusted rates. The observed differences between the cohort quantum and adjusted period quantum are due to three factors: deviation from the constant quantum assumption, deviation from the linear change assumption, and deviation from the constant shape assumption. The first two of these deviations do not cause errors in the adjusted quantum, which only requires the constant shape assumption. As a result even when

[6] Application of the tempo-adjustment formulas requires annual estimates of the total event rate and the rate of change in the period mean age of the event. The rate of change during year t is estimated as $0.5[MAE(t+1) - MAE(t-1)]$. Application of formulas in Table 4 then gives the time series of tempo-adjusted total event rates. Since the adjustment is sensitive to small errors in $r_p(t)$, the annual adjusted estimates tend to contain seemingly random fluctuations. To minimise these fluctuations, we use five-year moving averages of $TER^*(t)$ in place of annual values.

the adjusted quantum is accurately estimated, it can differ from the lagged cohort quantum.

In the applications summarised in Figures 8-10 the adjustment procedure appears to work well. The obvious anomalies in unadjusted quantum measures noted earlier are all removed by the tempo adjustment, and the adjusted quantum is close to the lagged cohort quantum, even though the conditions for this comparison are not fully met.

Quantum and tempo measures of the 2nd kind are much more widely used in the analysis of fertility and nuptiality than measures of the 1st kind, because age-specific rates of the 2nd kind (adjusted and unadjusted) are easier to calculate and more widely available. A disadvantage of rates of the 2nd kind is that they can be affected by compositional effects (Kohler and Ortega 2004). It is therefore generally considered preferable to derive quantum and tempo measures from rates of the 1st kind if such rates are available. As we will show below, however, these rates are affected by tempo effects, and therefore need adjustment.

4 Period quantum and tempo measures of the 1st kind

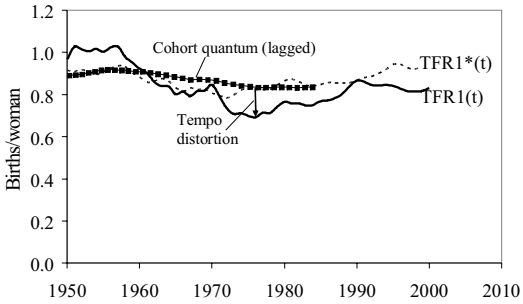
The preceding sections of this study examined quantum and tempo measures of the 2nd kind as well as tempo effects in these measures. The present section will cover these same topics for rates of the 1st kind. These rates are used extensively in life table analyses of the quantum and tempo of life-cycle events. This discussion will be briefer because the main concepts have already been introduced and because data on measures of the 1st kind are not widely available except for mortality.

4.1 Standard equations for quantum and tempo measures

Table 5 presents equations for estimating quantum and tempo measures derived from rates of the 1st kind[7] . Applying the general formulas in the first row to first birth, first marriage, and death produces quantum estimates TFR_{1_L}, $TNR_{1L}(t)$, and $TMR_L(t)$ and tempo estimates $MAB_{1L}(t)$, $MAM_{1L}(t)$, and $MAD_L(t)$. The subscript L signifies that these measures are based on the life table calculation using rates of the 1st kind. This distinguishes them from the corresponding measures based on rates of the 2nd kind (see Table 2).

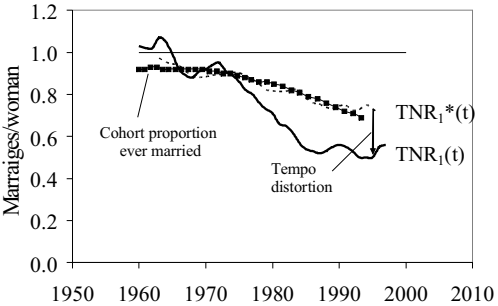
Period quantum based on rates of the 1st kind is defined as the proportion of persons ever experiencing the event in a hypothetical cohort subjected to these rates, as given by the standard life table calculation. The quantum of mortality $TMR_L(t)$ necessarily equals one because everyone eventually

[7] The first formula in the first line of Table 5 shows that $TMR_L(t)$ equals one minus an integral. This integral equals the proportion of individuals that never experiences the event.



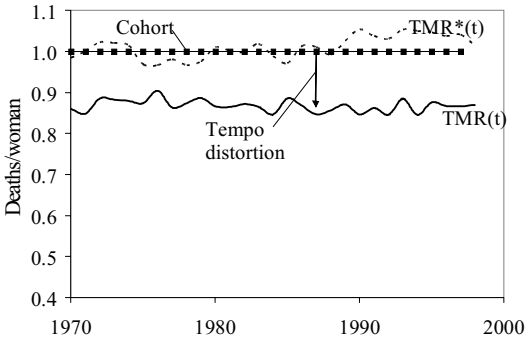
Source: Bongaarts and Feeney, 1998

Fig. 8. Observed and tempo adjusted total fertility rate, birth order one, United States.



Source: Council of Europe 2002

Fig. 9. Observed and tempo adjusted total first marriage rate, females, France.



Source: Estimated from data in Human Mortality Database

Fig. 10. Observed and tempo adjusted total mortality rate, females, England and Wales.

dies. The quantum of first birth $TFR_{1L}(t)$ and the quantum of first marriage $TNR_{1L}(t)$ are less than one because the rates from which they are calculated fall rapidly to zero at older ages.

The most widely used period tempo measure of the 1st kind is life table mean age at death $MAD_L(t)$, which is usually referred to as life expectancy. With $TMR_L(t) = 1$ the general tempo equation on the right in Table 5 simplifies to

$$MAD_L(t) = e_0(t) = \int_0^\infty e^{-\int_0^a \mu(x,t)\,dx}\,da\,, \tag{9}$$

which is the conventional expression for the life table estimate of life expectancy at birth conventionally denoted $e_0(t)$.

Cohort measures based on rates of the 1st kind are identical to the corresponding measures based on rates of the 2nd kind, but period measures based rates of the 1st kind do not in general equal the corresponding measures based on rates of the 2nd kind. This will be illustrated in the following section.

Table 5. Adjustments for tempo distortions in period quantum and tempo measures based on rates of the 2nd kind.

Event	**Quantum** (total event rate = proportion ever experiencing event)	**Tempo** (mean age at event)
General	$TER_L(t) = 1 - e^{-\int_0^\infty \mu(a,t)\,da}$	$MAE_L(t) = \frac{1}{TER_L(t)} \int_0^\infty e^{\int_0^a \mu(x,t)\,dx}\,da + + \int_0^\infty TER_L(t) - 1\,da$
First birth	$TFR_{1L}(t)$	$MAB_{1L}(t)$
First marriage	$TNR_{1L}(t)$	$MAM_{1L}(t)$
Death	$TMR_L(t)$	$MAD_L(t)$

4.2 Tempo effects

Tempo effects result from a depression or inflation in the numbers of events that occur in the numerators of rates. These effects therefore affect age-specific rates of the 1st kind as well as rates of the 2nd kind. Moreover, the effect is proportionally the same for the numerators of age-specific rates of the 1st and 2nd kind and it is determined by the distortion index, which varies with the rate of change in the mean age at the event. This point was first made by Bongaarts and Feeney (1998a) and subsequently by Kohler and Ortega (2002a, 2002b) in their analysis of tempo effects in fertility rates of the 1st kind.

Tempo distortions of measures of the 1st kind are generally less noticeable than distortions of measures of the 2nd kind. Tempo distortions in quantum

measures based on rates of the 2nd kind are obvious, for example, whenever these measures exceed one. This cannot happen for quantum measures based on rates of the 1st kind because the life table calculations used necessarily lead to values less than or equal to one. The absence of obvious anomalies in these measures does not mean that they are free of tempo distortions, however.

Tempo distortions in period quantum measures of the 1st kind are well established and uncontroversial in fertility (Sobotka 2003, 2004a,b; Kohler and Ortega 2002a, 2002b) and in nuptiality (Goldstein 2003; Winkler-Dworak and Engelhardt 2004). Mortality rates of the 1st kind also contain tempo effects, but the period mortality quantum derived from them always equals one because these rates rise with age.

Tempo distortions in period tempo measures of the 1st kind are much less established and we will therefore examine this issue in more detail. The theoretical basis for the existence of such an effect is that tempo measures of the 1st kind are derived from the same numbers of events that produce quantum measures of the 1st kind. As noted earlier, a rising mean age depresses rates of the 1st kind and hence depresses quantum measures calculated from them. When these depressed rates are then used in a life table to obtain a mean age, this mean will contain an upward distortion. Since means of the 2nd kind are not distorted, the difference between the means of the 1st and 2nd kind equals the tempo effect (assuming the constant shape assumption holds).

Tempo effects in the period mean age at first birth

Figures 11-13 compare mean ages at first birth of the 1st and 2nd kind in the Czech Republic, the Netherlands, and Spain. In all three countries these means have risen, but the means of the 1st kind are higher than those of the 2nd kind. The difference between these means is as expected from the operation of the tempo effect. According to the theoretical argument presented earlier, the mean of the 1st kind is distorted because the numerators of rates of the first kind contain tempo effects. Means of the 2nd kind are not distorted because tempo effects in the numerators of rates of the 2nd kind are offset by tempo effects in their denominators. As a result, in years when the mean age at first birth is rising, tempo effects raise the mean of the 1st kind (based on hazard rates) above the mean of the 2nd kind (based on incidence rates). Note that these means are nearly equal to one another in the Czech Republic before 1990 and in the Netherlands after 1997. These are periods when the mean age at first birth did not change and as a result there are no tempo effects. In Spain such a convergence of the two kinds of means is not observed because the mean of the 2nd kind rises throughout the period plotted in Figure 13.

In support of the argument that the mean of the 2nd kind is not distorted, Figures 11-13 include the mean ages at first birth of successive cohorts. The cohort mean age at first birth $M(c)$ for a cohort born in year $c = t - M(c)$ is plotted at time t. This cohort mean age is close to the mean age of the 2nd kind in the Czech Republic and in Spain and falls between the means

of the 1st and 2nd kind in the Netherlands. Theoretical work by Rodriguez (in this volume) and Goldstein (in this volume) has proved that $M(c)$ for people born in year $t - MAE(t)$ equals the mean of the 2nd kind at time t when the translation assumptions hold. Since these assumptions do not hold exactly, $M(c)$ is not exactly equal to the observed mean of the 2nd kind, but the correspondence is good and clearly better than for means of the 1st kind.

Tempo effects in the period mean age at death (i.e., in life expectancy)

Figures 14-16 compare the period mean ages at death of the 1st and 2nd kind for Denmark, England and Wales, and Sweden (as before mortality under age 30 is assumed to be absent). The results are broadly similar to those for first birth: the means have risen over time and the mean of the 1st kind (i.e., period life expectancy) exceeds the mean of the 2nd kind. The difference between the two means again equals the tempo effect. In addition, the lagged cohort mean age at death is close to the mean age of the 2nd kind, which is as expected in a population in which the translation assumptions hold for adult death rates[8]. In sum, our conclusion that the period mean age at a life-cycle event calculated with standard life table methods is distorted by tempo effects is based on and supported by the following findings:

a. The theoretical analysis of the preceding sections shows that a rising mean age at an event depresses numbers of events in the numerators of period age-specific hazard rates. This inflates the calculated period mean ages calculated from these rates. Similarly, a falling mean age at an event inflates numbers of events in the numerators of period age-specific hazard rates and depresses the mean ages calculated from these rates by standard life table methods.
b. The observed period mean age based on hazard rates exceeds the period mean age based on incidence rates in populations in which the mean age is rising. Figures 11-13 demonstrate this for first birth and Figures 14-16 for death. This difference is due to a tempo distortion in the mean age based on hazard rates, because, as noted, the mean age based on incidence rates is not affected.
c. The differences between the period mean ages based on hazard and incidence rates (i.e., the tempo effect) disappear when the mean age stops rising. This is evident for means of the first birth in the Czech Republic before 1990 in Figure 11 and for the Netherlands after 1997 in Figure 12, as well as for the mean ages at death in Denmark in the mid-1990s in Figure 14. These results are of course as expected because when there is no change in the tempo of an event there should be no tempo effect.

[8] In the calculation of period and cohort tempo of mortality the risk of mortality under age 30 is set to zero, thus insuring the comparability of estimates.

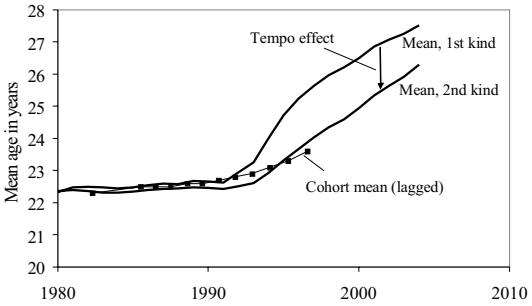


Fig. 11. Mean age at first birth. Means of 1st and 2nd kind and lagged cohort mean, Czech Republic.

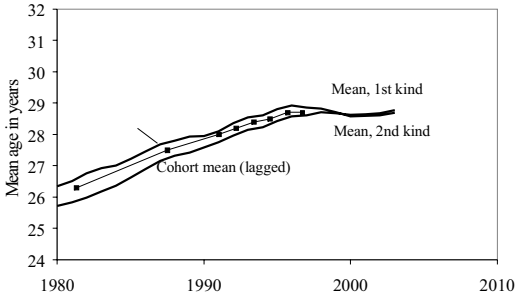
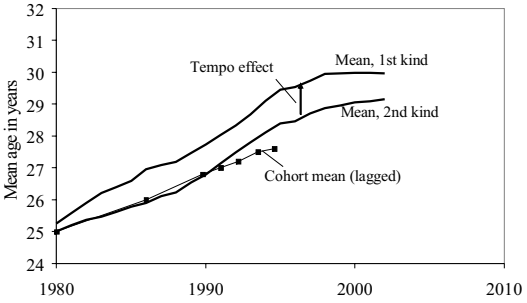


Fig. 12. Mean age at first birth. Means of 1st and 2nd kind and lagged cohort mean, the Netherlands.



Source: Sobotka,2004a; Sardon 2004b

Fig. 13. Mean age at first birth. Means of 1st and 2nd kind and lagged cohort mean, France.

d. The cohort mean age for a cohort born in year $c = t - M(c)$ is close to the period mean age of the 2nd kind. This is illustrated in Figures 11-16.

In addition, as shown below, the tempo-adjusted mean of the 1st kind is close to the observed mean of the 2nd kind.

A simple example of the mortality tempo effect

Since a tempo effect in life expectancy is a new and complex concept, we present a simple hypothetical example to demonstrate how the mortality tempo effect operates (Bongaarts and Feeney 2002, in this volume p. 11). Consider a stationary population with a life expectancy at birth of 70 years. Suppose further that a "life extension" pill is invented that defers the death of any person who consumes it by 3 months. If everyone in the population takes this pill on January 1 of year T, there will be no deaths during the first three months of the year. The number of deaths during this year is 25 per cent lower than it would have been without the pill, and the mean age at death is 70.25 years rather than 70 years. Since the pill's effect is the same at all ages, the level of the force of mortality function is also reduced by 25 per cent, and the age to which each value of the function is attached increases by 0.25 years. This change in the force of mortality function causes life expectancy at birth as conventionally calculated to rise to nearly 73 years for year T (see Figure 17).

In the following year, $T+1$, the number of deaths and the force of mortality function rise to the level observed before year T, but with values shifted forward to older ages by 0.25 years. Life expectancy at birth as conventionally calculated, having risen from 70 years prior to year T to nearly 73 years during year T, falls back to 70.25 years, as shown in Figure 17. This rise and fall in life expectancy at birth as conventionally calculated is a tempo distortion because it is at variance with the known trend in the mean length of life. Distortion of this kind occurs whenever the mean age at death changes.

This illustration demonstrates the operation of the tempo effect that distorts life expectancy under highly simplified hypothetical conditions. The example can be made more realistic in several ways. First, the life-extending pill can be taken year after year from year T onward. In that case, life expectancy will be distorted not only in year T but in every subsequent year as well. The mean age at death will rise over time and the observed life expectancy will continuously exceed the rising mean age at death due to the tempo effect. Second, the strength of the pill can vary from year to year, thus yielding tempo effects that also vary from year to year. Third, continuous change may be approximated by reducing intervals between pill taking while correspondingly reducing the pill's life-extending effect so that the annual "dose" remains the same. In the limit the mortality pattern in the hypothetical illustration approaches a real adult mortality pattern in a population in which the fixed-shape assumption holds. Subject to this assumption, improvements in adult

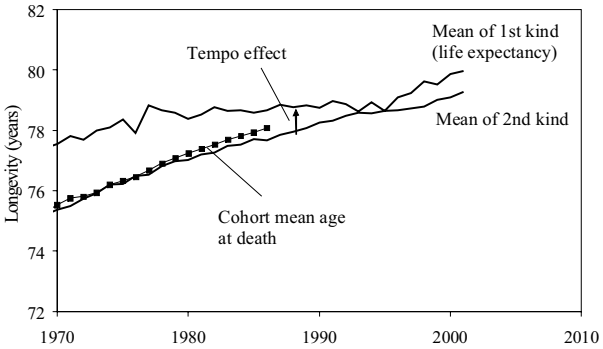


Fig. 14. Mean age at death. Means of the 1st and 2nd kind and lagged cohort mean. Females in Denmark. No mortality under age 30.

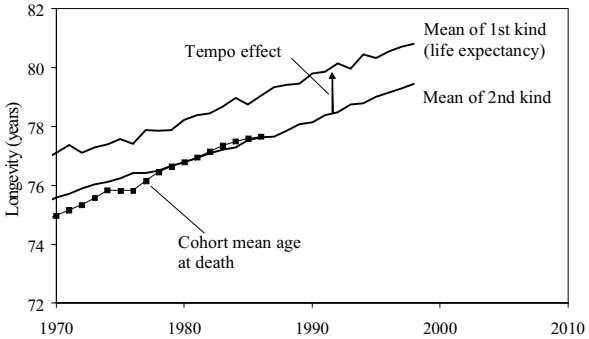
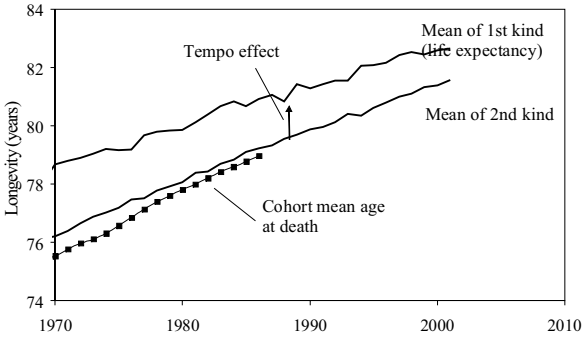


Fig. 15. Mean age at death. Means of the 1st and 2nd kind and lagged cohort mean. Females in England and Wales. No mortality under age 30.



Source: Estimated from data in Human Mortality Database (2005)

Fig. 16. Mean age at death. Means of the 1st and 2nd kind and lagged cohort mean. Females in Sweden. No mortality under age 30.

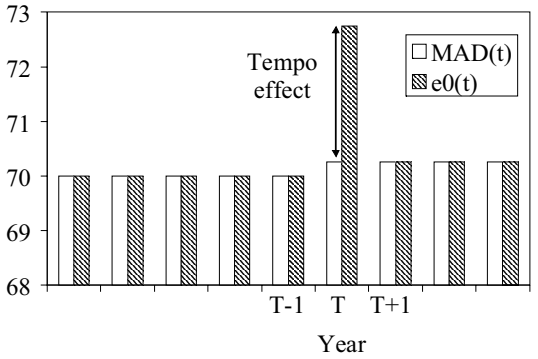


Fig. 17. Hypothetical illustration of effect of increase in mean age at death on conventionally calculated period life expectancy.

mortality can therefore be seen as resulting from the continuous provision of increments to life to all living individuals in every period, with the increments varying over time.

A similar illustration of the impact of a hypothetical "pill" to delay a birth could easily by provided, and it would show a similar tempo distortion of the mean age at birth calculated with a conventional life table.

4.3 Correcting tempo distortions

The method for removing tempo effects from rates of the 1st kind is the same as for rates of the 2nd kind: division of the numerators of the observed rates by the distortion index. Table 6 presents general equations for adjustment as well as applications to first births, first marriages, and deaths. Subject to a constant shape assumption[9], tempo distortions in summary measures of the 1st kind are removed by dividing the numerators of the hazard rates from which they are derived by $1 - r_p(t)$, where $r_p(t)$ denotes the rate of change in the period mean age of the event.

Observe that, for period measures of the 1st kind, tempo distortions occur for tempo as well as for quantum measures. This is in striking contrast to period measures of the 2nd kind, for which tempo measures are unaffected by tempo distortions if the constant shape assumption holds (because distortions in the numerator and denominator cancel out; see Table 4, right column). For this reason, tempo adjustments are best made using the rate of change in

[9] In the case of mortality, the constant shape assumption is applied to adult ages (30+) only. Bongaarts and Feeney (in this volume p. 11) demonstrate that for mortality the constant shape assumption is equivalent to assuming that the survival function $p(a,t)$ shifts to higher or lower ages as the mean age at death rises or falls over time and to assuming that the force of mortality $\mu(a,t)$ is proportional to the relative derivative of the survival function with respect to age.

the mean age of the 2nd kind to calculate the distortion index. Note that the procedure used here to make tempo adjustments is different from the one used by Kohler and Ortega (2002a), who rely on the rate of change in the mean age derived from the schedule of rates of the 1st kind. We believe that our approach is more accurate. Note also that if the translation assumption holds, the rates of change calculated from rates of the 1st and 2nd will be equal.

Table 6. Adjustments for tempo distortions in period quantum and tempo measures based on rates of the 1st kind.

Event	**Adjusted quantum** (total event rate)	**Adjusted tempo** (mean age at event)
General	$TER^*_L(t) = 1 - e^{-\int_0^\infty \frac{\mu(a,t)}{1-r_p(t)}\,da}$	$MAE^*_L(t) = \frac{1}{TER^*_L(t)} \int_0^\infty e^{\int_0^a \frac{\mu(a,t)}{1-r_p(t)}\,dx} da + \int_0^\infty TER^*_L(t) - 1\,da$
First birth	$TFR^*_L(t)$	$MAB^*_L(t)$
First marriage	$TNR^*_L(t)$	$MAM^*_L(t)$
Death	$TMR^*_L(t)$	$MAD^*_L(t)$

To illustrate the correction for tempo distortion in tempo measures, we apply the above procedure to mortality, to obtain a tempo-adjusted life expectancy (mean of the 1st kind). It follows that calculated life expectancy at birth may be adjusted for the tempo distortion by dividing the numerators of the observed age-specific death rates by $1 - r_p(t)$ and by using the resulting adjusted age-specific rates in the life table calculation (provided the constant shape assumption holds). This result is equivalent to substituting $TER^*_L(t) = TMR^*_L(t) = 1$ in the tempo equation in the top right cell of Table 6, giving the following tempo-adjusted life expectancy at birth

$$MAD^*_L(t) = e^*_0(t) = \int_0^\infty e^{-\int_0^a \frac{\mu(x,t)}{1-r_p(t)}\,dx}\,da\,, \tag{10}$$

where $r_p(t)$ denotes the rate of change in the period mean age at death $MAD(t)$. Because $1 - r_p(t) = TMR(t)$ (see formula (8) above), (10) may also be written as

$$MAD^*_L(t) = e^*_0(t) = \int_0^\infty e^{-\int_0^a \frac{\mu(x,t)}{TMR(t)}\,dx}\,da\,, \tag{11}$$

which gives more stable results in empirical application. The tempo distortion in the conventional life expectancy at birth equals the difference between $MAD_L(t)$ and $MAD^*_L(t)$.

Bongaarts and Feeney (in this volume p. 11) prove that the tempo-adjusted life expectancy at birth given by (10) or (11) equals the mean age at death calculated from rates of the 2nd kind (i.e., $MAD(t)$ in Table 2),

$$MAD^*_L = MAD(t)\,, \tag{12}$$

provided the constant shape assumption holds.

Table 7 shows empirical estimates for three alternative estimates of the mean age at death (average of annual values for 1970-1990, no mortality under age 30) for females in Denmark, England and Wales, and Sweden[10] :

$MAD(t)$, derived from rates of the 2nd kind (not distorted)
$MAD_L(t) = e_0(t)$, derived from rates of the 1st kind (distorted)
$MAD^*_L(t)$, derived from tempo-adjusted rates of the 1st kind (distortion corrected)

These results confirm that $MAD(t)$ and $MAD^*_L(t)$ have nearly the same value as predicted by (12). Table 7 also documents substantial tempo effects in the conventionally calculated life expectancy, $e_0(t) = MAD_L(t)$. The upward distortions in female life expectancy at birth for 1970-1990 are estimated at 1.5 years in Denmark, 1.4 years in England and Wales, and 1.9 years in Sweden. Using an indirect method Bongaarts and Feeney (2002) estimate a distortion of 3.3 years for Japan.

The preceding analysis has demonstrated that tempo-adjusted mortality tempo measures of the 1st and 2nd kind are equal under the constant shape assumption. As shown in Appendix B, this equality holds in general for both tempo-adjusted quantum and tempo measures for any life-cycle event whenever the observed proportion ever having experienced the event, $p(t)$, maintains its shape over time as the mean age at the event rises or falls over time. This condition holds approximately for adult mortality in contemporary low-mortality populations (Bongaarts and Feeney 2002, in this volume p. 11).

5 Conclusion

Demographers have developed a number of widely used methods to estimate the quantum and tempo of life-cycle events. The level of fertility, for example,

[10] The estimates of alternative measures of the mean age at death in Tables 7 and Figure 14-16 assume no tempo effects under age 30. For simplicity, life expectancy at birth is calculated as $e_0 = 30 + e_{30}$ and $e^*_0 = 30 + e^*_{30}$, ignoring mortality under age 30. In countries where mortality under age 30 is not small, we recommend the following more general equations for estimating observed and tempo-adjusted life expectancy:
$e_0 =_{30} L_0 + l_{30}e_{30} \quad e^*_0 =_{30} L_0 + l_{30}e^*_{30}$
Note also that Bongaarts and Feeney (in this volume p. 11) use yet another way to calculate the period mean age at death, as $\int_0^\infty p(a,t)\,da$, with $p(a,t)$ denoting the proportion of the cohort born at time $t-a$ who survive to age a. This estimate is identical to the variable called CAL, the cross-sectional average length of life, introduced by Brouard (1986) and Guillot (2003).

Table 7. Alternative estimates of the observed and tempo-adjusted period mean age at death: Average of annual estimates from 1970 to 1990 for females with no mortality under age 30[a].

	Mean age at death, females (average, 1970-1990)			
	$MAD(t)$ (from rates of the 2nd kind)	$MAD_L(t) = e_0(t)$ (from rates of the 1st kind)	$MAD^*_L(t) = e^*_0(t)$ (tempo-adjusted)	Tempo effect $MAD_L(t) - -MAD^*_L(t)$
Denmark	76.8	78.4	76.9	1.5
England and Wales	76.9	78.3	76.8	1.4
Sweden	78.2	80	78.1	1.9

Source: Bongaarts and Feeney 2002, in this volume p. 11. Death rates from University of California, Berkeley Mortality Data Base.
Note: a With no mortality under age 30, $e_0(t) = e_{30}(t) + 30$

is usually measured by the total fertility rate and the level of mortality by the life expectancy at birth. The wide availability, ease of interpretation, and up-to-date nature of these conventional period indicators have led to neglect of some of their deficiencies. Most analysts are aware of inaccuracies due to sampling error and incomplete vital registration, but they often neglect the pervasive influence of tempo distortions of many period indicators of life-cycle events.

Tempo distortions in period fertility measures were discovered more than half a century ago and are generally acknowledged. The post-war baby boom in the United States, for example, was due in part to a decline in the age at childbearing, and the recent low total fertility rates in many developed countries are in part due to delays in childbearing. This study argues that similar tempo distortions can occur in period measures of other life-cycle events, including marriage and death. This is the case even for measures derived from period life tables such as life expectancy at birth. These distortions are not generally recognised and are rarely if ever taken account of in empirical analysis.

Comparisons of period and cohort measures indicate that tempo distortions can be substantial in size. Distortions in the total fertility, marriage, and mortality rates of more than 10% were common during the 1980s. Using distorted age-specific death rates in a mortality life table leads to distorted estimates of life expectancy (typically exaggerated by 1-2 years).

The adjustment method proposed earlier by Bongaarts and Feeney is shown both by theoretical argument and by empirical example to be an effective, if approximate, solution to the problem of adjusting tempo and quantum measures for life-cycle events. Although this approach makes a simplifying assumption about changes over time in the age patterns of event rates, the results appear generally robust to deviations from this assumption.

The adjusted period tempo and quantum measures should be interpreted as variants of their conventional counterparts. The total fertility rate, for

example, is defined as the average number of births for a hypothetical cohort of women subjected throughout life to the age-specific birth rates observed in a given year. This is a hypothetical rate because no actual cohort will experience these observed period birth rates. The tempo-adjusted total fertility rate is a similarly hypothetical measure, but one that corrects for distortions caused by year-to-year tempo changes. Neither the observed nor the adjusted total fertility rate attempts to estimate the fertility rate of any actual cohort, nor do they attempt any prediction of future fertility. The goal of the tempo adjustment is simply to provide period quantum and tempo measures that are free of the tempo distortions in conventional measures.

Adjusted period measures are hypothetical in that they tell us what the observed period measure would have been if there had been no change in the timing of the event. This hypothetical measure does not correspond to the behaviour of a particular cohort because the translation assumptions are often violated. However, as we have seen in this chapter, the patterns of change observed in practice are often close enough to the translation assumptions that the adjusted period measures are approximate measures of lagged cohorts. Furthermore, the adjusted period measures give us an indication of what rates could be like in the future if postponement comes to an end.

Distorted views of past levels and trends in the quantum and tempo of life-cycle events may lead to misleading projections and to the adoption of sub-optimal social and health policies. It is therefore desirable for analysts to understand the strengths and weaknesses of period indicators of life-cycle events and to recognise and correct tempo distortions.

References

Bongaarts, J. (1999a). The fertility impact of changes in the timing of childbearing in the developing world. *Population Studies*, 53:277–89.

Bongaarts, J. (1999b). Fertility decline in the developed world: Where will it end? *American Economic Review*, 89(2):256–260.

Bongaarts, J. (2002). The end of the fertility transition in the developed world. *Population and Development Review*, 28(3):419–433.

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. (1998a). On the quantum and tempo of fertility. *Population and Development Review*, 24(2):271–291.

Bongaarts, J. and Feeney, G. (1998b). On the quantum and tempo of fertility: A reply. *Population and Development Review*, 26(3):560–564.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Brass, W. (1974). Perspectives in population prediction: Illustrated by the statistics of England and Wales. *Journal of the Royal Statistical Society A*, 137:55–72.

Brouard, N. (1986). Structure et dynamique des populations. la pyramide des années a vivre, aspects nationaux et exemples régionaux. *Espace-Populations-Sociétés*, 2(14-5):157–68.

Council of Europe (2002). Recent demographic developments in Europe 2002. Technical report, Council of Europe Publishing, Strasbourg.

Database, H. M. (2005). Data downloaded from www.mortality.org in July 2005.

Feeney, G., Wang, F., Zhou, M., and Xiao, B. (1989). Recent fertility dynamics in China: Results from the 1987 one percent population survey. *Population and Development Review*, 15:297–322.

Feeney, G. and Yu., J. (1987). Period parity progression measures of fertility in China. *Population Studies*, 41:77–102.

Foster, A. (1990). Cohort analysis and demographic translation: A comparative study of recent trends in age specific fertility rates from Europe and North America. *Population Studies*, 44:287–315.

Goldstein, J. Found in translation? a cohort perspective on tempo-adjusted life expectancy. *In this volume, also published in Demographic Research*, 14(5):71–84. 2006.

Goldstein, J., Lutz, W., and Scherbov, S. (2003). Long-term population decline in Europe: The relative importance of tempo effects and generational length. *Population and Development Review*, 29(4):699–707.

Goldstein, J. R. (2003). Late but not never: The tempo and quantum of first marriage in France. Unpublished manuscript,Office of Population Research, Princeton University.

Guillot, M. (2003). The cross-sectional average length of life (*CAL*): A cross-sectional mortality measure that reflects the experience of cohorts. *Population Studies*, 57(1):41–54.

Hajnal, J. (1947). The analysis of birth statistics in the light of the recent international recovery of the birth-rate. *Population Studies*, 1:137–164.

Hajnal, J. (1953). Age at marriage and proportions marrying. *Population Studies*, 7:111–136.

Hamilton, B. E., Sutton, P. D., and Ventura, S. J. (2003). Revised birth and fertility rates for the 1990s and new rates for Hispanic populations, 2000 and 2001: United States. *National Vital Statistics Reports*, 51:1–96.

Henry, L. (1972). Démographie: Analyse et modles. Paris: Librairie Larousse.

Henry, L. (1980). Fertility of marriages: A new method of measurement. Population Studies Translation Series, No. 3. New York: United Nations. Originally published 1953.

Heuser, R. L. (1976). Fertility tables for birth cohorts by color: United States 1917-73. Technical Report HRA, DHEW Publication. Rockville, MD: National Center for Health Statistics.

Hoem, B. and Hoem, J. M. (1999). Fertility trends in sweden up to 1996. *Population Bulletin (United Nations)*, 40/41:318–333.

Keilman, N. (1994). Translation formulae for non-repeatable events. *Population Studies*, 48(2):341–357.

Kim, Y. and Schoen, R. (2000). On the quantum and tempo of fertility: Limits to the Bongaarts-Feeney adjustment. *Population and Development Review*, 26(3):554–59.

Kohler, H.-P. and Ortega, J. A. (2002a). Tempo-adjusted period parity progression measures, fertility postponement and completed cohort fertility. *Demographic Research*, 6:92–144. www.demographic-research.org.

Kohler, H.-P. and Ortega, J. A. (2002b). Tempo-adjusted period parity progression measures: Assessing the implications of delayed childbearing for cohort fertility in Sweden, the Netherlands and Spain. *Demographic Research*, 6:146–190. www.demographic-research.org.

Kohler, H.-P. and Ortega, J. A. (2004). Old insights and new approaches: Fertility analysis and tempo adjustment in the age-parity model. *Vienna Yearbook of Population Research*, pages 55–87.

Kohler, H.-P. and Philipov, D. (2001). Variance effects in the Bongaarts-Feeney formula. *Demography*, 38(1-16).

Lesthaeghe, R. and Willems, P. (1999). Is low fertility a temporary phenomenon in the European Union? *Population and Development Review*, 25(2):211–228.

Ní Bhrolcháin, M. (1987). Period parity progression ratios and birth intervals in England and Wales, 1941-1971: A synthetic life table analysis. *Population Studies*, 41:103–125.

Ní Bhrolcháin, M. (1992). Period paramount? a critique of the cohort approach to fertility. *Population and Development Review*, 18:599–629.

Page, H. J. (1977). Patterns underlying fertility schedules: A decomposition by both age and marriage duration. *Population Studies*, 30:85–106.

Philipov, D. and Kohler, H.-P. (2001). Tempo effects in the fertility decline in Eastern Europe: Evidence from Bulgaria, the Czech Republic, Hungary, Poland and Russia. *European Journal of Population*, 17(1):37–60.

Preston, S. H., Heuveline, P., and Guillot, M. (2001). *Demography: Measuring and Modeling Population Processes*. Blackwell, Malden, MA.

Pullum, T. W. (1980). Separating age, period and cohort effects in white US fertility, 1920-70. *Social Science Research*, 9:225–244.

Rallu, J.-L. and Toulemon, L. (1994). Period fertility measures: The construction of different indices and their application to France, 1946-89. *Population: An English Selection*, 6:59–94.

Rodriguez, G. Demographic translation and tempo effects: An accelerated failure time perspective. *In this volume, also published in Demographic Research*, 14(6):85–110. 2006.

Ryder, N. B. (1956). Problems of trend determination during a transition in fertility. *Milbank Memorial Fund Quarterly*, 34:5–21.

Ryder, N. B. (1959). An appraisal of fertility trends in the United States. In *Thirty Years of Research in Human Fertility: Retrospect and Prospect*, pages 38–49. Milbank Memorial Fund, New York.

Ryder, N. B. (1964). The process of demographic translation. *Demography*, 1:74–82.

Ryder, N. B. (1980). Components of temporal variations in American fertility. In Hiorns, R. W., editor, *Demographic Patterns in Developed Societies*, pages 15–54. Taylor & Francis, London.

Ryder, N. B. (1983). Cohort and period measures of changing fertility. In Bulatao, R. A. and Lee, R. D., editors, *Determinants of Fertility in Developing Countries*, volume 2, pages 737–756. Academic Press, New York.

Ryder, N. B. (1986). Observations on the history of cohort fertility in the United States. *Population and Development Review*, 12:617–643.

Sardon, J. P. (1993). Un indicateur conjoncturel de mortalité: l'exemple de la France. *Population*, 48(2):347–368.

Sardon, J. P. (1994). A period measure of mortality. the example of France. *Population: An English Selection*, 6:131–150.

Sardon, J. P. (2000). The demographic situation of Europe and the developed countries overseas. *Population: An English Selection*, 12:293–328.

Sardon, J. P. (2004a). Unpublished special tabulations from data bank of Observatoire Démographique Européen provided to John Bongaarts on January 15.

Sardon, J. P. (2004b). Recent demographic trends in the developed countries. *Population: An English Selection*, 59(2):263–314.

Sato, R. (2001). Unpublished special tabulations from japanese vital registration data provided to john bongaarts.

Shryock, H. S. and Siegel, J. S. (1973). *The Methods and Materials of Demography. US Bureau of the Census.* US Government Printing Office, Washington, DC.

Smallwood, S. (2002). The effects of changes in timing of childbearing on measuring fertility in England and Wales. *Population Trends*, 109:36–45.

Sobotka, T. (2003). Tempo-quantum and period-cohort interplay in fertility changes in Europe. Evidence from the Czech Republic, Italy, the Netherlands and Sweden. *Demographic Research*, 8:152–214. www.demographic-research.org.

Sobotka, T. (2004a). *Postponement of childbearing and low fertility in Europe.* Doctoral thesis, University of Groningen. Dutch University Press, Amsterdam, xiv + 298 pp.

Sobotka, T. (2004b). Is lowest-low fertility in Europe explained by the postponement of childbearing? *Population and Development Review*, 30(2):195–220.

Van Imhoff, E. (2001). On the impossibility of inferring cohort fertility measures from period fertility measures. *Demographic Research*, 5(2):23–60.

Van Imhoff, E. and Keilman, N. (2000). On the quantum and tempo of fertility: Comment. *Population and Development Review*, 26(3):549–553.

Whelpton, P. K. (1954). *Cohort Fertility: Native White Women in the United States*. Princeton University Press, Princeton, NJ. [Reissued 1973, Port Washington, NY: Kennikat Press.].

Winkler-Dworak, M. and Engelhardt, H. (2004). On the tempo and quantum of first marriages in Austria, Germany, and Switzerland. *Demographic Research*, 10(9):231–263.

Zeng, Y. and Land., K. C. (2001). A sensitivity analysis of the Bongaarts-Feeney method for adjusting bias in observed period total fertility rates. *Demography*, 38:17–28.

Zeng, Y. and Land, K. C. (2002). Adjusting period tempo changes with an extension of Ryder'sbasic translation equation. *Demography*, 39:269–285.

Appendix A: Relationship between the completed fertility rate and the weighted average of tempo-adjusted period total fertility rates

Let age-specific fertility rates at time t and age a be denoted $d(a,t)$. The total fertility rate equals

$$TFR(t) = \int d(a,t)\, da \tag{1a}$$

The distribution of fertility by age at time t is denoted $f(a,t)$:

$$f(a,t) = \frac{d(a,t)}{TFR(t)} \tag{2a}$$

so that $\int f(a,t)\, da = 1$ and $d(a,t) = TFR(t) f(a,t)$.
The completed fertility rate for the cohort born in year t_0 equals

$$CFR(t_0) = \int d(a, t_0 + a)\, da = \int TFR(t_0 + a) f(a, t_0 + a)\, da \tag{3a}$$

rearranging (6) yields

$$TFR(t) = [1 - r_p(t)]\, TFR^*(t) \tag{4a}$$

and substitution of (4a) in (3a) gives

$$\begin{aligned} CFR(t_0) &= \int TFR^*(t_0 + a)[1 - r_p(t_0 + a)] f(a, t_0 + a)\, da \\ &= \int TFR^*(t_0 + a)\nu(a, t_0)\, da \end{aligned} \tag{5a}$$

where $\nu(a, t_0) = [1 - r_p(t_0 + a)] f(a, t_0 + a)$.
The weighted average of $TFR^*(t)$ is defined as

$$\overline{TFR}(t_0) = \frac{\int TFR^*(t_0 + a)\nu(a, t_0)\, da}{\int \nu(a, t_0)\, da} = \int TFR^*(t_0 + a)\omega(a, t_0)\, da \quad (6a)$$

where $\omega(a, t_0) = \frac{\nu(a,t_0)}{\int \nu(a,t_0)\, da}$
It follows from (5a) and (6a) that

$$CFR(t_0) = \overline{TFR}(t_0) \int \nu(a, t_0)\, da \quad (7a)$$

Equations (5a), (6a), and (7a) hold in general and do not require any simplifying assumptions. However, it can be shown that $\int \nu(a, t_0)\, da = 1$ and $\omega(a, t_0) = \nu(a, t_0)$ when the constant shape assumption holds. In that case $CFR(t_0) = \overline{TFR}(t_0)$.

Appendix B: Comparison of measures of the 1st and 2nd kind

If age-specific rates change without conditions, then period quantum and tempo measures of the 1st kind generally differ from measures of the 2nd kind. We will now demonstrate that this difference between measures of the 1st and 2nd kind disappears if the tempo effect is removed and if the shape of the proportion ever having experienced the event remains invariant as the mean age at the event changes. Holding the shape of $p(t)$ constant implies

$$p(a, t) = p(a - S(t), 0) \text{ for } a \geq S(t) \text{ and } p(a, t) = 1 \text{ for } a < S(t) \quad (1b)$$

where $S(t)$ is equal to the amount of the shift since $t = 0$. As shown by Bongaarts and Feeney (2002, in this volume p. 11) (1b) implies that

$$d(a, t) = [1 - r_p(t)]\frac{-\partial p(a, t)}{\partial a} \quad (2b)$$

and

$$\mu(a, t) = [1 - r_p(t)]\frac{\frac{-\partial p(a,t)}{\partial a}}{p(a, t)} \quad (3b)$$

Let the tempo-adjusted versions of $\mu(a, t)$ and $d(a, t)$ be denoted $\mu^*(a, t)$ and $d^*(a, t)$ respectively; then

$$d^*(a, t) = \frac{d(a, t)}{1 - r_p(t)} = \frac{-\partial p(a, t)}{\partial a} \quad (4b)$$

and

$$\mu^*(a, t) = \frac{d^*(a, t)}{p(a, t)} = \frac{\frac{-\partial p(a,t)}{\partial a}}{p(a, t)} \quad (5b)$$

It follows from (4b) and (5b) that

$$p(a,t) = 1 - \int_0^a d^*(x,t)\,dx = 1 - e^{-\int_0^a \mu^*(x,t)\,dx} \tag{6b}$$

Rearranging (6b) and integrating to m, the highest age at which the event is observed, gives

$$e^{-\int_0^m \mu^*(x,t)\,dx} p(a) = 1 - p(m,t) \tag{7b}$$

and

$$\int_0^m d^*(x)\,dx = 1 - p(m,t) \tag{8b}$$

Substitution of (7b) and (8b) in the equations for tempo-adjusted quantum of the 1st and 2nd kind (from Tables 4 and 6 respectively) shows that the tempo-adjusted quantum of the 1st kind

$$TER_L^*(t) = 1 - e^{-\int_0^m \frac{\mu(a,t)}{1-r_p(t)}\,da} = 1 - e^{-\int_0^m \mu^*(a,t)\,da} = 1 - p(m,t) \tag{9b}$$

equals the tempo-adjusted quantum of the 2nd kind

$$TER_L^*(t) = \int_0^m \frac{d(a,t)}{1-r_p(t)}\,da = \int_0^m d^*(a,t)\,da = 1 - p(m,t) \tag{10b}$$

Similarly, the tempo-adjusted mean age of the 1st kind

$$\begin{aligned} MAE_L^*(t) &= \frac{1}{TER_L^*(t)} \int_0^m e^{-\int_0^a \frac{\mu(x,t)}{1-r_p(t)}\,dx} + TER_L^*(t) - 1\,da \\ &= \frac{1}{1-p(m,t)} \int_0^m e^{-\int_0^a \mu^*(x,t)\,da} - p(m,t)\,da \\ &= \frac{1}{1-p(m,t)} \int_0^m p(a,t) - p(m,t)\,da \end{aligned} \tag{11b}$$

equals the tempo-adjusted mean age of the second kind

$$\begin{aligned} MAE^*(t) &= \frac{1}{TER_L^*(t)} \int_0^m \frac{a d(a,t)}{1-r_p(t)}\,da \\ &= \frac{1}{1-p(m,t)} \int_0^m a d^*(a,t)\,da \\ &= \frac{1}{1-p(m,t)} \int_0^m a \frac{\partial p(a,t)}{\partial a}\,da \\ &= \frac{1}{1-p(m,t)} \int_0^m p(a,t) - p(m,t)\,da \end{aligned} \tag{12b}$$

Whenever (1b) holds, tempo-adjusted quantum and tempo measures of the 1st and 2nd kind are equal to one another.

The finding that $TER^*_L(t) = TER^*(t)$ and $MAE^*_L(t) = MAE^*(t)$ is of interest because it implies that quantum and tempo measures of the first kind are equal to those of second kind even when the age-specific proportions of individuals exposed to the risk of an event are changing, provided (1b) holds. Bongaarts and Feeney (in this volume p. 11) have examined this assumption for mortality and found that it provides a good approximation of reality in recent decades among adults in countries with low mortality. But the assumption is probably less applicable to the fertility and nuptiality processes in which quantum changes can occur at the same time as tempo changes. Further research is required on the implication of deviations from this assumption.

II. Critiques, extensions and applications of the mortality tempo effect

Demographic translation and tempo effects: An accelerated failure time perspective*

Germán Rodríguez

Office of Population Research, Princeton University, 241 Wallace Hall, Princeton, NJ 08544, USA. E-mail: grodri@princeton.edu

Summary. In this chapter I review the concept of tempo effects in demography, focusing on the tempo adjustments proposed by Bongaarts and Feeney and drawing on the work of Ryder and Zeng and Land. I show that the period-shift model that underlies the proposed adjustments can be motivated from an accelerated failure time cohort perspective. I propose alternative measures of tempo under changing fertility and mortality that share a synthetic cohort interpretation with the adjusted measure of quantum. I stress similarities between the results for fertility and mortality, particularly in terms of mean age of childbearing and mean age at death, but also note some important distinctions. I conclude that the fertility adjustments can help distinguish quantum and tempo effects, but argue that in the case of mortality the Bongaarts-Feeney measure of tempo-adjusted life expectancy differs from conventional estimates because if reflects past mortality.

1 Introduction

How long do we live? According to the U.S. National Center for Health Statistics, "in 2002 the overall expectation of life at birth was 77.3 years"(Arias, 2004). The center makes clear that this measure represents "what would happen to a hypothetical (or synthetic) cohort if it experienced throughout its entire life the mortality conditions of a particular period in time", in this case 2002. In real life a child born in the U.S. in 2002 would probably live longer than 77.3 years on average, because we expect mortality to improve in the future.

Bongaarts and Feeney (2002, in this volume p. 11 and p. 29) have challenged the conventional wisdom, and created quite a stir in the demographic community, by postulating the existence of mortality "tempo effects" that bias standard measures of longevity, such as the period life expectancy, whenever mortality is changing. The measures are believed to be biased upwards when

expectation of life is increasing, so we don't live as long as we think. Bongaarts and Feeney (in this volume p. 11) note that "[e]stimates of the effect for females in three countries with high and rising life expectancy range from 1.6 yr in the U.S. and Sweden to 2.4 yr in France for the period 1980-1995".

The concept of tempo distortion originated in the field of fertility analysis, where one can draw a clear distinction between quantum and tempo, and refers to the fact that a reduction in period rates could be caused by delays in childbearing without any changes in completed cohort family size. Many demographers have found the extension of these ideas to mortality baffling because a reduction in period mortality rates can only mean that people will die later. With mortality the quantum is fixed, only tempo can change, and no one would mistake one for the other.

It is, of course, possible for cohort and period summaries of age-specific mortality rates to differ. But Bongaarts and Feeney (in this volume p. 11) make the stronger claim that "tempo effects distort both the observed death rates and the corresponding life expectancy". It is also quite likely that mortality rates are distorted by unobserved heterogeneity, particularly at old ages, but Vaupel (2002) reports that Bongaarts believes that "tempo effects can distort mortality in homogeneous populations".

Like others I have gone over the underlying mathematical argument and have found no fault. But I come up with a different interpretation of the Bongaarts-Feeney results. I show that working strictly within their framework, one can produce an estimate of expectation of life when mortality is declining that is *higher*, not lower, than the conventional estimate. This differs, of course, from the Bongaarts-Feeney adjustment, and I hope the argument will clarify exactly why this is the case. As Wachter (in this volume) has noted "every measure measures something", and we are just measuring different things. Specifically, I will argue that their measure combines the observed force of mortality with features of the age distribution that reflect past rather than current mortality.

Because so much of the work builds upon earlier results on fertility I start with a brief review of Ryder's (1964) famous translation formula. My main goal is to clarify its intent and the conditions under which it is valid. I then review the Bongaarts-Feeney (1998) tempo-adjusted total fertility rate and a synthetic-cohort interpretation due to Zeng and Land (2001, 2002). I show that the period-shift fertility model used by Bongaarts and Feeney can be motivated in terms of a cohort-delay model where the passage of time slows down. I then obtain a measure of mean age of childbearing under changing tempo that complements the Bongaarts-Feeney tempo-adjusted total fertility rate, yet differs from their tempo estimate.

Having laid the groundwork in the field of fertility, where these ideas are less controversial, I move to the field of mortality. I mention briefly why Ryder (1964) didn't pursue a translation formula for mortality, as well as how one might go about it knowing what we know today. I then turn to the Bongaarts-Feeney framework showing how their period-shift mortality model results from

a slowing down of time in an accelerated-failure-time framework. I then discuss, and I hope explicate, the various measures of longevity that have been proposed, noting how some of these indices depend on the past via the age structure. I also derive a synthetic cohort measure of life expectancy under changing mortality that provides an exact analog of the measure of fertility tempo derived earlier, yet differs substantially from the Bongaarts-Feeney tempo-adjusted measure of life expectancy.

While most of the chapter emphasizes parallels between the analysis of fertility and mortality, in the discussion I return to some of the fundamental differences noted at the outset. In the case of fertility we have recurrent events where a distinction between quantum and tempo is meaningful and, more importantly, adjustments can be useful in determining the extent to which period changes reflect quantum or tempo effects. In the case of mortality trends have an unambiguous interpretation as tempo effects. The fact that the proposed adjusted measures differ from conventional life expectancy is not due to a bias or distortion, but simply to the fact that they measure different things. Specifically, conventional life expectancy depends only on the force of mortality, whereas the adjusted measures are affected by age composition and thus past mortality.

2 Fertility

Let us consider a surface of age-period fertility rates where $f(a,t)$ is the fertility rate at age a and time t. This rate pertains both to period t, and to the cohort born at time $t-a$.

2.1 Translating fertility

Ryder (1964) was interested in the relative strengths and weaknesses of cohort and period summaries of these rates. Useful summaries for the cohort born at time t include the average number of children per woman, $TFR_c(t)$, a measure of the *quantum* of fertility, and the mean age of childbearing $\mu_c(t)$, a measure of the *tempo* of fertility, defined as

$$TFR_c(t) = \int f(a,t+a)\,da \quad \text{and} \quad \mu_c(t) = \int a f(a,t+a)\,da / TFR_c(t). \tag{1}$$

Together these indices tell us whether women have more or fewer children, and whether they have them earlier or later in life.

The aggregates can also be computed for periods, and are usually interpreted in terms of a synthetic cohort that goes through life bearing children at the current observed rates. The synthetic cohort representing period t has $TFR_p(t)$ children at an average age of $\mu_p(t)$ where

$$TFR_p(t) = \int f(a,t)\,da \quad \text{and} \quad \mu_p(t) = \int a f(a,t)\,da / TFR_p(t). \tag{2}$$

Ryder's chief concern was that period summaries provide a distorted view of the behavior of cohorts when fertility is changing, and he was able to formalize this view in a remarkable result.

Ryder (1964) assumes that $f(a,t)$ may be expanded in a Taylor series separately for each age. The most useful result is obtained by expanding rates for the cohort which is now at its mean age of childbearing and ignoring terms beyond the first derivative. If the cohort of interest has mean age of childbearing μ, and was thus born at $t-\mu$, we have

$$f(a, t-\mu+a) \approx f(a,t) + (a-\mu)f'(a,t). \tag{3}$$

Under this approximation Ryder obtained the following relationship between cohort and period TFRs:

$$TFR_c(t-\mu) = \frac{TFR_p(t)}{1-r_c}, \tag{4}$$

where r_c is the time derivative or rate of change of *cohort* mean age of childbearing at time $t-\mu$.

This remarkable formula shows that if cohorts postpone childbearing then, to a first order of approximation, the period TFR will fall *below* the cohort *TFR* (for the cohort at its mean childbearing age) by an amount that depends on how fast the mean age of childbearing is increasing. If mean age of childbearing is decreasing then the period *TFR* will rise *above* the corresponding cohort *TFR*. This in fact happened during the baby boom, when period TFRs rose to levels that exceeded the completed fertility of all active cohorts (Ryder, 1964; Schoen, 2004).

It is important to note that Ryder's result relies solely on a first-order Taylor series approximation to the rates at each age. Contrary to popular belief, there is no assumption that the shape of the period or cohort schedules is constant, or that the cohort and period *TFR*s are constant. To see this point note that one can generate rates $f(a,t)$ that satisfy the assumption of linearity by interpolating between any two arbitrary age schedules $f(a,0)$ and $f(a,\tau)$.

Ryder (1964) also considered a translation procedure for mean age of childbearing, introducing a second type of formula with stronger assumptions (which may account for some of the confusion). We will not pursue this development further because it is not central to the argument that follows, except to note Ryder's conclusion that "the period mean is a distorted version of the cohort mean" when quantum is changing, "just as the period sum is a distorted version of the cohort sum" when tempo is changing.

2.2 Tempo-adjusted fertility

Bongaarts and Feeney (1998) proposed a tempo-adjusted total fertility rate, usually denoted TFR^*, based on an expression that looks remarkably like Ryder's translation formula:

$$TFR^*(t) = \frac{TFR_p(t)}{1 - r_p(t)}. \tag{5}$$

There are, however, two subtle but important differences. First, the $r_p(t)$ on the right-hand-side is the rate of change in the *period*, not the cohort, mean age of childbearing at time t. This is much easier to calculate from available data. Second, TFR^* is not a cohort rate, but rather a pure-period measure representing tempo-corrected fertility, as we will see presently.

A third difference I should mention is that Bongaarts and Feeney recommend applying their procedure separately by birth order, using rates that divide births of a given order by all women. I ignore this breakdown to keep the argument simple. (I also believe that parity-specific fertility is best analyzed using hazard rates where births of order k are divided by women at parity $k-1$, but that's an argument best left for another time; see van Imhoff and Keilman (2000) and the rejoinder by Bongaarts and Feeney (2000).)

We will derive the adjustment in Equation 5 considering a situation where all cohorts start delaying fertility at the same time and rate without reducing their completed family size. The situation where quantum is fixed is simpler– and more relevant to the analysis of mortality—than where quantum is changing as well, although the Bongaarts-Feeney adjustment can be applied in both cases. The assumption of a constant rate also simplifies things, in particular it leads to explicit cohort results, although Equation 5 can also be applied when the rate of change varies over time.

It will be useful to introduce a function $F(a,t)$ representing the cumulative fertility or average parity of women age a at time t (the cohort born at time $t-a$). This schedule can be obtained as a *cohort* integral, by accumulating fertility along a diagonal of the Lexis diagram:

$$F(a,t) = \int_0^a f(x, t-a+x)\, dx. \tag{6}$$

The age-period specific rates $f(a,t)$ are the *cohort* derivatives of these rates, and can be recovered by differentiating $F(a, c+a)$ with respect to a, i.e. with respect to both age and time.

Let us also introduce a fertility schedule $f_0(a)$ with corresponding cumulative schedule $F_0(a)$, total fertility rate $TFR_0 = \int f_0(a)\, da$ and mean age of childbearing $\mu_0 = \int a f_0(a) da / TFR_0$. This baseline schedule will represent the situation at time zero, so that $F(a,0) = F_0(a)$. If fertility has been constant for a long time we could view all rates prior to time zero as generated by the baseline schedule, but this assumption is not necessary for the developments that follow. All we need is the assumption that just before time zero women were following the cumulative schedule $F_0(a)$.

Now suppose that at time zero all cohorts *slow down* their pace of childbearing at the same rate r. Let us give this statement a precise meaning. The cohort that has reached average parity $F_0(a)$ at age a and time zero, and would have been expected to reach parity $F_0(a+1)$ a year later, will

instead climb only as far as $F_0(a+1-r)$. This is similar to taking a pill that prevents all births (and stops a woman's biological clock) for a fraction r of the year, but I prefer to work in continuous time. The same idea is used in Coale's (1971) classic nuptiality model, where he speeds up or slows down the Swedish schedule of first marriages. The device of accelerating or slowing down the passage of time is also used in survival analysis, as we will see in Section 3.

It turns out that this slowing down of time is exactly equivalent to a period shift in the cumulative fertility schedule, so that

$$F(a,t) = F_0(a-rt), \qquad t \geq 0. \tag{7}$$

For example the cohort age a at time zero had parity $F(a,0) = F_0(a)$ and will now move to $F(a+1,1) = F_0(a+1-r)$.

If we now take *cohort* derivatives, differentiating with respect to both age and time (which of course vary together for a cohort) we obtain

$$f(a,t) = f_0(a-rt)(1-r), \quad t \geq 0. \tag{8}$$

This shows that when all cohorts slow down the pace of childbearing at the same rate r the age-specific rates are instantly deflated by a factor $1-r$ and start shifting to older ages.

The simplest way to prove Equation 8 is to write the period-shift model for a cohort that reaches age a at time $t = c + a > 0$, which is

$$F(a, c+a) = F_0(a - r(c+a)) = F_0(a(1-r) - rc), \tag{9}$$

and then take derivatives with respect to a for fixed c to obtain

$$f(a, c+a) = f_0(a(1-r) - rc)(1-r) = f_0(a - r(c+a))(1-r). \tag{10}$$

Integrating the period schedule in Equation 8 over a for fixed t we obtain the period *TFR*, and we can also obtain the period mean age of childbearing. As long as the cumulative schedule continues to shift at a rate r,

$$TFR_p(t) = TFR_0(1-r) \quad \text{and} \quad \mu_p(t) = \mu_0 + rt. \tag{11}$$

The period *TFR* declines at time zero by a factor $1-r$ as a result of the delay. This could be misinterpreted as a change in the quantum of fertility when in fact it is a pure tempo effect. The fact that the derivative of period mean age of childbearing is r provides an ingenious way to recover the baseline *TFR* simply dividing by $1-r$, which leads to the Bongaarts-Feeney formula 5. The key assumption required is that all cohorts delay fertility at the same time and rate.

This leads to a direct interpretation of the tempo-adjusted *TFR* as a *counterfactual* measure; paraphrasing Bongaarts and Feeney (1998), it provides an estimate of what the period *TFR* would have been if cohorts had not delayed childbearing at time t. Note that this is indeed a pure period measure

as claimed; it estimates TFR_0, which does not correspond to the completed family size of any real cohort unless fertility has been constant for the last thirty five years or so. It can, however, be interpreted as the completed family size of a synthetic cohort, as we will see below.

It is interesting to note that Bongaarts and Feeney adjust the quantum but not the tempo of fertility, considering the mean age of childbearing unaffected by tempo distortions. This can be seen to be the case in the present framework because $\mu_p(0) = \mu_0$, a result that obtains because the factor $1-r$ appears both in the numerator and the denominator of the mean. Delays affect the mean age of childbearing only after time zero. This point will be quite important when we turn to an analysis of mortality.

2.3 A synthetic cohort interpretation

In the previous section we focused on period measures. Let us now consider what happens to the cohort that starts childbearing at time zero, when the passage of time slows down. Let a_0 denote the lowest age of childbearing, so the cohort in question was born at time $-a_0$. From Equation 8, we see that this cohort would follow the schedule

$$f^\dagger(a) = f_0(a - r(a - a_0))(1 - r) = f_0(a(1 - r) + ra_0)(1 - r). \tag{12}$$

Integrating this expression over all ages a we find the total fertility rate for this cohort to be

$$TFR^\dagger = \int f_0(a(1 - r) + ra_0)(1 - r)\, da = TFR_0, \tag{13}$$

where the results follows by changing variables from a to $y = a(1 - r) + ra_0$ and noting that the Jacobian $da/dy = 1/(1 - r)$ cancels out the multiplier $1 - r$. This result is due to Zeng and Land (2001), who provide a simplified derivation of the Bongaarts-Feeney adjustment.

Because $TFR^\dagger = TFR^*$, the Zeng-Land approach leads to an interesting interpretation of the Bongaarts-Feeney measure in synthetic cohort terms, as the number of children that a cohort would have under current *conditions*, if by that we mean the current rates and the fact that they are shifting to older ages at a constant rate r.

The corresponding mean age of childbearing for this cohort can easily be obtained using the same change of variables technique, but appears to have been overlooked in the literature:

$$\mu^\dagger = \int a f_0(a(1 - r) + ra_0)(1 - r)\, da / TFR_0 = \frac{\mu_0 - ra_0}{1 - r}. \tag{14}$$

The notation could be streamlined considerably if we measured age from a_0 as done by Zeng and Land (2001), in which case Equation 14 would simplify to $\mu^\dagger = \mu_0/(1 - r)$ and we would have the remarkable result that under a

period shift the quantum and tempo of fertility are affected exactly the same way.

Bongaarts and Feeney (1998) argue that TFR^* removes a tempo distortion from TFR, and one could make the point that $\mu^\dagger$ removes a tempo distortion from μ. I prefer the more neutral view that the two sets of indices measure different things: TFR (and μ) tell us how many children a synthetic cohort would have (and when) if it followed a *fixed* period fertility schedule with constant shape, quantum and tempo. In contrast, TFR^* (and $\mu^\dagger$) tell us how many children the synthetic cohort would have (and when) if it followed a *shifting* period schedule with constant shape and quantum but changing tempo.

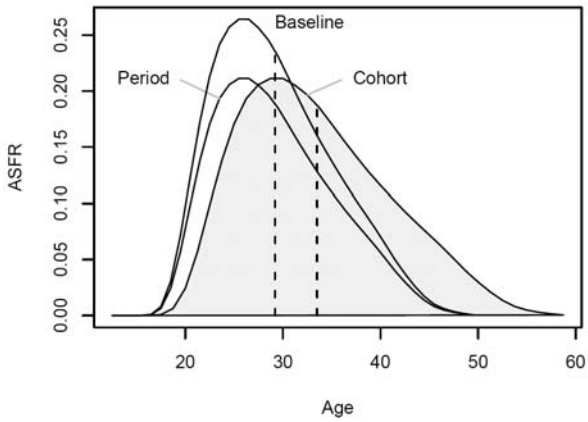


Fig. 1. Period and cohort rates when childbearing is delayed.

Figure 1 illustrates these ideas with a Coale-Trussell (1974) fertility schedule where 90% of women marry, age at marriage has mean 23 and standard deviation 4, the level of natural fertility (M) is 1 and the control parameter (m) is -1. Under this schedule the TFR is 4 children per woman and the mean age of childbearing is 29.2. Suppose, however, that women start delaying fertility at the rate of $r = 0.2$ years per year. As shown in Equation 8, the period age-specific fertility rates would be instantly reduced by 20%, a necessary consequence of the fact that women have slowed down childbearing. The curve labelled "period" shows the deflated schedule, which has a TFR of 3.2 children per woman but the same mean age of childbearing as the original. The curve labelled "cohort" shows the schedule followed by the cohort just starting its reproductive career, assuming the shift continues indefinitely at the same rate. This cohort would have 4.0 children per woman, on average at age 33.5 given by Equation 14.

Figure 2 shows how a shift in a period schedule leads to a stretched cohort schedule. Here we plot the cumulative schedule $F_0(a)$ in the example at 10

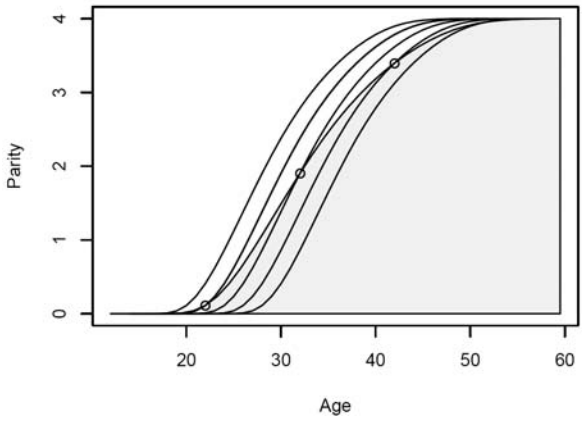


Fig. 2. How a period shift in a parity schedule translates into a cohort delay.

year intervals. We also show in gray the parity schedule for the cohort starting reproductive life when the shift starts, and we mark the points where it "borrows" its cumulative fertility from the three central curves. Note that all schedules lead to a completed family size of four, but the cohort takes longer to climb that far.

To summarize, we have illustrated how a reduction in period fertility from 4.0 to 3.2 can result from delayed childbearing without changes in quantum. Noting that mean age of childbearing increases 0.2 years per year we obtain a TFR^* of 4.0. We can interpret this number as a counterfactual estimate of what the period TFR would have been if women had not delayed childbearing, in which case the mean age of childbearing would still be 29.2. We can also interpret it as the number of children that a synthetic cohort would have if the delay continued indefinitely, in which case mean age of childbearing would be 33.5. The last estimate pairs TFR^* with $\mu^\dagger$, the estimate of mean age of childbearing under changing tempo proposed here.

2.4 Cohort and period shifts

The foregoing results generalize to multiple cohorts if we assume that the cumulative period schedule $F(a,t)$ continues to shift according to Equation 7. For later cohorts this means not only that once childbearing starts it proceeds at a slower pace than before, but also that the start of childbearing itself is delayed. This implication of period-shift models will be of some significance when we turn to mortality, and represents a departure from accelerated failure time models.

Following exactly the same change of variables technique we used for the Zeng-Land cohort, we can show that the cohort born at time t for $t \geq -a_0$ has

$$TFR_c(t) = TFR_0 \quad \text{and} \quad \mu_c(t) = \mu^\dagger + r_c(t + a_0) \tag{15}$$

where r_c is the rate of change of cohort mean age of childbearing, and is related to the period derivative by

$$r_c = \frac{r}{1 - r}. \tag{16}$$

Equation 16 is due to Zeng and Land (2002), who noted that period changes in tempo provide a distorted view of cohort changes in tempo. (They use the notation r^* for r_c.) Note that the cohort considered earlier was born at $t = -a_0$, and that evaluating these expressions at that value leads to $TFR^\dagger$ and $\mu^\dagger$.

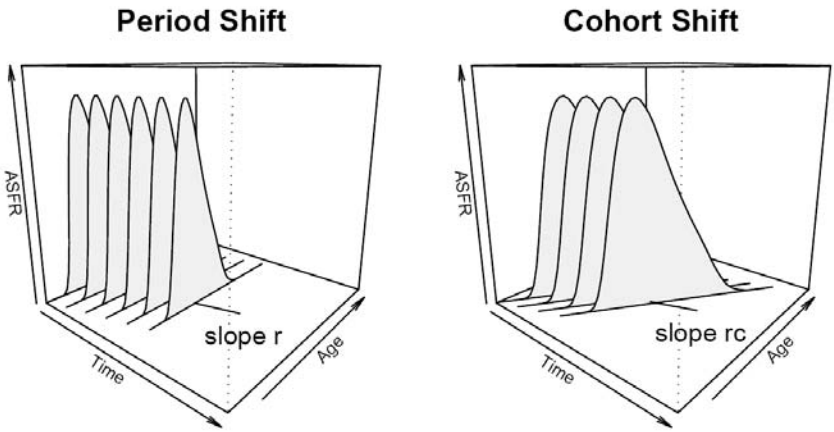


Fig. 3. Shifting period and cohort fertility schedules.

An interesting implication of these results is that a shift in period fertility schedules generates a parallel shift in cohort fertility schedules, with both moving up the age axis but at slightly different rates r and r_c. Figure 3 illustrates this idea using model Coale-Trussell schedules. The left panel shows a period schedule that is shifting to older ages at the rate of $r = 0.2$ years per year, and the right panel shows the corresponding cohort schedules shifting at the rate of $r_c = 0.25$ years per cohort.

Thus, under a simple linear shift model cohort and period quantum are constant and differ by a factor $1 - r$ at time zero and later. Cohort and period tempo change over time. The period mean age of childbearing increases at the rate of r years per year starting from μ_0 at time zero. Cohort mean age of childbearing varies between μ_0 and $\mu^\dagger$ for the active cohorts at time zero, and increases at the rate of r_c years per cohort for cohorts that start their reproductive careers after that. These results provide a way to translate cohort

and period quantum and tempo, but the assumptions required are stronger than for a simple counterfactual interpretation of TFR^*.

3 Mortality

Let us now turn our attention to mortality, focusing on a surface of age-period specific rates $\mu(a,t)$ representing the force of mortality at age a and time t for the cohort born at $t-a$. The rates along a diagonal can be used to compute a *cohort* life table, but the data required are often not available and the calculation can only be completed after the cohort has died.

More often the mortality rates for fixed t are used to compute a *period* life table, which may be interpreted in terms of a synthetic cohort that goes through life subject to the force of mortality prevailing at time t. Bongaarts and Feeney's concern is that period measures, including the period expectation of life and the rates themselves, may be distorted by a tempo effect.

3.1 Mortality translation

Ryder (1964) noted that "the development of translation procedures has proven more difficult for mortality functions than for fertility functions" because of the multiplicative relationships involved in an attrition process, although he made some headway working with the logarithms of the rates. Keilman (1994) later obtained useful translation formulas for the hazards of non-repeatable events, but these do not lead to simple summary results such as Equation 4.

Further progress can be made working with a *survival* surface where $S(a,t)$ represents the probability that someone born at time $t-a$ will survive to age a at time t,

$$S(a,t) = \exp\{-\int_0^a \mu(x, t-a+x)\, dx\}. \tag{17}$$

A nice feature of this surface is that integrating along a diagonal leads to cohort life expectancy:

$$e_0^{(c)}(t) = \int_0^\infty S(a, t+a)\, da. \tag{18}$$

Unfortunately, integrating over a for fixed t does *not* lead to period life expectancy unless mortality is constant. It does, however, lead to a meaningful alternative period measure of longevity, the cross-sectional average length of life (CAL) described by Guillot (2003):

$$CAL(t) = \int_0^\infty S(a,t)\, da. \tag{19}$$

The survival probabilities $S(a,t)$ for fixed t may be interpreted as the age distribution of a population that has a constant stream of births and is subject to the mortality risks $\mu(a,t)$. Bongaarts and Feeney (in this volume p. 11) call this the *standardized* age distribution. CAL is a function of this age distribution and thus depends on past mortality, a point to which we will return later.

In addition to life expectancy and CAL it will be useful to define $\alpha = \int aS(a)\,da \,/\, \int S(a)\,da$, the mean age in the stationary population implied by a survival schedule $S(a)$. A straightforward application of Ryder's (1964) translation formula, which would expand the survival probabilities for the cohort now at its mean stationary age around the current age distribution using a first-order Taylor series, yields

$$e_0^{(c)}(t-\alpha) = \frac{CAL(t)}{1-r_c}, \tag{20}$$

where r_c is the rate of change in the cohort mean stationary age. This shows that, to a first order of approximation, CAL falls below cohort life expectancy when mortality is declining, to an extent determined by the speed of the decline, provided we line up cohorts and periods using mean stationary age.

Guillot (in this volume) applies Ryder's ideas using a somewhat different approach, but reaches essentially the same conclusions. He divides $CAL(t)$ by an index of distributional distortion to obtain an adjusted measure, which can be interpreted as a weighted average of the life expectancies of all cohorts alive at t. He then notes in an application to France that the result is close to the life expectancy of the cohort born at time $t-A(t)$, where $A(t)$ is the mean age of the stationary population at time t, between 30 and 37 years for France in the twentieth century. Here we divide by $1-r_c$ instead of the distortion index, and use cohort rather than period mean age. But we both conclude that when mortality declines CAL falls below the life expectancy of the cohort near its mean stationary age. (I later show under different assumptions that CAL equals the life expectancy of the cohort now at its mean age at death.)

One could take this result to mean that CAL provides a distorted view of cohort life expectancy, or is subject to a tempo effect when mortality is declining, in much the same way that the period TFR distorts cohort fertility. I prefer to view it as indicating that when mortality is declining the age structure lags behind the cohort mortality schedule. In other words, it takes a while for a population to forget its past.

I realize that applying a formula developed for the quantum of fertility to the tempo of mortality seems unusual, if not plain wrong, but Ryder's result is quite general. Given any age-period surface, it relates a cohort integral to a period integral and to the rate of change of the first cohort moment. In fertility we applied it to age-specific rates, so the integrals are measures of quantum and the first moment is tempo. In mortality we applied it to survival probabilities (or age distributions), so the integrals are mean survivals and the first moment is mean stationary age.

3.2 The Bongaarts-Feeney model

The Bongaarts-Feeney model of mortality change is formally identical to the fertility model, except that the period schedule that shifts over time is the standardized age distribution $S(a,t)$ rather than the parity schedule $F(a,t)$. In this section we motivate the model in terms of a slowing down of the passage of time, just as we did for fertility. Later we discuss various period and cohort measures of longevity under the model.

Let $S_0(a)$ denote a survival function and let $d_0(a)$ and $\mu_0(a)$ denote the corresponding density and hazard functions. This could be a conventional period life table or a mathematical model. We will assume that at time zero survival is governed by $S_0(a)$ in the sense that all cohorts are following this schedule. This is equivalent to assuming that the population is stationary with age distribution $S_0(a)$.

Suppose, however, that at time zero all cohorts postpone death at the same rate r. Consider specifically the cohort that has reached age a at time zero, of which a fraction $S_0(a)$ is still alive. We would expect a fraction $S_0(a+1)$ to be alive a year later at age $a+1$, but instead we observe that the proportion surviving has increased to $S_0(a+1-r)$. It is precisely as if the cohort had aged only $1-r$ years in one year. This type of model is known in the statistical literature as an accelerated life model, see for example Kalbfleisch and Prentice (2002). The situation is similar to taking a pill that prevents death (and stops aging) for a fraction r of the year, but I prefer to view the process as developing in continuous time.

Remarkably, this model is equivalent for all active cohorts to a period shift in the standardized age distribution, where

$$S(a,t) = \begin{cases} 1 & \text{if } a < rt \\ S_0(a-rt) & \text{if } a \geq rt \end{cases} \tag{21}$$

For example the survival probabilities for the cohort considered in the previous paragraph are $S(a,0) = S_0(a)$ and $S(a+1,1) = S_0(a+1-r)$. If we compute a cohort derivative, differentiating Equation 21 with respect to both age and time, and changing sign, we obtain a density reflecting the age distribution of deaths at each time

$$d(a,t) = \begin{cases} 0 & \text{if } a < rt \\ d_0(a-rt)(1-r) & \text{if } a \geq rt. \end{cases} \tag{22}$$

Note that $d(a,t)$ is a probability density function only for a cohort, i.e. if we consider $d(a, c+a)$ for fixed c. The period profile is not a real density but a collection of densities for various cohorts, and in this model it integrates to $1-r$, not one. Bongaarts and Feeney (in this volume p. 11) call the integral of $d(a,t)$ for fixed t the total mortality rate (TMR). Watcher (in this volume) notes that it can be interpreted as a period count of deaths.

If we divide the deaths $d(a,t)$ by the numbers exposed $S(a,t)$ we obtain the age-period specific force of mortality

$$\mu(a,t) = \begin{cases} 0 & \text{if } a < rt \\ \mu_0(a - rt)(1 - r) & \text{if } a \geq rt. \end{cases} \tag{23}$$

This is both a period and a cohort hazard, pertaining to time t and to the cohort born at $t - a$. Note that when all cohorts start delaying death at the same rate the hazard is instantly deflated by a factor $1 - r$ and starts shifting to older ages. This is clearly a tempo effect, as it is caused by a delay in death. I don't believe, however, that it is a distortion. The only way that cohorts can delay death is by dying at lower rates, so I view the reduction in hazards as real. The interesting question concerns the implications of this change for longevity.

It will be useful to introduce for completeness two additional functions defined by Bongaarts and Feeney (in this volume p. 11) in (their) Equations 5a and 5b. If we differentiate $S(a,t)$ with respect to time only (as opposed to time and age simultaneously) we obtain the death density

$$d_s(a,t) = d_0(a - rt), \tag{24}$$

and dividing this by the survivors $S(a,t)$ we obtain the hazard

$$\mu_s(a,t) = \mu_0(a - rt). \tag{25}$$

These are proper density and hazard functions for $a \geq rt$ and can best be viewed as inherent features of the standardized age distribution $S(a,t)$, so I will call then the age-distribution density and hazard, respectively. Note that under the period shift model the observed force of mortality $\mu(a,t)$ is proportional to the age distribution hazard $\mu_s(a,t)$, with proportionality factor $1 - r$. This is called the *proportionality* assumption in the Bongaarts-Feeney framework.

I should also note that Bongaarts and Feeney consider a more general shift model where the rate of delay is not a constant r but a function of time $r(t)$. I stick to the linear case because it is simpler and leads to explicit results for cohorts.

3.3 Four measures of longevity

Bongaarts and Feeney (in this volume p. 11) consider four measures of longevity, denoted M_1 to M_4. Three of them are equal under the period-shift model of the previous section. The odd one out is period life expectancy.

The first measure is cohort average length of life (CAL)

$$M_1(t) = CAL(t) = \int_0^\infty S(a,t)\, da. \tag{26}$$

This measure is easily computed by integrating the standardized age distribution. From Equation 21 we find that under the period shift model

$$CAL(t) = CAL(0) + rt, \tag{27}$$

where $CAL(0)$ is both CAL and the conventional expectation of life in the baseline schedule $S_0(a)$. CAL may be computed as an ordinary mean age at death where deaths are obtained by applying the age-distribution hazard $\mu_s(a,t)$ to the standardized age distribution $S(a,t)$. Interestingly, CAL doesn't change when cohorts start postponing death, but it starts increasing at the rate of r years per year as long as the shift (or slow down of time) continues. This occurs because CAL is based solely on the age structure at time t, and does not respond to changes in mortality until these are reflected in the age structure.

The second measure is standardized mean age at death

$$M_2(t) = \int_0^\infty ad(a,t)\,da \,/\, \int_0^\infty d(a,t)\,da, \tag{28}$$

which is based on the standardized age distribution of deaths at time t. The deaths in this index result from applying the current force of mortality $\mu(a,t)$ to the standardized age distribution $S(a,t)$, and may thus be viewed as a measure that depends both on current mortality risks and the current age distribution.

Under the period-shift model the force of mortality $\mu(a,t)$ and the age-distribution hazard $\mu_s(a,t)$ are proportional, with proportionality factor $1-r$. Because this factor appears both in the numerator and denominator of the mean it cancels out, so $M_2(t) = M_1(t)$ as noted by Bongaarts and Feeney (in this volume p. 11). If the proportionality assumption is not satisfied, however, the two indices will differ.

The third measure is conventional period life expectancy

$$M_3(t) = e_0^{(p)}(t) = \int_0^\infty \exp\{-\int_0^a \mu(x,t)\,dx\}\,da. \tag{29}$$

This index may also be viewed as an ordinary mean age at death where deaths result from applying the force of mortality $\mu(a,t)$ to the stationary population implied by that hazard, which is of course the period survival function $\exp\{-\int_0^a \mu(x,t)\,da\}$ (not to be confused with $S(a,t)$). This measure depends on the current force of mortality only.

Under the period shift model the force of mortality $\mu(a,t)$ is proportional to $\mu_s(a,t)$ and therefore the period survival function is a power of the standardized age structure, but there is no simple relationship between $M_3(t)$ and either $M_1(t)$ or $M_2(t)$.

Note that when cohorts start postponing death the conventional expectation of life reacts instantly. Because it depends only on the force of mortality $\mu(a,t)$, which has been deflated by a factor $1-r$, conventional life expectancy e_0 will increase. This is again a tempo effect, but in my view is not a distortion. Conventional life expectancy is just a summary of age-period specific

mortality, and responds appropriately by increasing when the rates decline. In particular, the synthetic cohort interpretation of e_0 as the mean lifetime implied by the current rates continues to be correct.

The fourth measure is the Bongaarts-Feeney tempo-adjusted life expectancy. This index seeks to remove the tempo effect from the force of mortality dividing by $1 - r$ and is therefore defined as

$$M_4(t) = \int_0^\infty \exp\{-\int_0^a \mu(x,t)/(1-r)\, dx\}\, da. \tag{30}$$

Under the period-shift model $\mu(a,t)$ is proportional to $\mu_s(a,t)$ with proportionality factor $(1-r)$ and therefore $M_4(t) = M_1(t) = M_2(t)$, as noted by Bongaarts and Feeney (in this volume p. 11). In this case the adjusted measure can be viewed as an ingenious way to estimate CAL or mean age at death from the observed hazard. If the model does not hold, however, $M_4(t)$ is a different measure that ostensibly depends only on the current force of mortality and the rate of delay r, but in practice requires knowledge of the standardized age distribution for estimation. Watcher (in this volume) provides a characterization of $M_4(t)$ that clarifies this issue.

To summarize, when cohorts start delaying death conventional life expectancy reacts instantly, whereas the other three measures react more slowly, increasing only as the changes work their way into the age structure. The fundamental issue is whether this is a bias or distortion in conventional life expectancy. I argue that it is just a reflection of the fact that when mortality declines the age structure lags behind the force of mortality. To further explore this issue we now look at the cohort implications of the period-shift model.

3.4 Cohort survival

Consider again the cohort born at the time the period shift, or the slowing down of the passage of time, starts. This cohort would have been expected to follow the schedule $S_0(a)$ but instead will follow a stretched schedule, where the probability of surviving to age a is

$$S^\dagger(a) = S_0(a(1-r)). \tag{31}$$

This result follows directly from the period-shift model in Equation 21 and shows that each calendar year the cohort ages only $1 - r$ years.

Figure 4 illustrates how a period shift leads to a cohort delay using a Weibull distribution that is shifting towards higher ages at a rate of 0.2 years per year, an artificially high rate chosen to make the illustration clear. I show the schedule at the start of the process as well as 25, 50, 75 and 100 years later, and superimpose the survival probabilities that would apply to a synthetic cohort undergoing this regime, highlighting the ages where the cohort survival

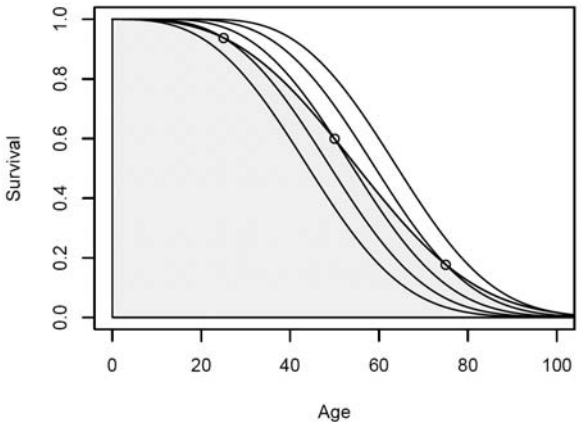


Fig. 4. How a period shift in survival translates into a cohort delay.

"borrows" its probability from the three central curves. The analogy to Figure 2 for fertility should be obvious.

We can compute the expectation of life under $S^\dagger(a)$ using the same change of variables technique that we used in the case of fertility:

$$e_0^\dagger = \int_0^\infty S_0(a(1-r))\,da = \int_0^\infty S_0(y)\frac{dy}{1-r} = \frac{e_0}{1-r}. \tag{32}$$

We find that if $r > 0$ the expectation of life under a shifting schedule exceeds the value it would have if the schedule remained fixed. The area under the original curve is e_0, the shaded area under the stretched curve is $e_0^\dagger$.

Note by way of illustration that life expectancy in the U.S. today is 77.3 under a fixed mortality schedule, but would be 85.8 if the schedule shifted 0.1 years per year, which is the observed gain in period life expectancy between 2001 and 2002. The value 85.8 is computed simply as 77.3/0.9.

Let us return to $S^\dagger(a)$, the survival function that applies to our synthetic cohort. Differentiating we find the density to be

$$d^\dagger(a) = \frac{d}{da}S^\dagger(a) = \frac{d}{da}S_0(a(1-r)) = d_0(a(1-r))(1-r). \tag{33}$$

The hazard, computed as the ratio of deaths to survival, is

$$\mu^\dagger(a) = d^\dagger(a)/S^\dagger(a) = \mu_0(a(1-r))(1-r). \tag{34}$$

Thus, if the mortality schedule shifts 0.1 years per year, a 60 year old would be exposed to 90% of the risk that would have applied at age 54 under a static schedule. These results are consistent with Equations 22 and 23 in the previous section, and thus with equations 8b and 8c in Bongaarts and Feeney (in this volume p. 11). (We showed before that their $\frac{d}{dt}M_2(t) = r$.)

We note again that as soon as time slows down the hazard is deflated by a factor $1 - r$, which is how the cohort manages to live longer. Consider an example where the baseline survival $S_0(a)$ is Weibull with parameters p and λ, so $S_0(a) = \exp\{-(\lambda a)^p\}$. In this case the stretched survival $S^\dagger(a)$ is also Weibull with parameters p and $\lambda^\dagger = \lambda(1-r)$, so the shift and consequent slowing down of the passage of time translate into a proportionate reduction in the hazard at all ages. Kalbfleisch and Prentice (2002) show that the Weibull is the only distribution where the accelerated life and proportional hazards families coincide.

For an example more relevant to human mortality, at least in adult ages, consider a Gompertz model with parameters α and β, where the baseline hazard $\mu_0(a) = \exp\{\alpha + \beta a\}$ increases exponentially with age. In this case the stretched survival is also Gompertz but with parameters $\alpha^\dagger = \alpha + \log(1-r)$ and $\beta^\dagger = \beta(1-r)$, a result that follows directly from the general expression given above. In this case the change in the hazard is not proportional, but relatively larger at older ages. For a country such as the U.S., where adult mortality is roughly Gompertz, a shift of 0.1 years per year starting at age 30 would reduce the hazard by 10% at age 30, 30% at age 60 and 46% at age 90. As a result a 30 year old, who is expected to live another 48.4 years under current conditions, would live on average about 53.8. (These calculations are based on $\alpha = -9.696$ and $\beta = 0.0855$, which implies $\alpha^\dagger = -9.545$ and $\beta^\dagger = 0.07694$. Note that for a shift starting at age a_0 rather than zero $\alpha^\dagger = \alpha + \log(1-r) + \beta r a_0$. The value of $e_0^\dagger = 53.8$ can be obtained as 48.4/0.9 or by numerical integration of the Gompertz hazard.)

These results can be extended to multiple cohorts, just as we did in the case of fertility, by assuming that the standardized age distribution continues to shift at a constant rate. Using essentially the same argument as in the previous section, we can show that the cohort born at time $t > 0$ goes through the survival schedule

$$S(a, t+a) = \begin{cases} 1 & \text{if } a < tr/(1-r) \\ S_0(a - r(t+a)) & \text{otherwise} \end{cases} \tag{35}$$

and thus has life expectancy

$$e_0^{(c)}(t) = e_0^\dagger + r_c t, \tag{36}$$

where $e_0^\dagger$ is the life expectancy of the cohort born at time zero and r_c, the rate of change in cohort life expectancy, is

$$r_c = \frac{r}{1-r}. \tag{37}$$

The cohort born at time zero experiences just a stretching of the survival function $S_0(a)$, which yields a plausible model for all ages. Subsequent cohorts, however, are assumed to experience no mortality until they reach age $r_c t$, at which time they join a stretched and shifted schedule. This feature

makes the model less realistic in multiple-cohort settings unless one restricts its applicability, as Bongaarts and Feeney do, to the adult ages, say above 30, in low mortality populations.

With these caveats, the foregoing results allow us to relate period *CAL* or mean age at death to cohort life expectancy. As we noted in the previous section, when mortality declines the age structure lags behind the force of mortality and as a result

$$CAL(t) < e_0^{(p)}(t) < e_0^{(c)}(t). \tag{38}$$

Under the period-shift model we can be a bit more precise. We can show that the Bongaarts-Feeney measure M_4, which is then the same as *CAL*, M_1 and M_2, is the life expectancy of the cohort now at its mean age at death:

$$CAL(t + e_0^{(c)}(t)) = e_0^{(c)}(t), \tag{39}$$

a result easily verified by direct substitution, noting that the cohort born at t has mean age at death $(e_0 + rt)/(1 - r)$. Alternatively, one can go back in time and note that the cohort dying today was born at time $(t - e_0^{\dagger})/(1 + r_c)$ and has life expectancy $CAL(t)$.

Goldstein (in this volume) has also derived the translation formula (39) and has used it to show that under a continuing linear shift the cohort born today would have life expectancy given by equation (32); this provides increased confidence in these results.

To summarize, conventional life expectancy e_0 measures how long a new born would live under current rates. This may not be a realistic estimate if mortality is declining. Under a period-shift model we have shown that a new born would in fact live longer, $e_0^{\dagger}$ years. On the other hand period *CAL*, mean age at death and the Bongaarts-Feeney adjusted measure M_4 would all be lower, corresponding to the mean age at death of the cohort now reaching its life expectancy, provided the assumptions underlying the simpler linear shift model are satisfied.

3.5 A proportional hazards model

We now consider an example where the assumption is not quite satisfied, and therefore *CAL*, M_2 and M_4 differ. Specifically, consider a population with a constant stream of births and no mortality before age 30. Suppose the force of mortality follows a Gompertz function with $\alpha = -9.997$ and $\beta = 0.0855$, which as noted earlier fits very closely the U.S. 2002 life table. Suppose further that mortality has been constant long enough for the population to become stationary. In this case all four measures, *CAL*, mean age at death, e_0 and the Bongaarts-Feeney tempo-adjusted life expectancy M_4 are 78.45.

Suppose now that at time zero the force of mortality declines 20% at all ages. The conventional period life expectancy, being just a summary of

age-specific mortality, would increase instantly to 80.97 to reflect this improvement. One has to be careful no to conclude that all cohorts will live this long, as the calculation applies only to the cohort age 30 at time zero, assuming mortality remains constant thereafter. *CAL*, on the other hand, doesn't change at time zero but starts increasing immediately afterwards as the decline in mortality is reflected on the standardized age distribution. Eventually the population becomes stationary again and *CAL* reaches 80.97. Figure 5 shows the trajectory of *CAL* for this example.

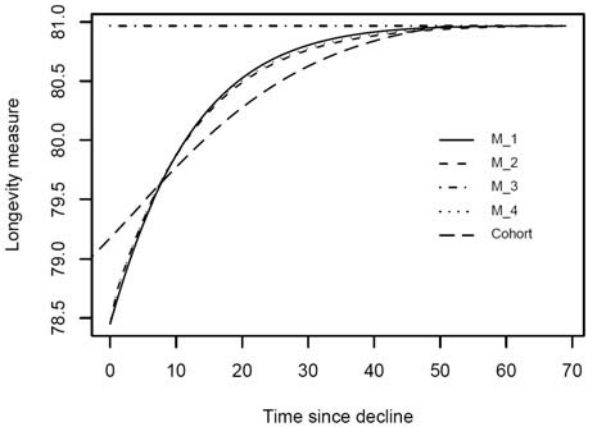


Fig. 5. Measures of longevity after a one-time reduction in hazard.

Mean age at death doesn't change instantly either. Although this index depends on the observed force of mortality, which is 20% lower at time zero, the reduction factor appears both in the numerator and denominator and cancels out. It is only as the reduction works its way into the age structure that mean age at death starts to increase, eventually reaching 80.97. Figure 5 shows that the trajectory of mean age at death is very similar to *CAL*. The Bongaarts-Feeney tempo-adjusted measure depends on the force of mortality and a correction factor based on r, which I estimated using the TMR. (Using a numerical derivative of $M_2(t)$ gives very similar results except for the first two years.) The key result is that M_4 is very similar to the other two measures. It takes them nearly sixty years to fully reflect the instantaneous change in mortality that occurred at time zero.

The figure also shows cohort life expectancy, estimated assuming that mortality was constant both before and after time zero at the specified level. We plot a cohort's life expectancy on the year when it reaches its mean age at death. We note that the three measures of longevity track the increase in cohort life expectancy, albeit only approximately.

4 Discussion

This chapter has emphasized similarities between the analysis of fertility and mortality. I have argued that Ryder's translation formula can be applied quite generally to demographic surfaces. When the surface represents age-specific fertility rates the formula translates period and cohort quantum. When the surface represents survival probabilities the formula translates period and cohort tempo, but using *CAL* rather than conventional life expectancy. The common theme is that period and cohort demographic summaries can differ in times of change. I believe that labelling these differences a bias or distortion has been unfortunate. Period aggregates provide convenient summaries, while cohort aggregates are often needed to fully understand the underlying process.

I have also stressed the fact that the Bongaarts-Feeney framework is essentially the same for fertility and mortality, postulating a period shift in a cumulative schedule representing average parity or survival probabilities. The shift can be motivated by assuming that all cohorts delay childbearing or postpone death at the same rate, and is closely linked to accelerated failure time models used in survival analysis. The shift results in a proportionate reduction in fertility or mortality rates, which also move to older ages. The model applies to multiple cohorts but requires assuming that later cohorts experience not just a slowing down of time but also a delay in the onset of exposure, an assumption that may be less realistic and, in the case of mortality, requires restricting application to adult ages in low mortality populations. I have also proposed measures of tempo under changing fertility or mortality which complement the Zeng-Land interpretation of the Bongaarts-Feeney adjustment by applying to the same synthetic cohort.

Having stressed similarities between fertility and mortality, it is perhaps appropriate to remind ourselves of some fundamental differences. In the case of fertility a reduction in age-period specific rates could represent changes in the quantum or tempo of fertility: women could be having fewer children or just having them later (or both). By assuming that delays occur at all ages at the same rate the Bongaarts-Feeney framework can ingeniously separate the two types of change. In our illustration we could have misinterpreted a reduction in *TFR* from 4.0 to 3.2 as a change in completed family size, but because it was accompanied by an annual increase of 0.2 years in mean age of childbearing–which would lead to just such a reduction–we concluded that it was a pure tempo effect. This does not mean, incidentally, that the reduction in period rates is not real. The only way cohorts can still have 4.0 children but over a longer time is by having them at a slower pace. The new measure of tempo introduced here tells us how much longer it would take.

There are two reasons why mortality is different, even if the same period-shift model applies. First, mortality is a pure tempo phenomenon; everyone dies exactly one time and the only question is when. Consequently, a reduction in the period force of mortality can only mean that cohorts are delaying death.

There is no risk of misinterpretation, and therefore, one might argue, no need for adjustment. Bongaarts and Feeney implicitly acknowledge this point when they note that mean age at death, which they view as a direct analog of mean age of childbearing, needs no adjustment. They do adjust the force of mortality, of course, but I view this adjustment as merely a device to bring the conventional calculation of life expectancy inline with *CAL* or mean age at death. I see no bias or distortion in the observed force of mortality, just as I see no bias in age-specific fertility, and the best proof of that is the fact that cohort survival is determined entirely by $\mu(a,t)$, not by its tempo-adjusted version. The question then is whether we should use standardized mean age at death or conventional life expectancy as a measure of longevity.

That brings us directly to the second reason why mortality is different, and it has to do with exposure. In fertility all women are exposed to have a birth, whether they have had one before or not, which makes $f(a,t)$ a true event-exposure rate. Both the cohort and period *TFR* and mean age of childbearing are summaries of these rates and are not affected by exposure. In the case of mortality only survivors are at risk of dying, which is why analytical interest usually focuses on the force of mortality $\mu(a,t)$, which acts on survivors $S(a,t)$ to produce deaths $d(a,t)$. For a cohort the choice of measure is immaterial because exposure is itself determined by the force of mortality and as a result conventional life expectancy and mean age at death are identical. For a period the two measures can be quite different when mortality is changing. Conventional life expectancy depends only on the period force of mortality $\mu(a,t)$, whereas mean age at death depends also on $S(a,t)$ and thus on the population's past mortality history. We have seen that under the strong assumption of a linear-shift model, mean age at death coincides with the life expectancy of the cohort now reaching its mean age at death.

The question we asked at the outset, 'How long do we live?', can thus be seen to have different answers depending on our precise definition of 'we'. Conventional life expectancy applies to a hypothetical cohort that is exposed to a constant set of rates. It has the great merit of also applying to everyone else when mortality is constant. But when mortality is changing the construction is less useful; why ask how long someone would live subject to these rates if they are changing? We know that they would probably live longer than that, and we can estimate how much longer if we are willing to make strong assumptions about future changes. In particular, a continuing linear shift to older ages leads to $e_0^\dagger$, the simple measure of life expectancy under changing mortality proposed here. It is also the case that when mortality is declining no cohort has yet lived that long, or even as long as e_0 would imply. The Bongaarts-Feeney measure tells us how long those dying today have lived, standardizing for cohort size, when the proportionality assumption holds. The fact that those dying today haven't lived as long as today's newborns will probably live, under either fixed or changing rates, is not a bias or distortion; it's just a fact of life.

The foregoing discussion has emphasized the practical interpretation of various measures of longevity while implicitly accepting the conventional view that mortality change is driven by the hazard function. But the Bongaarts-Feeney approach is fundamentally different; it views mortality change as driven by gains in longevity that shift the age distribution. This deflates the hazard by a factor $1 - r$ and shifts it to older ages. Unfortunately, it is difficult to differentiate these frameworks empirically because the age patterns in low-mortality countries are very close to a Gompertz model, where a proportionate reduction in the hazard cannot be distinguished from a shift to older ages. But if mortality were to stop declining we would soon know, because the period-shift model predicts an increase in the hazard as the factor $1 - r$ disappears and our past catches up with us, whereas the conventional view is that the hazard would stay constant. Faced with such choice, one may very well prefer to see hazards continue to decline and live longer with the uncertainty.

Acknowledgments

I am grateful to John Bongaarts and James Vaupel for inviting me to participate in the Workshop on Tempo Effects in Mortality held in New York, November 18-19, 2004, sponsored by the Max Planck Institute for Demographic Research and the Population Council. I learned a lot from all the participants. I am also grateful to John Bongaarts for detailed comments on an earlier draft and for a stimulating email exchange.

References

Arias, E. (2004). United States life tables, 2002. *National Vital Statistics Reports*, 53(6):271–291.

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. The quantum and tempo of life-cycle events. *In this volume, also published in in Vienna Yearbook of Population Research*, pages 115–151. 2006.

Bongaarts, J. and Feeney, G. (1998). On the quantum and tempo of fertility. *Population and Development Review*, 24(2):271–291.

Bongaarts, J. and Feeney, G. (2000). On the quantum and tempo of fertility: Reply. *Population and Development Review*, 26(3):560–564.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Coale, A. J. (1971). Age patterns of marriage. *Population Studies*, 25(2):193–214.

Coale, A. J. and Trussell, T. J. (1974). Model fertility schedules: Variations in the age structure of childbearing in human populations. *Population Index*, 40:185–258.

Goldstein, J. R. Found in translation? A cohort perspective on tempo-adjusted life expectancy. *In this volume, also published in Demographic Research*, 14(5):71–84. 2006.

Guillot, M. Tempo effects in mortality: An appraisal. *In this volume, also published in Demographic Research*, 14(1):1–26. 2006.

Guillot, M. (2003). The cross-sectional average length of life (CAL): A cross-sectional mortality measure that reflects the experience of cohorts. *Population Studies*, 57(1):41–54.

Kalbfleisch, J. D. and Prentice, R. L. (2002). *The Statistical Analysis of Failure Time Data*. John Wiley and Sons, New York, 2nd edition.

Keilman, N. (1994). Translation formulae for non-repeatable events. *Population Studies*, 48(2):341–357.

Ní Bhrolcháin, M. (1992). Period paramount? a critique of the cohort approach to fertility. *Population and Development Review*, 18:599–629.

Ryder, N. B. (1964). The process of demographic translation. *Demography*, 1(1):74–82.

Schoen, R. (2004). Timing effects and the interpretation of period fertility. *Demography*, 41(4):801–819.

van Imhoff, E. and Keilman, N. (2000). On the quantum and tempo of fertility: Comment. *Population and Development Review*, 26:549–553.

Vaupel, J. W. (2002). Life expectancy at current rates vs. current conditions: A reflexion stimulated by Bongaarts and Feeney's "How long do we live?". *Demographic Research*, 7. Article 8.

Wachter, K. W. Tempo and its Tribulations. *In this volume, also published in Demographic Research*, 13(9):201–222. 2005.

Zeng Yi and Land, K. C. (2001). A sensitivity analysis of the Bongaarts-Feeney method for adjusting bias in observed period total fertility rates. *Demography*, 38:17–28.

Zeng Yi and Land, K. C. (2002). Adjusting period tempo changes with an extension of Ryder's basic translation equation. *Demography*, 39(2):269–285.

Lifesaving, lifetimes and lifetables*

James W. Vaupel

Max Planck Institute for Demographic Research, Konrad-Zuse-Strasse 1, 18057 Rostock, Germany. E-mail: jwv@demogr.mpg.de

Summary. Mortality change roils period rates. In the short term, conventional calculations of age-specific probabilities of death and life expectancy in the period immediately after the change depend on how many lives have been saved. In the long term, the probabilities and period life expectancy also depend on how long these lives have been saved. When mortality is changing, calculations of period life expectancy do not, except in special circumstances, measure the life expectancy of a cohort of newborns that hypothetically live all their lives under the new mortality regime.

1 Introduction

When a life is saved, how long is death averted? When death rates are declining, how should period lifetables be estimated? This demographic essay explains why and how the answer to the second question hinges on the answer to the first. My thinking was stimulated by the pathbreaking research by Bongaarts and Feeney (2002; in this volume p. 11 and p. 29) on why conventional calculations may lead to distorted estimates of period life expectancy.

The conventional formula for period life expectancy at current death rates can be expressed as

$$e_0 = \int_0^w e^{-\int_0^a \mu(x)\,dx}\,da\,, \tag{1}$$

where $\mu(x)$ is the force of mortality (hazard of death) at age x as estimated from observed counts of age-specific deaths and the age-specific population at risk. In contrast, life expectancy under current conditions is given by

$$e_0^* = \int_0^w e^{-\int_0^a \mu^*(x)\,dx}\,da\,, \tag{2}$$

where is the force of mortality that would be experienced by a cohort of newborns that lived all their lives under the mortality regime that prevailed at the time of their birth (Vaupel 2002). Note that this concept of life expectancy under current conditions is a period measure based on the hypothetical notion that the mortality regime at the time of the cohort's birth continues unchanged until the last member of the cohort dies.

It is helpful to consider a special, simple case. Suppose that there is a mortality regime that prevails up until some point in time after which a new mortality regime prevails. Although there may have been human populations in the past that lived under a more or less fixed mortality regime, a sudden but long-term shift from one regime to another may rarely if ever been experienced. In contemporary human populations mortality conditions are changing incessantly. In laboratory experiments, however, with non-human populations, it is possible to switch from one mortality regime to another. For instance, many experiments have been performed with animals such as fruit flies or mice that are given one kind of diet up until some point and a different kind of diet afterwards (e.g., Mair et al. 2003, Carey et al. 1998). The Max Planck Institute for Demographic Research houses a lightbulb laboratory in which large numbers of small bulbs can be lit at either 5 or 6 volts. A population of lightbulbs can be kept under harsh 6 volt conditions until some moment and then at a more salubrious 5 volts thereafter.

To simply the exposition, it is also helpful to make three other assumptions. The number of births in the population during each time interval is assumed to be constant. The population is assumed to be closed to migration. And the new mortality regime is assumed to be more favorable than the old regime. In particular, every individual under the new conditions would live at least as long as under the older conditions and some individuals would live longer. Again, such assumptions do not pertain to actual human populations, but they can be achieved in laboratory experiments. So it is possible to imagine a concrete instance of the kind of mortality shift I will consider in this chapter-think, e.g., about laboratory flies fed a poor diet and then a better diet or about lightbulbs lit at 6 volts and then at 5 volts. In any case, the assumptions are not of fundamental importance. The theory can be generalized. The assumptions, however, drastically simplify the exposition of the theory.

Consider age-specific death rates in the interval right after the shift from the unfavorable mortality regime to the favorable regime. To be specific, consider lightbulbs during the day after voltage has been lowered from 6 to 5 volts. Suppose that the population of lightbulbs consists of different cohorts that were turned on on different days. Let $\mu(x)$ in formula (1) above be the force of mortality for bulbs that were turned on x days ago, as estimated from observations of how many bulbs died over the course of the day. Then e_0 , life expectancy at current rates, can be calculated using (1). Suppose a cohort of lightbulbs is illuminated at 5 volts until the last bulb fails. If laboratory conditions are held constant, then observations of this cohort can be used to estimate $\mu^*(x)$ in formula (2) and hence e_0^* , life expectancy under current

conditions. The main thrust of this chapter is to demonstrate that, except in special circumstances, $\mu(x)$ will not equal $\mu^*(x)$ and hence e_0 will differ from e_0^* . Annette Baudisch, Jutta Gampe, Mieke Reuser, Dirk Vieregg and I are currently conducting lightbulb experiments to provide empirical evidence for this assertion; in this chapter I will present the theoretical case.

Vaupel, Manton and Stallard (1979) considered populations of individuals that are heterogeneous with respect to their age-specific chances of death. They showed that if mortality conditions are changing, then life expectancy at current rates does not, except in special circumstances, equal life expectancy under current conditions, i.e., that $e_0 \neq e_0^*$. They developed a model of gamma-distributed frailty (i.e., relative risk of death) and used it to derive formulas for μ^* and e_0^* . Further research (e.g., Vaupel and Yashin 1985, 1987a, 1987b; Vaupel, Yashin and Manton 1988; and Vaupel 2002) extends this line of thinking to other kinds of models of heterogeneous populations. The fundamental concept of these models is that a cohort's mortality at some age depends not only on current conditions but also on the historical conditions the cohort has suffered.

Bongaarts and Feeney (2002) formulated a delayed-death model such that health improvements in some year add an increment δ to the remaining lifespans of everyone over 30. Note that this increment does not depend on the mortality history of a population but only on a change in current conditions. In the delayed-death model it is also the case that, except in special circumstances, $e_0 \neq e_0^*$. Bongaarts and Feeney (2002, in this volume p. 11 and p. 29) suggest various formulas for estimating e_0^* . Some scholars think that these formulas are problematic (e.g., Wachter in this volume). I will not consider this issue here, but I will show that Bongaarts and Feeney's basic point, that $e_0 \neq e_0^*$, is correct.

The general conclusion of the present chapter is that $e_0 \neq e_0^*$ (and $\mu(x) \neq \mu^*(x)$) under much broader circumstances than those considered by Vaupel, Manton and Stallard (1979) or by Bongaarts and Feeney (2002, in this volume p. 11 and p. 29). A discrepancy can arise whenever mortality conditions are changing. The two life expectancies will differ when lives saved at age x are extended by an average increment that is not equal to remaining life expectancy at age x at current death rates. Furthermore, the two life expectancies will differ when lives lost at age x are shortened by an average decrement that is not equal to conventionally-calculated remaining life expectancy at age x. Bongaarts and Feeney's delayed-death model and the various models of heterogeneity in innate and acquired frailty are special cases.

2 How saving a life alters life expectancy

Consider a stylized population with constant age-specific death rates. Suppose that the population is closed to migration and that the number of births each year is constant. Suppose that at some age x in some year y a life is saved.

How will the conventional lifetable for that year differ from the lifetable for previous years? The answer is easily derived if the following formula is used:

$$e_x = \frac{D_x}{N_x} \cdot \frac{1}{2} + \frac{N_x - D_x}{N_x} \cdot (1 + e_{x+1}) \,, \tag{3}$$

where e_x is remaining life expectancy at age x, N_x is the number of individuals who celebrate their xth birthday in year y and D_x is the number of deaths among those individuals. This formula is consistent with conventional lifetable methods and from the values of e_x age-specific death rates and other lifetable statistics can be calculated. In this formulation e_x is calculated using data for Lexis rhombuses that extend from the start of year y to the end of year $y + 1$, but other formulations based on Lexis squares or on Lexis rhombuses that stretch over two years of age and one year of time could also be used.

If a death is averted at age x, then

$$e_x^* = \frac{D_x - 1}{N_x} \cdot \frac{1}{2} + \frac{N_x - (D_x - 1)}{N_x} \cdot (1 + e_{x+1}) \,. \tag{4}$$

Subtracting 3 from 4 yields

$$e_x^* - e_x = \frac{1}{N_x} \cdot (0.5 + e_{x+1}) \,. \tag{5a}$$

If n_x deaths are averted, then

$$e_x^* - e_x = \frac{n_x}{N_x} \cdot (0.5 + e_{x+1}) \,. \tag{5b}$$

The expression $0.5 + e_{x+1}$ can be interpreted as the remaining life expectancy of someone whose life was saved at age x (under the assumption that such a life is saved, on average, halfway through the year.)

The implication of (5b) is clear and important: conventional lifetable calculations are consistent with the assumption that when lives are saved at some age x the beneficiaries gain, on average, the remaining life expectancy at that age. More precisely, the assumption is that each beneficiary will face the same age-specific hazards of death for the remainder of his or her life as those faced by individuals with lifespans greater than x.

The actual average lifespan gained by the resuscitated may, however, be more or less than remaining life expectancy. To be concrete let $0.5 + e_{x+1}$ equal ten but suppose that each resuscitated individual dies one year after being saved. Further suppose that $\frac{n_x}{N_x}$ is one percent. Then conventional calculations yield an increase in remaining life expectancy at age x of a tenth of a year whereas the actual gain is only a hundredth of a year. In other words, conventional calculations lead to a distorted estimate of real life expectancy.

Before analyzing this distortion further, it is useful to consider mortality improvements at several ages. Suppose that n_x deaths are averted at age x and that n_{x+1} deaths are averted at age $x + 1$. Then

$$e^*_{x+1} = \frac{D_{x+1} - n_{x+1}}{N_{x+1}} \cdot \frac{1}{2} + \frac{N_{x+1} - (D_{x+1} - n_{x+1})}{N_{x+1}} \cdot (1 + e_{x+2}) \qquad (6)$$

and

$$e^*_x = \frac{D_x - n_x}{N_x} \cdot \frac{1}{2} + \frac{N_x - (D_x - n_x)}{N_x} \cdot (1 + e^*_{x+1}) \, . \qquad (7)$$

Note that e^*_x depends on e^*_{x+1} rather than on e_{x+1} . More generally, if mortality improvements are made at older ages, then persons whose lives are saved at some younger age are assumed to gain, on average, the remaining life expectancy at this age taking into account the progress at older ages.

3 Individual lifetimes

To further explore why and when $e_0 \neq e^*_0$, the notion of individual lifetimes is useful. Let X_i be the lifetime (i.e., age at death) of some specific individual i. Let $X_i(x, y)$ be the total lifetime of this individual if he or she survives to age x in year y, under the assumption that the mortality conditions prevailing in year y persist for the rest of the individual's life. Hence $X_i(0, y_0)$ is the age at which the individual would die if mortality conditions remain the same as in the individual's year of birth y_0 .

Suppose mortality is being reduced. For simplicity assume that mortality levels remain the same from year y_0 to just before year y and then fall suddenly. Suppose that individual i survives to age x in year y. Let x^- and y^- denote the individual's exact age and the exact time just prior to the mortality improvement. Because before year y there is no change in death rates, $X_i(x^-, y^-) = X_i(0, y_0)$. Mortality improvement means that for at least some individuals $X_i(x, y) > X_i(x^-, y^-)$.

Kenneth Wachter suggested a helpful way of thinking about this model. Imagine that each individual is given a ticket at birth that entitles the person to a specific lifespan. This lifespan at the time of birth y_0 is denoted above by $X_i(0, y_0)$. Individuals keep their lifespan tickets until time y. Some may die before this time. Among the survivors, some and perhaps all individuals get a new ticket with a new lifespan, namely the lifespan denoted above by $X_i(x, y)$. Because the new mortality regime is assumed to persist indefinitely, individuals keep their new lifespan until death. Babies born at time y get tickets that are consistent with the new mortality regime. All babies born at any time after y similarly get tickets that are consistent with the new mortality regime. Consider three possibilities.

First, suppose everyone's life is extended by some increment δ:

$$X_i(x, y) = X_i(x^-, y^-) + \delta \, . \qquad (8)$$

This is the elegantly simple model suggested by Bongaarts and Feeney (2002, in this volume p. 11 and p. 29). The increment δ would be gained not only by everyone alive at time y, but also by all future generations. One way to

capture this notion is to allow x to be negative. If x is negative, then the person will be born in x years.

Second, suppose that all individuals henceforth age at a slower pace such that time in the future is stretched out by the factor $1 + \rho$:

$$X_i(x, y) = x + (1 + \rho)(X_i(x^-, y^-) - x) \,. \tag{9a}$$

This model can also be formulated as:

$$X_i(x, y) = X_i(x^-, y^-) + \delta_i \,, \tag{9b}$$

where

$$\delta_i = \rho(X_i(x^-, y^-) - x) \,. \tag{9c}$$

Note that the increment δ_i depends on how much longer the individual would have lived under the conditions prevailing before the mortality improvement. As in the case of constant δ , the mortality improvement would benefit future generations as well as those alive at time y.

Third, suppose that some individuals gain from the mortality improvement and some do not. In particular, suppose that there is a chance π that a specific individual's life will be extended and a corresponding chance $1 - \pi$ that the individual's life will not be extended. That is, suppose

$$\Pr\left\{X_i(x, y) = X_i(x^-, y^-) + \delta_i\right\} = \pi \tag{10a}$$

and

$$\Pr\left\{X_i(x, y) = X_i(x^-, y^-)\right\} = 1 - \pi \,; \tag{10b}$$

where Pr denotes "probability of" and the increment δ_i is greater than zero. Further suppose that δ_i is a random variable that has the same distribution as the distribution of remaining lifespans at age X_i . That is, suppose that when the life is saved of an individual who would have died at some age X_i, then this individual thereafter faces the same life chances as individuals whose lifespans are greater than X_i. If $\overline{\delta_i}$ is the expected value of δ_i, then $\overline{\delta_i}$ equals remaining life expectancy at age X_i. Hence, individuals whose lives are saved at age X gain, on average, the remaining life expectancy at age X. This model is consistent with the conventional approach to estimating life expectancy, as shown above.

Note that in all three cases the individual does not have to be on the verge of death. That is, X_i may be larger than x. A person who would have died at some age in some future year might have the scythe of death averted in an earlier year. Indeed, as noted above, the person might not even be born yet. (If X_i is smaller than x, then the individual has died and the improvement is too late.)

4 The triangle of turbulence

The three special cases described above are illustrative of the range of possibilities. It is also useful to consider a stylized model of the general situation. Consider a hypothetical population that is closed to migration. The number of births each year is constant. Until year 0 an unchanging mortality regime has prevailed for longer than any individual has lived. Then, on January 1st of that year, a new, more favorable mortality regime starts and persists for longer than the maximum lifespan of individuals in the population. Let $D_y(x)$ denote the number of deaths over the course of year y among those who were x years old on January 1st of that year. Note that $D_y(x)$ is the number of individuals such that $x \leq X_i(x, y) < x+1$. Let D_y^0 denote the number of deaths of babies who are born in year y and who die the same year. Assume that for any two years $y_1 < 0, \quad y_2 < 0$, when the old mortality regime prevailed, $D_{y_1}(x) = D_{y_2}(x)$ and $D_{y_1}^0 = D_{y_2}^0$. (In a study of empirical data, stochastic variation would have to be considered, but the purpose of the stylized model here is cogent exposition rather than statistical analysis.) For cohorts born in any two years, $y_1 \geq 0, \quad y_2 \geq 0$, when the new mortality regime prevails, $D_{y_1+x}(x) = D_{y_2+x}(x)$ and $D_{y_1}^0 = D_{y_2}^0$. The values of interest lie in a triangle on the x,y plane, the triangle bounded by the x axis at $y = 0$ and by the diagonal cohort line along which $x = y$. There can be a turbulent pattern of death counts in this triangle even though the entire triangle lies in the domain of the new mortality regime.

To understand this, consider the number of lives saved in the first year of the new mortality regime among those who are x years old when the new regime starts on January 1st of year 0. The number is simply $D_{-1}(x) - D_0(x)$ This difference gives the number of individuals who would have died under the old regime who do not die under the new regime. In terms of the ticket concept, it is the number of individuals who get a new ticket that entitles them to a longer life such that they do not die in the year that they would have died if they had not gotten a new ticket. Because the number of individuals who are age x on January 1st of year 0 equals the number on January 1st of year -1, the values of $D_{-1}(x) - D_0(x)$ determine the gain in conventionally-calculated life expectancy, $e_0(0) - e_0(-1)$. The number of lives saved, however, does not reveal the increment of additional lifespan that is gained by the resuscitated. As discussed above, conventional calculations assume that the increment is determined by the remaining lifespans of those who lives were not saved but this may not be the case.

In all years after time 0 a constant mortality regime prevails and there is a constant number of births each year and no migration. So why should the number of deaths at some age vary from year to year? In particular, why should the number of deaths at each age in each year not be the same as the number at the age in year 0? The reason is that individuals whose lives were saved in year 0 are dying in various years and at various ages and these postponed deaths are adding to the death counts. Under the new mortality

regime some individuals who would have died after year 0 also are gaining lifespan extensions. As those resuscitated in year 0 and subsequent years die, they add to death counts.

Except in special cases, notably the conventional life expectancy model, the shifting pattern of age-specific death counts will result in changing values of age-specific death rates in the triangle and changing values of conventionally-calculated period life expectancy. The mortality regime changes on January 1st of year 0, but there is a wake of mortality turbulence that lingers on, gradually diminishing, until it completely peters out after the death of the last individual born under the old mortality regime whose lifespan was extended under the new regime.

5 How large is the distortion?

How distorted is the conventional calculation of life expectancy when death rates are declining? The results above show that the answer depends on how long death is averted. As noted earlier, Bongaarts and Feeney (2002) suggest an ingenious estimate. Let $X_i(x-1, y-1)$ be the total lifetime, under the mortality regime prevailing in year $y-1$, of an individual who attains age $x-1$ at the start of year $y-1$ and who survives to reach age x at the start of year y. Suppose there are improvements in health conditions at the start of year y that benefit everyone equally. That is, suppose the new age at death is

$$X_i(x, y) = X_i(x-1, y-1) + \delta \quad \forall i \,. \tag{11}$$

Then if deaths are uniformly distributed over the course of a year, the number of lives saved is $n_x = \delta \cdot D_x$. If, for instance, δ is 0.25 (i.e., three months), then one-quarter of deaths would be averted (namely, all the deaths from October 1 through December 31). In this view, health improvements delay death equally for everyone.

This is an extreme assumption-and an elegant one. It can be relaxed by letting δ vary with age and letting δ vary, in the interval $0 \leq \delta < 1$, across individuals.

These are details. The key idea is that annual health progress lets many people gain a short additional span of life. Conventional lifetable calculations, in contrast, are consistent with the notion that lifesaving helps a few people gain (on average) a long increment of life, namely, remaining life expectancy at the age when they would have died.

If deaths are being delayed by a fraction of a year δ each year, then $e_0^*(y)$, the true life expectancy at birth in year y, is given by

$$e_0^*(y) = \int_0^w \exp\left(-\int_0^a \frac{\mu(x, y)}{1-\delta}\, dx\right) da \,, \tag{12}$$

Note that the starting age 0 might be age 30 or some other age after which the delayed-death model is assumed to hold. As described above, death counts are

assumed to be reduced by the factor δ, so the "observed" force of mortality has to be adjusted by dividing by $1 - \delta$. Bongaarts and Feeney suggested several approaches to estimating δ, reviewed by Bongaarts (in this volume). The validity of the estimates is controversial, as discussed by Wachter (in this volume). Further research is needed to resolve the issues. It seems likely, however, that if conventionally-calculated life expectancy is increasing, say, at a quarter of a year per year because of a uniform delay in deaths, then δ is probably close to 0.25. If so, $e_0^*(y)$ can be two or three years less than $e_0(y)$, as argued by Bongaarts and Feeney (2002, in this volume p. 11).

6 Considerations about true life expectancy

The true life expectancy of a synthetic cohort living under current mortality conditions might not be as low as the delayed-death model suggests. It might, however, be lower than conventional lifetable calculations imply. The issues here are complicated and require extensive research. Let me adumbrate some key considerations.

If life expectancy in some year is *defined* as the value implied by current death rates using the conventional formula, then life expectancy cannot be considered distorted. It is simply the value implied by current rates. Demographers, however, usually describe life expectancy as the average lifespan of a synthetic cohort of individuals who live all their lives under current mortality conditions. Life expectancy under current mortality conditions may not be equal to life expectancy calculated using current mortality rates. As explained above, a discrepancy can arise when mortality changes. When death rates are decreasing, then life expectancy under current mortality conditions is less than life expectancy under current mortality rates if those whose deaths are averted have a remaining lifespan that is less than the remaining life expectancy implied by conventional calculations. The bigger the difference between the actual increments in lifespans and lifetable values of remaining life expectancies, the bigger the discrepancy. A similar kind of discrepancy, of opposite sign, can arise when death rates are increasing.

At some ages (e.g., infancy and childhood) most of the lives saved, at least historically and perhaps also today, may have been extended for many years. Bongaarts and Feeney acknowledge this and apply their delayed-death approach only after age 30, after 1950, and in developed countries.

At least some mortality improvements have occurred because specific causes of death have been reduced. To the extent that some people were at higher risk than others, then a fraction of the population gained substantial increments of life. LeBras (in this volume) discusses this.

Bongaarts and Feeney (2002, in this volume p. 11) show that in developed countries the age-specific force of mortality after age 30 can be approximated by parallel Gompertz curves that shift outward to higher ages over time. This finding is consistent with the delayed-death model and the notion that health

progress is helping everyone above 30 more or less uniformly. If lifesaving were due to multiple actions that reduced various causes of death at various ages, then a more complicated pattern of change in age-specific mortality might be expected.

The notion that general improvements in health conditions are delaying death uniformly for everyone does, however, have an implication that may be questionable in some circumstances. The delayed-death model assumes that everyone benefits from the delay–at all ages (at least above 30) and even if a person would not have died for many years. Hence, a 60 year-old who would otherwise have died this year and a newborn who would have died at age 60 some 60 years from now are both assumed to gain the same extension of life. The newborn, however, will enjoy the improved health conditions for six decades before benefiting whereas the 60 year-old hardly experiences the improved conditions. Whether this is important depends on the nature of the mortality improvement. If, for instance, the improvement is a drug that adds three months between the onset of a disease and death, then the delayed-death model would be appropriate.

Finally, let me highlight an implausible implication of the conventional lifetable approach. The implication was discussed in an article on "Repeated Resuscitation: How Lifesaving Alters Life Tables" (Vaupel and Yashin 1987a). Suppose death rates are lower at every age in some year compared with some previous year. A person who lived his or her entire life under the better mortality conditions might benefit from not dying at the age he or she would have died under the inferior conditions. This person might also benefit at a later age from the lifesaving implied by the lower death rates. Hence, the person might be repeatedly resuscitated. The article provides formulas and several examples. Here let me quote a single example. Consider the simple case such that death rates in the more favorable mortality regime are half as high, at all ages, as in the less favorable regime. Then, as explained by Vaupel and Yashin (1987a), "at the moment death would have occurred, half of the individuals are reprieved-and the other half die as before. [H]alf of the cohort do not benefit from lifesaving...." That is, life expectancy at birth for those whose lives are not saved is the life expectancy under the unfavorable mortality regime. It seems implausible to me, at least under the conditions that have recently prevailed in developed countries, that mortality improvements that cut death rates in half would be of no direct benefit to half the population. The halving of death rates would be achieved as a result of substantial improvements in health and such improvements would probably, it seems to me, help nearly everyone live at least a bit longer.

These various considerations suggest that both the conventional lifetable approach and the delayed-death model may be extreme cases. On the one hand, it seems unlikely that mortality improvements result in death being averted for a few people who gain, on average, remaining life expectancy. Those whose lives are saved probably tend to be relatively frail or vulnerable and their remaining lifetimes are probably, on average, shorter than the re-

maining life expectancy of those not rescued from death. On the other hand, it also seems unlikely that mortality improvements give everyone the same lifespan increment, regardless of age and regardless of how long an individual will live under the new health regime. It is therefore useful to consider other models. One approach is provided by models of heterogeneity in innate or acquired frailty, as I discussed in an earlier reflexion on Bongaarts and Feeney's contributions (Vaupel, 2002). Another approach is sketched below.

7 A model of stretched lifetimes

For cold-blooded animals life runs more slowly when temperatures are lower. In particular, the trajectory of age-specific death rates is stretched out over a longer period of time at lower vs. higher temperatures (Mair, Goymer, Pletcher, Partridge 2003). Reliability engineers use "accelerated-failure-time models" to describe this kind of phenomenon. It is a misleading term from the perspective of this chapter because the focus here is on reductions rather than increases in mortality. Hence, I will refer to "stretched-lifetime models". The basic idea is that the deaths that would have occurred over some period of time t occur over a longer period of time $(1+\rho)t$. This model is summarized above in formulas (9a), (9b) and (9c).

Consider the simple case when ρ is one. If life expectancy was e_0 before the mortality improvement, then it is $2 \cdot e_0$ after the improvement. That is, a cohort living under the new conditions will live twice as long on average. From a period perspective, the number of deaths in some time interval following the improvement will be half as many as the number of deaths in a similar time interval before the improvement. Hence, conventional lifetable calculations will be based on death rates that have been cut by a factor of two. If death rates are the same at all ages, then cutting death rates by a factor of two is equivalent to stretching time by a factor of two. But if death rates change over age, this is not the case. In some countries women suffer about half the age-specific chance of death as men, but in these countries women do not live twice as long as men but only about six years longer. In the case of stretched lifespans when mortality increases with age, conventional lifetable calculations *underestimate* true life expectancy.

8 Quantum and tempo vs. proportions and increments

So far, the word tempo has not appeared in this chapter. This may seem strange in a contribution, inspired by the work by Bongaarts and Feeney on tempo distortions, that is appearing in a collection of articles on tempo effects on mortality. The notion that there are quantum and tempo effects on fertility was developed by Norman Ryder: quantum refers to the number of births and tempo to the pace of childbearing. For demographers deeply versed in this

tradition it may be useful to consider lifesaving from a tempo perspective. The concept of tempo effects on fertility stimulated Bongaarts and Feeney to question conventional calculations of life expectancy. It seems to me, however, that the tempo-quantum metaphor may sometimes be more misleading than helpful, at least in studies of mortality, and that the metaphor has to be treated with great caution.

To my mind, a better starting point is the fact that all populations are heterogeneous and that changes in conditions will affect individuals differently. If mortality is reduced, then some individuals may gain no additional life, some may gain a little, and some may gain a lot. So a natural vantage point for me is to think (e.g., as in Vaupel 2002) about what proportion of people gain how much. In the case of reductions in period fertility, it may similarly be useful to consider how many women (and men) are postponing childbearing by how long (and how many are choosing not to have an additional child at all.)

The three cases considered in this chapter-the conventional lifetable approach, the delayed-death model, and the stretched-lifetime model-neglect heterogeneity. In the conventional approach, all individuals resuscitated at some age, together with all individuals who would otherwise have survived this age, face the same schedule of mortality at subsequent ages. The delayed-death model assumes that all individuals gain the same increment of life. The stretched-lifetime model slows time equally for everyone. Because all populations are heterogeneous, all three perspectives are wrong. Nonetheless, all three perspectives may prove useful. Each of the models may provide serviceable approximations or bounds for at least some kinds of lifesaving interventions in some contexts. Alternative models that build on the theory of heterogeneous populations may also prove useful.

9 Directions for research

The conventional lifetable approach, the delayed-death model, the stretched-lifetime model, and various models of innate and acquired heterogeneity may all offer useful perspectives for understanding the fundamental nature of mortality change. Some kinds of change may extend a few people's lives for an average period that may approach remaining life expectancy. Other kinds of change may extend many people's lives for a short time. Still other kinds of change may slow the clock of aging. All populations are heterogeneous, so each of these kinds of changes may affect individuals somewhat differently. Hence it is a question of both/and rather than either/or. That is, research is not needed to determine which model is right and which models are wrong. All the models are wrong. The research required is research to determine which model or mix of models is most helpful in understanding mortality change at various ages, various times, and as a result of various kinds of interventions.

There are two directions for this research: theoretical thinking about the nature of lifesaving and empirical analyses that test alternative theoretical

hypotheses. This chapter and previously published studies about tempo effects on mortality and on the theory of heterogeneous populations have contributed to theoretical understanding. Great progress has been made in understanding how lifesaving may, theoretically, affect lifetimes and lifetables. The burst of innovation since Bongaarts' and Feeney's (2002) stimulating insight is greater than that in any comparable period since 1661, when Graunt's seminal study was presented. Theoretical research on the impact of lifesaving is burgeoning with vitality and fresh new growth. Further thinking will almost certainly produce a spate of further ideas.

As noted in the introduction, the basic thrust of this chapter is that life expectancy at current rates will generally differ from life expectancy under current conditions when the following two conditions both hold. First, current mortality conditions differ from past mortality conditions. Second, the lives saved or lost at each age x (because of the change in mortality conditions) are extended or shortened by an average amount that is not equal to remaining life expectancy at age x at current death rates. There may be special circumstances when the second condition is true but the two life expectancies turn out to be the same. In particular, at some ages x lives might be saved for less than remaining life expectancy and at other ages by more and the effects might cancel out. Such a coincidence, however, will be unusual. Bongaarts' and Feeney's delayed-death model is a special, extreme case of the second condition. Other cases include the stretched-lifetime model and various heterogeneity models. The heterogeneity among individuals does not have to be fixed and innate: the heterogeneity can be acquired as individuals experience various events that weaken or strengthen them.

Because the phrase "tempo distortions of mortality" is generally used with reference to the delayed-death model, some broader phrase should be used to describe the general fact that when mortality is changing conventional lifetables do not, except in special circumstances, describe the age-trajectory of mortality that a cohort would experience under current conditions. Let me suggest use of the phrase "the theory of mortality turbulence" to allude to the general phenomenon. The notion is that, except in special cases, mortality change creates a wake of turbulence, of disequilibrium, that temporarily distorts death rates. In this case, the current rates may not equal the death rates that will eventually prevail when the turmoil ceases.

The turbulence could be due to various factors. In addition to delays in death, stretched lifespans, and the impact of differential mortality in heterogeneous populations, it may be the case that the longer individuals have lived under the unfavorable mortality regime, the more debilitated they are and the less they are to benefit from more favorable conditions. On the other hand, individuals who have survived the old regime may have been strengthened (in terms, say, of immune response). Such "hormesis" may enable them to benefit more from the new regime than cohorts born under the new regime. The shift in mortality regime may be disturbing to individuals: they may need time to adapt to the new conditions. For example, animals brought from the wild

into a zoo or laboratory may suffer especially high mortality during an initial period.

A broader theory of demographic disturbance might be developed to study transient distortions produced by fertility change, marriage change, etc. Bongaarts and Feeney (in this volume p. 29) argue that tempo distortions influence a variety of demographic rates. Similarly and more generally, there may be a wake of turbulence following change in fertility, marriage and other demographic regimes. Such turbulence will occur if period statistics are based on data that reflect how many events (e.g., births or marriages) are averted in the period but do not capture the length of time the events are postponed.

Empirical research is required to test hypotheses arising from the theory of mortality turbulence. Data on cohort mortality can be used to distinguish between interventions that stretch lifetimes vs. those that lower age-specific mortality but do not decelerate the rate of increase in death rates with age (Mair et al. 2002). What are urgently needed are empirical tests that distinguish between the conventional view of lifesaving and the Bongaarts-Feeney delayed-death model. It seems clear that both perspectives are wrong and that true period life expectancy probably lies somewhere between the conventional estimate and a delayed-death estimate. But where in-between? It may well be that for remaining life expectancy at age 30 in developed countries since 1950 Bongaarts and Feeney are closer to the truth. Indirectly relevant evidence can be advanced to support their position. The discussion, however, will remain speculative until direct tests can be developed. This is the key challenge today for basic research on mortality.

The lightbulb experiments being conducted at the Max Planck Institute for Demographic Research are a first step. Experiments with various animal models, flies or nematode worms for instance, will also be important. As theoretical understanding of mortality turbulence develops and as empirical results are found in laboratory experiments, then it may become possible to develop and refine strategies for analyzing human data. If the Bongaarts-Feeney delayed death model is consistent with the results of laboratory experiments, then it might also hold for human populations under some circumstances. More generally, the lightbulb and other experiments will help illuminate how the terrain of the triangle of mortality turbulence is shaped by different kinds of mortality change.

Acknowledgements

For constructive discussions and comments, I thank Kenneth Wachter, John Bongaarts and Griff Feeney, Annette Baudisch, Jutta Gampe and Elisabetta Barbi, and two anonymous reviewers.

References

Bongaarts, J. Five period measures of longevity. *In this volume, also published in Demographic Research*, 13(21):547–558. 2005.

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. The quantum and tempo of life cycle events. *In this volume, also published in Vienna Yearbook of Population Research*, pages 115–151. 2006.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Carey, J. R., Liedo, P., Müller, H.-G., Wang, J.-L., and Vaupel, J. W. (1998). Dual modes of aging in mediterranean fruit fly females. *Science*, 281(5379):996–998.

LeBras, H. Mortality tempo versus removal of causes of mortality: opposite views leading to different estimations of life expectancy. *In this volume, also published in Demographic Research*, (13):615–640. 2005.

Mair, W., Goymer, P., Pletcher, S. D., and Partridge, L. (2003). Demography of dietary restriction and death in drosophila. *Science*, 301:1731–1733.

Vaupel, J. W. (2002). Life expectancy at current rates vs. current conditions. *Demographic Research*, 7(8):365–378.

Vaupel, J. W., Manton, K. G., and Stallard, E. (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography*, 16:439–454.

Vaupel, J. W. and Yashin, A. I. (1985). Heterogeneity's ruses: Some surprising effects of selection on population dynamics. *American Statistician*, 39(3):176–85.

Vaupel, J. W. and Yashin, A. I. (1987a). Repeated resuscitation: How lifesaving alters life tables. *Demography*, 24(1):123–135.

Vaupel, J. W. and Yashin, A. I. (1987b). Targeting lifesaving: Demographic linkages between population structure and life expectancy. *European Journal of Population*, 2(3-4):335–360.

Vaupel, J. W., Yashin, A. I., and Manton, K. (1988). Debilitation's aftermath: Stochastic process models of mortality. *Mathematical Populations Studies*, 1(1):21–48.

Wachter, K. W. Tempo and its tribulations. *In this volume, also published in Demographic Research*, 13(9). 2005.

Tempo and its tribulations*

Kenneth W. Wachter

2232 Piedmont Ave University of California, Berkeley, CA 94720-2120, USA.
E-mail: wachter@demog.berkeley.edu

Summary. Bongaarts and Feeney offer alternatives to period life expectancy with a set of demographic measures equivalent to each other under a Proportionality Assumption. Under this assumption, we show that the measures are given by exponentially weighted moving averages of earlier values of period life expectancy. They are indices of mortality conditions in the recent past. The period life expectancy is an index of current mortality conditions. The difference is a difference between past and present, not a "tempo distortion" in the present. In contrast, the Bongaarts-Feeney tempo-adjusted Total Fertility Rate is a measure of current fertility conditions, which can be understood in terms of a process of birth-age standardization.

1 Tempo

In the study of fertility, a distinction between quantum and tempo in the spirit of Norman Ryder (1964) is universally acknowledged. A woman may have more or fewer children, and she may have them earlier or later in her life. It makes sense to ask for period measures of total fertility which adjust for changes in the timing of childbearing independent of changes in numbers of children at the individual level. John Bongaarts and Griffeth Feeney (1998) provided such a fertility measure which has gained many adherents, including the present author.

In the study of mortality, no distinction between quantum and tempo exists at the individual level. A person has one death, his or her own, and mortality pertains to whether death comes early or late. It makes no obvious sense to adjust away the effects of changes in the timing of death, thus adjusting away changes in mortality itself. New papers by Bongaarts and Feeney (2002) and (in this volume p. 11) came as a surprise, offering a family of measures put forward to adjust period life expectancy for effects which they called tempo distortions. The different measures in the family coincide with each other under a condition on the age and time-specific hazard rates

called the "Proportionality Assumption" which the authors find to be approximately satisfied by adult mortality schedules in various developed countries over some recent decades.

Any measure measures something. The question is whether the something being measured is a version of current period life expectancy freed from some kind of distortion. This chapter puts the spotlight on a representation which helps in visualizing what the new measures do measure. The new measures do not measure current mortality conditions but rather the cumulative effects of earlier mortality conditions. The period life expectancy does measure current mortality conditions.

The words "current conditions" are used here in their ordinary English-language sense. Current mortality is the mortality that can be currently observed by counting deaths and counting person-years at risk. An alternative usage introduced by Vaupel (2002) in which "current conditions" is used as shorthand for "current latent conditions" in a latent-structure representation is discussed in Section 6.

The representation of the Bongaarts-Feeney measures takes the form

$$M(t) \approx \int_{-\infty}^{t} w_t(\tau) e_0(\tau)\, d\tau \tag{1}$$

Here $e_0(t)$ is period life expectancy at time t. (In applications, e_0 is replaced by e_{30} since the approach is intended solely for adult mortality.) $M(t)$ is a Bongaarts-Feeney measure of adjusted life expectancy. For each t, $w_t(\tau)$ is a probability distribution defining weights over a set of lagged time periods $\tau < t$. As functions of the lag $s = t - \tau$, the weights are nearly exponential and nearly independent of t.

The representation is an approximation which holds to first order in the time derivative of M under the hypothesis that Bongaarts and Feeney's Proportionality Assumption is sufficiently nearly satisfied that the different measures in the family are equivalent to each other within the limits of the approximation. Details are spelled out in Section 3.

The representation shows that the Bongaarts-Feeney measure M is a weighted average of period e_0 values from the recent past. The period life expectancy itself at time t depends only on current age-specific hazard rates for time t. The Bongaarts-Feeney measure depends on past as well as current age-specific hazard rates. When longevity has been increasing, past values of e_0 are lower than current values, and the Bongaarts-Feeney measure averages over these lower past values and produces a value below present-day e_0. When longevity has been decreasing, past values exceed current values, and the Bongaarts-Feeney measure averages over these higher past values and hovers above present-day e_0.

The word "distortion" is out of place when contrasting M to e_0. The measures measure different things. If one cares about average mortality levels in the recent past, one can use one of the Bongaarts-Feeney measures. If one

cares about mortality levels under current conditions, one can use the period life expectancy.

The representation (1) gives concrete form to the general observation that the Bongaarts-Feeney mortality measures are functions not solely of current mortality but also of the population age structure that would be produced by past mortality conditions given a hypothetical constant stream of prior births. This dependence was pointed out in their initial paper (2002 ,p. 23). Bongaarts and Feeney noted that their adjusted measure could not be calculated directly from period hazard rates "because $\mu^*(a,t)$ [their adjusted hazard rates] are in general not observable". They discussed a need for a century or more of age-specific death rates for their calculations.

In this same early paper, Bongaarts and Feeney (2002, Eq. 12), introduced a differential equation (originally under Gompertzian assumptions) which agrees to first order with equation (7) of Section 3. They imposed a boundary condition which allowed them to estimate values of their measure at each time t from the sequence of prior values of period life expectancy, in effect implementing a numerical calculation of the representation (1). The equation for $M(t)$ in terms of coefficient values for time t is a differential equation, not an algebraic equation. It is therefore not a recipe for calculating the value of M at time t solely from period information for time t. The solution M is only defined with respect to the boundary conditions and time trajectories of the coefficients. This dependence on the past is the fundamental property of the Bongaarts-Feeney mortality measures.

Definitions of the measures are given in Section 2. The representation is presented in Section 3 with examples in Section 4 and discussion in Section 5. Proposals to relate the Bongaarts-Feeney measures to latent structure representations of mortality are analyzed in Section 6. Unlike the adjusted life expectancies, Bongaarts and Feeney's adjusted total fertility measure at a time t depends only on age-specific fertility rates in an arbitrarily small neighborhood of t. It is independent of population age structure and independent of past levels of fertility. This fundamental difference between the proposed mortality adjustments and the fertility adjustments precludes any close analogy between them. The difference is highlighted in Section 7, which presents an interpretation of the fertility adjustments in terms of a process of birth-age standardization.

2 Measures

Clarity is promoted by expressing the measures under discussion in standard demographic notation.

$\mu(a,t)$ is the hazard rate at age a at time t;

$N(a,t) = N(0,t)\exp(-\int_0^a \mu(x,t-a+x)\,dx)$ is the number of population members aged a at time t expressed as a density with respect to $da\,dt$;

$N(0,t) = 1$ is a normalization on initial cohort size which keeps the number of births per unit time constant at unity;
$e_0(t) = \int \exp\left(-\int_0^a \mu(x,t)\,dx\right) da$ is the period expectation of life;
$d(a,t) = N(a,t)\mu(a,t)$ is the count of deaths at age a and time t;
$D_+(t) = \int N(a,t)\mu(a,t)\,da = \int d(a,t)\,da$ is the period count of total deaths;
$N_+(t) = \int N(a,t)\,da$ is the period total population;

The basic condition on the population distribution $N(a,t)$ is the normalization which sets the size of every cohort at birth equal to unity, equivalent to dividing the numbers aged a at time t by the numbers aged 0 at time $t-a$ for all a and t. Given this normalization, the measures $M_1 \ldots M_4$ introduced in the notation of their *PNAS* article (Bongaarts and Feeney 2003, also published in this volume p. 11) correspond to familiar population quantities:

- M_1 is the total population count $N_+(t)$, equal to the "Cross-Sectional Average Length of Life" $CAL(t)$ introduced by Nicolas Brouard (1986) and Michel Guillot (2003);
- M_2 is the period mean age at death, $MAD(t)$ in the terminology of Bongaarts and Feeney (in this volume p.29) , given by

$$\int aN(a,t)\mu(a,t)\,da/D_+(t);$$

- M_3 is the period life expectancy $e_0(t)$;
- M_4 is an adjusted life expectancy defined by

$$M_4(t) = \int \exp\left(-\int_0^a \frac{\mu(x,t)}{1-\frac{d}{dt}M_1(t)}\,dx\right) da \tag{2}$$

In Bongaarts and Feeney (in this volume p.29), the derivative of M_1 in (2) is replaced by the derivative of M_2, producing a closely related measure which might reasonably be called M_5.

The total population count changes over time by the addition of births and subtraction of deaths, so the time derivative of $N_+(t) = M_1(t) = CAL(t)$ is $1 - D_+(t)$. Dividing the hazard rates for time t at every age by the count of total deaths, retaining an unchanged population $N(a,t)$ at risk, resets the total deaths to unity. In other words, the rates inside the integral in the definition of M_4 are rates which, given the age structure, would make period deaths equal normalized period births. Caution is advisable in interpreting these measures. The measure CAL does not always correspond to the statistical expectation of a waiting time, even though the formula might seem to suggest so. The measure M_4 employs a proportional adjustment to hazards, whether or not hazards have been changing proportionally in the past.

The "Proportionality Assumption" of Bongaarts and Feeney (in this volume p.11) is a condition on the partial derivatives of $N(a,t)$ for all a and t in terms of a function $r(t)$ varying in a neighborhood of zero:

$$\frac{\partial N(a,t)}{\partial t} = -r(t)\frac{\partial N(a,t)}{\partial a} \tag{3}$$

(This $r(t)$ is the same as $1-p(t)$ in Bongaarts and Feeney (in this volume p.11, Eq. 6).) It should be borne in mind that the condition expressed in terms of N for given a and t involves a whole family of constraints on the hazard rates μ at earlier ages and earlier times which produce the value of N and its rates of change with age and time. It is not a local condition confined to a neighborhood of a and t.

Equation (3) determines a family of parallel curves giving contours of constant N over time. The shape of the age distribution is preserved and shifted up or down as shown in Bongaarts and Feeney (in this volume p.11). Specifically, setting $F(t) = \int_0^t r(\tau)\, d\tau$, (3) provides for a vanishing time derivative for $N(a+F(t),t)$, allowing $N(a,t)$ to be expressed in terms of $N(a,0)$. The hazards $\mu(a,t)$, defined from the partial derivatives of the logarithm of N at time t and hence from the partial derivatives at time zero, have to take the form

$$\mu(a,t) = (1-F'(t))\psi(a-F(t)) \tag{4}$$

Here ψ is a non-negative function of age a vanishing for negative a, defined from derivatives of the logarithm of N at time zero.

Three other results proved in Bongaarts and Feeney (in this volume p.11) follow readily from (3). Integrating both sides of (3) with respect to a shows that the time derivative of $M_1(t)$, that is, of $CAL(t)$, is given by $M_1'(t) = 1-D_+(t) = r(t)$. Integrating $\int ad(a,t)da = \int aN(a,t)\mu(a,t)da$ by parts yields the equality $M_2 = M_1$. Writing the hazard rate quotient $\mu(a,t)/(1-r(t))$ as the partial derivative with respect to a of $-\log(N(a,t))$ shows that $M_4 = M_1$.

3 Representation of M

When the Proportionality Assumption holds, the equality of M_1, M_2, and M_4 allows us to set $M = M_1 = M_4$ in the equation defining M_4 and obtain a differential equation satisfied by the common values of M_1, M_2, and M_4:

$$M(t) = \int \exp\left(-\int_0^a \frac{\mu(x,t)}{1-\frac{d}{dt}M(t)}\, dx\right) da \tag{5}$$

When the Proportionality Assumption does not hold exactly, this equation can also be regarded as defining a measure of interest in its own right, which could take a place beside M_1, M_2, and M_4 in the family of measures. Indeed, the original measure introduced in (2002, 23) was a solution to a version of

this equation. It is expected that all these measures will be close to each other when the Proportionality Assumption is approximately valid. One could, for example, stipulate that $\mu(a,t)$ agree to first order in some parameter ϵ with the corresponding values for a set of hazard rates that do satisfy the Proportionality Assumption. Weaker conditions might also suffice to guarantee agreement to order $O(\epsilon)$ among the measures. All that is at stake here is approximate consistency among the different choices of measures in the family. Once Equation (5) is in hand, the further arguments leading to our representation do not depend on the Proportionality Assumption.

We obtain our representation by expanding the right-hand side of (5) in powers of $r = M'(t)$ for each t. The value of the right-hand side at $r = 0$ is the period life expectancy. The inner integrand $\mu/(1-r)$ in (5), being proportional to μ, brings into play the familiar machinery of proportional hazards. As in (1985, 80), the derivative with respect to r is a multiple of "lifetable entropy" given, at $r = 0$, by minus the quantity

$$g(t) = \int_0^\infty e^{-\int_0^a \mu(x,t)\,dx} \int_0^a \mu(y,t)\,dy\,da \tag{6}$$

The result is an equation which is a first-order approximation to (5) when $M'(t)$ is uniformly small:

$$M(t) = e_0(t) - g(t)M'(t) \tag{7}$$

Under appropriate regularity conditions mentioned below, the differential equation (7) has a unique solution bounded at minus infinity given by the integral already presented in Equation (1):

$$M(t) = \int_{-\infty}^t w_t(\tau)e_0(\tau)\,d\tau \tag{8}$$

The time-dependent weights $w_t(\tau)$ are given in terms of the reciprocals of $g(\tau)$ by the expression

$$w_t(\tau) = g^{-1}(\tau)\exp\Big(-\int_\tau^t g^{-1}(s)\,ds\Big) \tag{9}$$

For each t, these positive weights integrate up to unity over τ and define a probability distribution. The inner integral in (9) can be used to define an alternative time-like coordinate in terms of which the weights become exponential functions.

It is easy to verify that (1) formally satisfies (7) by differentiating the right-hand side of (1) with respect to the argument t which occurs both in the limit of integration and in the function $w_t(\tau)$. The derivative of $w_t(\tau)$ with respect to t is $-w_t(\tau)/g(t)$ and $w_t(t) = 1/g(t)$.

$$
\begin{aligned}
e_0(t) - g(t)M'(t) &= e_0(t) - g(t)\frac{d}{dt}\int_{-\infty}^{t} w_t(\tau)e_0(\tau)\,d\tau \\
&= e_0(t) - g(t)w_t(t)e_0(t) - g(t)\int_{-\infty}^{t}\frac{d}{dt}w_t(\tau)e_0(\tau)\,d\tau \\
&= e_0(t) - g(t)(1/g(t))e_0(t) - \int_{-\infty}^{t} -w_t(\tau)e_0(\tau)\,d\tau \\
&= M(t)
\end{aligned}
$$

The function $g(t)$ is strictly greater than zero, so long as lifetable deaths in the period lifetable are not concentrated all at a single age, which is always true if μ is finite. We assume further that $1/g(t)$ and $e_0(t)/g(t)$ are integrable on bounded intervals and that $g(t)$ is bounded, making the weights in (9) finite and the solution in (8) the unique one bounded at minus infinity (1955, pp. 67,97).

In expanding the right-hand side of (5), we could have expressed the difference between the values at zero and at r using the derivative evaluated at r instead of at zero. The answers would agree to first order. The derivative at zero from (6) has the advantage of being a purely period measure. But the derivative at r, obtained from (6) by substituting $\mu/(1-r)$ for μ, is also informative. It is exactly constant when the Proportionality Assumption is exactly valid. It follows that $g(t)$ must be nearly constant so long as the Proportionality Assumption is nearly valid, making the weights $w_t(t-s)$ as a function of the lag s nearly equal to a fixed exponential distribution $(1/g)\exp(-s/g)$.

A clear conclusion follows from this representation: This candidate for a "tempo-adjusted expectation of life" is, to first order, an explicit moving average of recent past values of the period expectation of life. When levels of survival are increasing, current values of $e_0(t)$ exceed past values. What Bongaarts and Feeney are interpreting as a "tempo distortion" is simply the difference produced by focussing on the present instead of focussing on the recent past.

Period life expectancy is sensitive to sudden changes affecting mortality at many ages. It is meant to be so. That is an advantage, not a drawback. When period life expectancy falls, deaths are surging. People are dying. It is no mirage or distortion of reality.

A rise or fall in hazard rates concentrated in time but spread over many ages will have effects spread over many cohorts, so a large temporary change in period life expectancy should and does correspond to a suite of small changes in cohort life expectancy for many cohorts. Averaging period measures over a stretch of time that includes large parts of the lifespans of many cohorts naturally leads to values in line with the average values of the corresponding cohort measures. The retrospective averaging implemented by the Bongaarts-Feeney measures has this kind of outcome. The period life expectancy, for its part, is a faithful indicator of current conditions.

4 The moving average

To see how the representation of the Bongaarts-Feeney measures works out in practice, consider Swedish female adult mortality, example B of Bongaarts and Feeney (in this volume p.11, Figure 6). The measures are only meant to apply after about age 30, so we let age $a = 0$ correspond to age 30 and condition on survival to that age. Single-year age-specific mortality rates from 1861 to 2001 are taken from the Human Mortality Database (2004) assembled by John Wilmoth at Berkeley, allowing calculation of CAL and MAD for ages above 30 from 1941 onwards.

In these Swedish data, the entropy measure g (for ages above 30) is close to 9 back to about 1945, a level reached after a gradual long-term drop from Nineteenth Century values around 13. The gradual changes in g imply slight changes in exponential weights, but for measures after 1941 the moving average (8) with changing weights (9) is only slightly different from a moving average with fixed exponential weights set with $g = 9$. (The mean difference is 0.063 years and the maximum difference is 0.186 years.) Thus we are essentially dealing with a simple exponential distribution with a nine-year mean. The Bongaarts-Feeney measures CAL, MAD, and M_4, where they agree with each other, are given by a simple exponential weighted average of past values of period life expectancy, with an average look-back time of 9 years.

For example, consider the calculation of M for $t = 2001.0$. The year from December 2000 back to January 2000 is the first year back. The weight for this year, applied to period life expectancy centered at mid-year, is the integral of $(1/9)\exp(-s/9)$ between 0 and 1, or $e^{-0/9} - e^{-1/9}$. The weight for the second year back (1999) is $e^{-1/9} - e^{-2/9}$, etc. M is the weighted average, the sum of weights times life expectancies back over time:

$$\begin{aligned} M &= (e^{-0/9} - e^{-1/9})e_{30}(2000) + (e^{-1/9} - e^{-2/9})e_{30}(1999)\ldots \\ &= (0.10516)(52.587) + (0.09410)(52.451)\ldots \\ &= 51.55 \end{aligned}$$

For 2001, comparing M to values of CAL, MAD, and M_4 calculated directly from single-year mortality rates, we see that the weighted average $M = 51.55$ years falls a little above $CAL = 51.43$ years between $M_4 = 51.52$ years and $MAD = 51.58$ years. The period life expectancy e_{30} is a year higher, at 52.63 years.

It is instructive to see with formulas how the weighted average recovers the values of CAL and MAD when the Proportionality Assumption holds. As before, we let $a = 0$ correspond to human age 30. Thanks to (4), we have $\mu(a,t) = (1 - F'(t))\psi(a - F(t))$ with a baseline age schedule ψ and a shift function $F(t)$ whose time derivative equals the proportionality factor $r(t)$. Values of CAL and MAD at time zero are given by $\eta = \int exp(-\int_0^a \psi(x)dx)da$ and the values at time t include the shift $F(t)$:

$$CAL(t) = MAD(t) = \eta + F(t) \tag{10}$$

The same Taylor expansion as in (7) for life expectancies under proportional hazards yields

$$e_0(t) \approx \eta + F(t) + gF'(t) \tag{11}$$

Here the coefficient g can be set equal to the rescaled entropy derived from ψ which is constant over time. It is given by formula (6) with $\psi(x - F(t))$ in place of $\mu(x, t)$. Since ψ vanishes for negative a and the outer integral runs over all a, the formula is unchanged when $F(t)$ is deleted from the arguments of ψ, leaving an expression independent of t.

The weights are given by $w_t(t - s) = (1/g)\exp(-s/g)$. The weighted average is an integral with respect to this exponential probability distribution whose mean is g:

$$\begin{aligned} M &= \int_0^\infty e_0(t-s)(1/g)e^{-s/g}\,ds \\ &= \int (\eta + F(t-s) + gF'(t-s))\,(1/g)e^{-s/g}\,ds \\ &= \eta + F(t) - \int (F(t) - F(t-s))(1/g)e^{-s/g}\,ds \\ &\qquad + \int F'(t-s))\,e^{-s/g}\,ds \end{aligned}$$

Integrating the third term by parts yields $-\int F'(t-s)e^{-s/g}\,ds$, exactly cancelling the fourth term, so that

$$M = \eta + F(t) = CAL(t) = MAD(t) \tag{12}$$

When the proportionality factor $r(t) = F'(t)$ is constant, we have the case of linear shifts analyzed by Goldstein (in this volume) and by Rodriguez (in this volume). The graphs of $e_0(t)$ and $CAL(t) = MAD(t)$ are parallel straight lines with slope r. Lagged life expectancy is the linear function $e_0(t - s) = e_0(0) + r(t - s)$. Its average is $e_0(0) + r(t - g)$ since the average value for s is g. Thus $CAL(t)$ comes out to be the lagged value $e_0(t - g)$.

When g is calculated from a hazard function given by a Gompertz model $\alpha e^{\beta a}$, we have $g = (1/\beta) - (\alpha/\beta)e_0$. The second term is usually two orders of magnitude smaller than the first term, so $g \approx 1/\beta$. Suppose that hazards change over time according to a Gompertz model with constant β and more or less exponentially declining $\alpha(t)$ approximated, say, by $\alpha(0)\exp(-r\beta t)$. Suppose also that $\alpha(0)$ is small enough that young mortality can be neglected or set to zero. Then the Proportionality Assumption comes to be satisfied with something close to a linear shift of slope r. In principle the Proportionality Assumption could hold under different, non-Gompertzian conditions, but in

the empirical examples known to the present author it seems to arise in this way.

Since the weights in the moving average representation fall off exponentially, the remote past has negligible impact, and the full moving average can be replaced by an average reaching back over a finite span of years. The representation is meant to hold to first order in M'. In the Swedish data, M' is on the order of 0.15 and second-order terms are on the order of 0.02. A span of $6g$ years, or 54 years, includes all but $\exp(-6g/g) = \exp(-6) = 0.002$ of the weight from the exponential distribution. Periods that represent the early adult life experience of cohorts older than $30 + 54 = 84$ years have only minor impact on CAL and MAD.

Mathematically speaking, when the Proportionality Assumption is only tenable for some limited span $t > T$, the solution (8) to the differential equation (7) (which is the solution vanishing at minus infinity) needs to be replaced by the solution satisfying an appropriate boundary condition at $t = T$, that is, one making $M(T) = CAL(T)$. The moving average only reaches back to T and the term introduced by the boundary condition tapers exponentially as time goes by.

Figure 1 shows mortality measures for Swedish women from 1941 to 2001, all calculated beyond age 30. The upper solid line is period life expectancy. The lower solid line is CAL, trending steadily upward with an average slope of 0.17 per year. The dashed line for MAD hugs CAL from 2001 back to 1975 but separates from it at earlier times just outside the range of years shown in Bongaarts and Feeney (in this volume p.11, Figure 6B). The separation signals failure of the Proportionality Assumption. The moving average M is the dotted line. The measure M_4, not shown in the plot, is close to M before 1970 and close to CAL after 1980. Where CAL and MAD diverge from each other, the moving average M turns out to strike a balance between them.

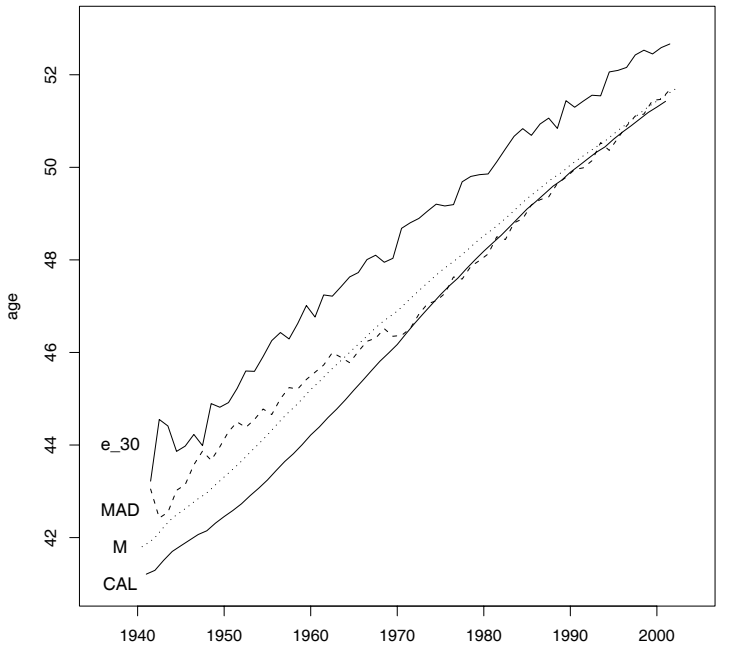


Fig. 1. Mortality measures for Swedish women 1941-2001.

5 Period counts of deaths

Period counts of deaths play an important role in the formulas for the mortality measures and an important role in the analogies which Bongaarts and Feeney (in this volume p.29) seek to develop. In their papers they give a new name to the period count of deaths $D_+(t)$, calling it the "Total Mortality Rate" or "*TMR*". They liken this quantity to the Total Fertility Rate, Total First Marriage Rate, and other indices for processes that, unlike mortality, admit a distinction between quantum and tempo at the individual level.

Ordinarily, one would expect instead to define the "*TMR*" with a formula parallel to the formula for the *TFR*:

$$TFR(t) = \int f(a,t)\, da \tag{13}$$

$$TMR(t) = \int \mu(a,t)\, da \tag{14}$$

The period count of deaths is a count, not a rate. Bongaarts and Feeney defend their practice of calling it a rate by taking the usual denominator, those at risk of the event, and adding on a set of "ghosts", those who would have been at risk had they not exited from the population by dying. The same construction can be applied with fertility to obtain period counts of births $B_+(t)$ from the fertility rates, albeit counts that need not agree with initial cohort sizes:

$$B_+(t) = \int N(a,t)f(a,t)/N(0,t-a)\,da \tag{15}$$

$$D_+(t) = \int N(a,t)\mu(a,t)/N(0,t-a)\,da \tag{16}$$

The tempo adjustment for fertility in (1998) is an adjustment to the *TFR*, not $B_+(t)$, whereas the tempo adjustments for mortality in Bongaarts and Feeney (in this volume p.11) involve adjustments to $D_+(t)$, not to the *TMR*, which is generally infinite.

The normalization which enforces a constant unit stream of births into the population means that the population is increasing when and only when $D_+(t)$ is less than 1, that is, when births exceed deaths, and decreasing when $D_+(t) > 1$. This quantity $D_+(t)$, the period count of deaths per unit birth, is less than 1 if mortality has been higher in the past than in the present. The higher death rates of the past deplete the surviving population at risk of dying and thus reduce current deaths. This outcome is not a tempo effect. It can remain true even if current mortality is increasing rather than declining.

Replacement of the hazard rates $\mu(a,t)$ by rates $\mu(a,t)/D_+(t)$ in the formula for M_4 does, as mentioned, bring total deaths into equality with normalized total births so long as the population age structure is retained unaltered. However, this transformation cannot be achieved by a systematic reassignment of times of death, because any reassignment necessarily alters the population age structure. The substitution underlying the M_4 measure is a form of standardization for the total flow of deaths which is difficult to interpret in terms of any assumptions about individual experience.

6 Current latent conditions

A question arises as to whether measures equivalent or similar to those of Bongaarts and Feeney might be definable from some latent structure representation of mortality. Vaupel (in this volume p.93) writes about such possibilities. An example predicated on the heterogeneous frailty model of Vaupel, Manton, and Stallard (1979) is given by Vaupel (2002). Starting from any $\mu(x,t)$, for each choice of a frailty dispersion parameter σ, one can define hypothetical latent baseline hazards $\mu^o(x,t)$ by the equation

$$\mu^o(x,t) = \mu(x,t)\exp\bigl(\sigma^2 \int_0^x \mu(a, t-x+a)\, da\bigr) \tag{17}$$

This formula is a *representation.* For any observed $\mu(x,t)$ it supplies a latent $\mu^o(x,t)$ which will reproduce it. From μ^o, Vaupel defines a measure which he calls a version of life-expectancy "under current conditions", that is, under current latent rather than current observed conditions.

Vaupel's frailty-based measures are well defined but they are at a far remove from the Bongaarts-Feeney measures. They depend on population heterogeneity, whereas Bongaarts and Feeney's arguments apply to wholly homogeneous populations. In empirical cases like the Swedish series, the frailty-based measures fluctuate in tandem with period life expectancy, lack the smoothing properties of *CAL*, *MAD*, and M_4, and differ only by small amounts from period life expectancy.

The interesting feature of the frailty-based measures is conceptual. Although current μ^o is calculated from past values of μ, one can imagine an experiment for measuring current μ^o from current observations. Take a random sample of people who had lived in a country with negligible mortality up to age x, transplant them to a country beset by μ, and identify μ^o with any higher hazards that such higher-mean-frailty refugees experience. In practice, debilitation probably dominates culling, and the experiment would founder, but the concept is coherent.

Recognizing the absence of connection between his frailty-based measures and the actual Bongaarts-Feeney measures, Vaupel (in this volume p.93) goes on to sketch a different approach which might also come under the heading of "mortality under current latent conditions". The latent variables are tickets associated with predestined ages of death. Life is like a pastiche of an old Beatles song

"I have a ticket to die."

Vaupel's chapter presents examples rather than a general treatment. In some examples, the proposal is to have ticket values that can change either deterministically or stochastically over time, depending on the current ticket value but not on the current age of the holder. When a person's age catches up with his or her current ticket value, the person dies.

We may write $V(U,t)$ for a ticket process started at an initial state indexed by U and varying over time t. U has some probability distribution across the population. In versions with deterministic transitions, $V(U,t)$ is a function of U and t, usually a continuous function. In versions with stochastic transitions, $V(U,t)$ is a Markov process started at a state indexed by U unfolding either with discrete time steps and discrete states corresponding to age groups, or with continuous time and age. The distribution of ticket values at birth for a cohort born at time τ is the marginal distribution of $V(U,\tau)$ generated by the randomness in U and the randomness, if any, in V given U. The distribution of ages at death for the cohort is the distribution of the random variable

$$\min\{x : V(U, \tau + x) \leq x\} \tag{18}$$

A person dies when he or she first reaches an age coinciding with the age currently on his or her ticket.

Detailed treatment is beyond the scope of this chapter, but we proffer some reflections based on early analysis.

If $V(U, t)$ can be specified, then a current measure can be defined to equal the period mean of V. That part is easy. What is difficult is the representation problem. No equation like (17) is at hand for taking observed $\mu(x, t)$ and writing down some specific V that generates it. Without a representation formula, one has no well-defined measure and nothing to compare with Bongaarts and Feeney's proposed adjustments.

One can, of course, make up ticket models *de novo* and endeavor to test their goodness of fit to μ values like the Swedish series. That may be interesting, but testing goodness of fit is not what Bongaarts and Feeney are doing. They are defining measures. From any μ, they obtain measures to contrast with period life expectancy, and they argue for an automatic adjustment to period life expectancy whenever observed past hazards differ from present ones.

To make ticket models relevant to Bongaarts and Feeney's proposals, one needs, then, to focus on the representation problem. With deterministic transitions, the only apparent prospect is a version of Feeney's (in this volume) derivations. See also Wilmoth (2005). We can let U be a uniform random variable marking a cohort member's predestined proportional placement in a rank ordering of the cohort from oldest to youngest by age at death. Define the quantile function

$$Q(U, \tau) = \min\{x : \int_0^x \mu(a, \tau + a)\, da = -\log(U)\} \tag{19}$$

For each fixed U and t , the equation $Q(U, t - v) = v$ may have a unique solution v, and if it does, we can set $V(U, t) = v$. In such cases the measure, the period mean of V, comes out to equal CAL.

However, unique solutions do not always exist. The same cases that defeat Feeney's (in this volume) attempt at generality prevent this construction from yielding a general representation of mortality schedules. Cases that fail occur when the partial derivative of Q with respect to τ takes values less than or equal to -1. These tickets are intrinsically cohort objects that resist alignment by periods. A person's U value is a cohort percentage. Today's ticket values only have meaning insofar as we match values for current survivors to values for current decedents who share the same U, fixed by their cohort's prior history. Unlike Vaupel's frailty-based μ^o values, the current values of these latent variables have no independent reality in the present that can be easily discerned. No experiment is on the table which would allow us to elicit present-day ticket values from present-day observations alone.

Turning to ticket models with stochastic transitions, we encounter the representation problem in a different guise. Here the specification of $V(U,t)$ is drastically underdetermined. Analysis in continuous time is technically challenging, but the issues can be scrutinized in discrete time with Markov chains with finitely many states corresponding to age groups numbered from 1 to k. Each transition matrix at each time t contains $k(k-1)$ elements that need to be determined. The observed distribution of deaths for each cohort, which the model has to match, is specified by $k-1$ quantities. Thus, ignoring endpoint effects, T cohorts give $(k-1)T$ equations in $k(k-1)T$ unknowns. Already with $k=3$, a wide range of different solutions are allowed. Subject to some messy inequalities, one can choose one's solution at will to make the resulting period measure agree with any of a wide variety of arbitrary sequences. Without some natural set of identifying restrictions, as yet to be discovered, the ticket model framework with stochastic transitions gives nothing definite to compare with Bongaarts and Feeney's measures.

7 Total fertility

It would be an unhappy outcome if the limitations of the proposed measures for adjusted life expectancies undermined confidence in the tempo-adjusted measures for total fertility proposed earlier by Bongaarts and Feeney (1998). Unlike the mortality measures, the fertility measures are standardized indicators of current conditions. The adjusted total fertility rate at time t depends only on age-specific fertility rates $f(a,t)$ in an arbitrarily small neighborhood of t. It does not depend on age structure and it does not depend on past fertility rates. It has a direct interpretation in terms of individual experience. This section offers a formulation of the adjusted fertility measures which highlights these attractive features.

Age-specific fertility rates $f(a,t)$ are written here as a function of continuous age a and continuous time t. As usual, the period Total Fertility Rate $TFR(t)$ and period mean age at childbearing $A(t)$ are given by

$$TFR(t) = \int f(a,t)\, da \tag{20}$$

and

$$A(t) = \frac{\int a\ f(a,t)\, da}{TFR(t)} \tag{21}$$

A simple procedure for producing an adjusted index is to define a coordinate transformation which, in effect, reassigns the timing of births within cohorts leaving numbers of births invariant within cohorts. The transformation is chosen so that, after reassignment has been carried out, a period computation of mean age at childbearing would give a constant outcome, thus erasing period variations in timing. The post-reassignment value for the mean

age can be set arbitrarily to some standard value A_s, perhaps most sensibly to a long-term average for cohort mean ages at childbearing conditional on survival through childbearing years.

The transformation Ψ is given by

$$a \to \alpha = a - A(t) + A_s \tag{22}$$

$$t \to \tau = t - A(t) + A_s \tag{23}$$

We assume that $A(t)$ is differentiable and we impose the reasonable assumption that the period mean age at childbearing never increases by as much as a full year per year, so that the time derivative $A'(t)$ is always less than 1. Then the transformation is invertible and has a finite Jacobian given by

$$\frac{\partial\, \alpha, \tau}{\partial\, a, t} = 1 - A'(t) \tag{24}$$

The inverse function $t(\alpha, \tau)$ only depends on τ. Age-specific fertility rates after reassignment are given by

$$\tilde{f}(\alpha, \tau) = \frac{f(a(\alpha, \tau), t(\tau))}{1 - A'(t(\tau))} \tag{25}$$

This definition guarantees agreement between integrals over subsets S in the Lexis plane:

$$\int\int_S \tilde{f}\, d\alpha\, d\tau = \int\int_{\Psi^{-1}S} f\, da\, dt \tag{26}$$

An adjusted or standardized Total Fertility Rate $STFR$ can be defined from $\tilde{f}$:

$$STFR(\tau) = \int \tilde{f}(\alpha, \tau)\, d\alpha = \frac{TFR(t(\tau))}{(1 - A'(t(\tau))} \tag{27}$$

These integrals are taken over α for fixed τ, unlike the double integrals of Equation (26). It is readily verified that the period mean age of childbearing defined from $\tilde{f}$ remains constant at a level A_s and that integrals of $\tilde{f}$ along diagonals of the Lexis diagram are identical to integrals of f itself.

Kohler and Philipov (2001) introduce this Jacobian-based formulation for tempo adjustments, although they deviate from it in the definition of their own generalized measure. The transformation shifts fertility backwards or forwards along cohort lifelines on the Lexis diagram. The cohort quantum of fertility measured by a cohort TFR (conditional on survival) is unchanged. The positioning of births along the lifelines of mothers in the cohort is adjusted in such a way as to hold the transformed period mean age at birth constant at the chosen standard value A_s.

The size of $STFR$ defined by Equation (27) is the same as Bongaarts and Feeney's tempo-adjusted TFR. It is expressed as a function of the hypothetical coordinate τ rather than the real time coordinate t, but, if desired, it can

be attributed back to t, since the transformation is invertible. Although τ depends on the choice of the standard age A_s, the measure itself does not depend on it. The mathematics would be the same if we took A_s equal to zero, but visualization is easier if we take it equal to some realistic benchmark age.

The reassignment process expressed by our coordinate transformation can be regarded as a kind of standardization. It differs from familiar kinds of demographic standardization like the standardization of Crude Birth Rates for effects of age distributions. But it serves a parallel purpose. Just as one asks, "What would a Crude Birth Rate turn into if population age group sizes were set to standard values?", one can ask, "What would a Total Fertility Rate turn into, if period mean ages at childbirth were set to a standard values?" In this sense, the Bongaarts-Feeney tempo adjustment for fertility can be viewed as a process of birth-age standardization.

This way of viewing the measure clarifies several issues. Bongaarts and Feeney's fertility measure does not depend on any behavioral assumptions about fertility, any more than an age-standardized birth rate depends on behavioral assumptions. It does, however, suggest a thought experiment, because one can imagine individuals changing the timing of their births in such a way as to change the observed *TFR* into the adjusted or standardized one.

For applications of their measure, Bongaarts and Feeney recommend applying their adjustment separately parity-by-parity to birth-order-specific frequencies. These are not the same as age and parity-specific rates. Each numerator includes only births of a given parity while the corresponding denominator includes person-years from women of all parities. These quantities sum up to the overall age-specific fertility rates, so they comprise an additive decomposition. Conceptual difficulties arising from reliance on such frequencies or "rates of the second kind" in place of occurrence-exposure rates or "rates of the first kind" have been pointed out by Van Imhoff and Keilman (2000).

As a formal procedure, nothing prevents the kind of standardization achieved by Equation (22) from being applied separately to any additive decomposition of age-specific fertility rates:

$$f(a,t) = \sum_i f_i(a,t) \tag{28}$$

Any such decomposition in terms of some categorization of births can be accommodated. Birth order is one option, but mother's marital status, mother's education, region of birth, and sex of baby are among a host of others. When a transformation is applied to each f_i and the resulting $STFR_i$ are added together to produce an aggregate $STFR$, the result is an index which has been standardized for changes in period mean ages at childbearing within each of the subgroups. No behavioral claims need be at issue. It is probably a mistake to make a fetish of the decomposition by parity. The fact that one particular breakdown among many would allow a complicated re-expression in terms of

occurrence-exposure rates need have no deep bearing on the nature of the adjustment.

In summary, Bongaarts and Feeney's tempo adjustment for the Total Fertility Rate can be viewed as a process of standardization. It erases effects of changes in period mean ages while preserving cohort quantum (conditional on survival). There is a clear distinction at the individual level between something that is being reset and something that is being left invariant. The adjustment does not rely on any behavioral model or structural representation of fertility processes. Like traditional standardized measures, it is a valuable device for comparing cases, controlling for a particular source of variation.

No such process of standardization makes sense in the context of mortality, because there is no distinction at the individual level between something to reset and something to leave invariant. The timing of a person's death is what is being assessed when we assess mortality. Controlling for changes in the timing of death is tantamount to controlling for mortality itself.

Discussions of quasi-behavioral models and structural representations in the context of Bongaarts and Feeney's proposed mortality measures serve to highlight the gulf between these measures and their fertility measure. No elaborate modeling is required with fertility.

Bongaarts and Feeney's adjusted Total Fertility Rate is a current measure, whose value at a time t depends only on values and slopes of age-specific fertility rates at time t. Altogether otherwise, the mortality measures they propose as alternatives to period life expectancy are not current measures. They average over mortality conditions observed in the past. Under the Proportionality Assumption which makes the measures coincide with each other, the measures average over conditions in the past in a particular simple way, as a weighted moving average of prior period life expectancies, as shown in this chapter.

Mortality measures like CAL and MAD are valuable for studying changing hazard schedules, smoothing as they do over sudden changes. Everyone agrees that changing hazards make cohort life expectancies diverge from period life expectancies and that the divergence is worthy of attention. But measures that depend on past hazards serve different purposes from period life expectancy, which depends on current hazards. The past may differ from the present. This fact is not a "tempo" distortion. Adjustments for "tempo" are only meaningful when there is a meaningful distinction between quantum and tempo in individual experience.

References

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. The quantum and tempo of life cycle events. *In this volume, also published in Vienna Yearbook of Population Research*, pages 115–151. 2005.

Bongaarts, J. and Feeney, G. (1998). On the quantium and tempo of fertility. *Population and Development Review*, 24(2):271–291.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Brouard, N. (1986). Structure et dynamique des populations. *Espaces, Populations, Sociétés*, 2(14-15):157–168.

Coddington, E. and Levinson, N. (1955). *Theory of Ordinary Differential Equations*. McGraw Hill, New York.

Feeney, G. Increments to life and mortality tempo. *In this volume, also published in Demographic Research*, 14(2):27–47. 2006.

Goldstein, J. Found in translation? A cohort perspective on tempo-adjusted life expectancy. *In this volume, also published in Demographic Research*, 14(5):71–84. 2006.

Guillot, M. (2003). The cross-sectional average length of life (CAL). *Population Studies*, 57(1):41–54.

Human Mortality Database (February 2004). *Available at www.mortality.org*. University of California, Berkeley, USA, and Max Planck Institut für Demografische Forschung, Rostock, Germany.

Imhoff, E. V. and Keilman, N. (2000). On the quantium and tempo of fertility:comment. *Population and Development Review*, 26(3):549–553.

Keyfitz, N. and Caswell, H. (2005). *Applied Mathematical Demography, Third Edition*. Springer Verlag, New York.

Kohler, H. P. and Philipov, D. (2001). Variance effects in the Bongaarts-Feeney formula. *Demography*, 38(1):1–16.

Rodriguez, G. Demographic translation and tempo effects: An accelerated failure time perspective. *In this volume, also published in Demographic Research*, 14(6):85–110. 2006.

Ryder, N. (1964). The process of demographic translation. *Demography*, 1(1):74–82.

Vaupel, J. W. Lifesaving, lifetimes and lifetables. *In this volume, also published in Demographic Research*, 13(24):597–614. 2006.

Vaupel, J. W. (2002). Life expectancy at current rates vs. current conditions. *Demographic Research*, 7(8):365–377.

Vaupel, J. W., Manton, K. M., and Stallard, E. (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography*, 16:439–454.

Wilmoth, J. R. (2005). On the relationship between period and cohort mortality. *Demographic Research*, 13(11):231–280.

Tempo effects in mortality: An appraisal*

Michel Guillot

Center for Demography and Ecology, University of Wisconsin-Madison,
1180 Observatory Drive, Madison, WI 53706, USA.
E-mail: mguillot@ssc.wisc.edu

Summary. This study examines the existence of tempo effects in mortality and evaluates the procedure developed by Bongaarts and Feeney for calculating a tempo-adjusted life expectancy. It is shown that the performance of Bongaarts and Feeney's index as an indicator reflecting current mortality conditions depends primarily on specific assumptions regarding the effects of changing period mortality conditions on the timing of future cohort deaths. It is argued that, currently, there is no clear evidence about the existence of such effects in actual populations. This chapter concludes that, until the existence of these effects can be demonstrated, it is preferable to continue using the conventional life expectancy as an indicator of current mortality conditions.

1 Introduction

There are three main uses of period indicators – such as the total fertility rate (TFR) or the life expectancy at birth (e_0) – in demography. First, period indicators are used as summaries of period age-specific rates, in order to allow easy comparisons of arrays of rates across populations and time periods. For example, a TFR that is lower in Population A than in Population B implies that at least one age-specific fertility rate is lower in Population A. In order to give a metric to these summary measures that is easy to interpret in terms of the underlying demographic processes, demographers use the classic synthetic-cohort scenario, which simulates a cohort of individuals exposed throughout their entire life to the age-specific rates of one particular period. This transforms a set of period age-specific mortality rates, for example, into years of life, interpreted as the life expectancy at birth "under current rates".

Second, period summary measures are used as indicators of current "conditions", which can be defined as all underlying factors affecting demographic behavior. For example, an increase in life expectancy is often interpreted as a

sign that progress is being made with respect to public health, medical technology, personal health behaviors, living standards, or other factors affecting survival. One way to conceptualize how current conditions may produce a certain level of a demographic indicator is to hypothesize about a scenario in which current conditions stay constant in the future. Under this scenario, one would expect period demographic indicators to eventually stabilize at a level that would be the product of these constant conditions. In the remainder of this chapter, I will refer to levels of demographic indicators that would eventually be observed in the population if current conditions remained constant in the future as the "stationary-equivalent" levels, or levels "under current conditions".

Third, period summary measures are used as proxies for tracking the changing behavior of real cohorts in the absence of complete cohort information. For example, an increase in the period life expectancy at birth is often interpreted as an indication that "we are living longer", i.e., that life expectancy is also increasing for real cohorts of individuals.

While the first use of period summary measures does not present any particular problem, the second and third uses are potentially undermined by the presence of "tempo effects". In fertility, tempo effects traditionally refer to the impact on the period *TFR* of changes in the timing of births within cohorts (Ryder, 1980). For example, in a population where cohort fertility levels are constant, indicated by a constant cohort *TFR*, but where the timing of births is changing, the period *TFR* may not equate the value of the constant cohort *TFR* and thus poorly reflects the behavior of real cohorts. Because of tempo effects, it is inappropriate to use the period *TFR* of 3.7 in 1955 in the US as an indicator of the level of fertility for some actual cohort, since no cohort contributing births during that year experienced such high fertility levels (the highest cohort *TFR* among cohorts active in 1955 is 3.2, for the cohort born in 1930). Also, the below-replacement period *TFR*s currently observed in a number of countries may poorly reflect current fertility conditions, because cohorts may be currently delaying their births while retaining fertility goals at or above replacement. If the conditions affecting individuals' completed fertility remain constant in these countries, the cohort *TFR* may eventually stabilize at a level that is higher than the one indicated by current period *TFR*s. Tempo effects thus pose a challenge for the interpretation of levels and trends in period *TFR*s.

Tempo effects have been extensively studied for fertility and marriage (Ryder, 1956, 1964, 1980; Keilman, 1994; Bongaarts, 1998, 1999; Kohler, 2002; Goldstein, 2003; Winkler-Dworak and Engelhardt, 2004). Various approaches have been proposed to adjust period measures for tempo effects. It is important to state that the solution for the adjustment may vary depending on the purpose of the correction, i.e., measuring period conditions or tracking real cohort behavior. In fertility, the first purpose involves estimating the level at which the *TFR* would eventually stabilize if factors affecting individuals' completed fertility remained constant at the levels of a particular period. The

second purpose involves estimating the *TFR* that would have been observed during that particular period if cohorts had not modified the timing of their births, while retaining their potentially changing completed fertility. These two scenarios differ and may thus yield different solutions. Differences in objectives explain in part why different procedures for tempo adjustments in fertility have yielded different results.

More recently, the concept of tempo effects has been applied to mortality (Bongaarts and Feeney, 2002, in this volume p. 11 and p. 29). The authors argue that the conventionally-calculated period life expectancy at birth is affected by tempo effects whenever mortality is changing. They propose an alternative period measure of longevity, which they claim adjusts for tempo effects. Although not explicitly stated, the purpose of the adjustment is to obtain a measure that better reflects current conditions, i.e., the level at which the life expectancy at birth would eventually stabilize if mortality conditions, defined as all factors affecting survival, remained constant at current levels.

In this chapter, I first examine the existence of tempo effects in mortality, by looking at historical discrepancies between period and cohort mortality measures. I then discuss the strategy proposed by Bongaarts and Feeney. I argue that the performance of Bongaarts and Feeney's tempo-adjusted life expectancy as an indicator reflecting current mortality conditions depends primarily on specific assumptions regarding the effects of changing period mortality conditions on the timing of future cohort deaths, and that currently there is no clear evidence about the existence of such effects in actual populations. I conclude that until the existence of such effects can be demonstrated, it is preferable to continue using the conventional life expectancy as an indicator of current mortality conditions.

2 The existence of tempo effects in mortality

There are interesting parallels between mortality and fertility with regards the study of tempo effects. The mortality index for which the parallel best applies is the total mortality rate (*TMR*) (Bongaarts and Feeney, in this volume p. 11). In a cohort (real or synthetic), the *TMR* is the number of lifetime deaths divided by the initial size of the cohort. In a life table with a radix of one, the *TMR* can be calculated by adding all age-specific life table deaths. Obviously, the *TMR* in a cohort, real or synthetic, is invariably one. The following equation pertains to a real cohort born at time t:

$$TMR_c(t) = \int_0^\infty d_c(x,t)\,dx \tag{1}$$

where $d_c(x,t)$ is the number (or proportion) of deaths at age x for a cohort born at time t (radix= 1).

The *TMR* can also be calculated in a cross-sectional fashion by calculating for each cohort the proportion of deaths occurring during a particular period,

and by summing these proportions across all cohorts:

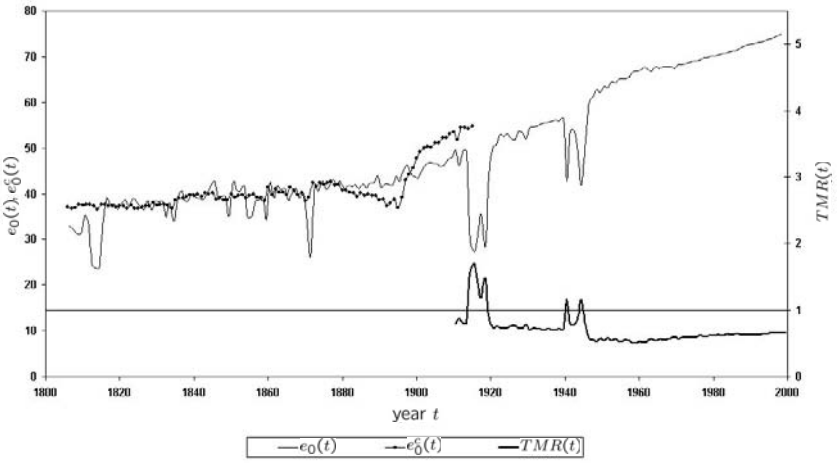
$$TMR(t) = \int_0^\infty d_c(x, t-x)\, dx \tag{2}$$

The period *TMR* can be interpreted as the proportion of cohort deaths that are occurring during period t. If all cohorts have the same age distribution of deaths, the period *TMR* is constant at 1.00. If the age distribution of deaths changes from cohort to cohort, however, the period *TMR* deviates from one. For example, if cohort deaths are being progressively spread out over a longer period of time, with smaller proportions occurring during a given period, the period *TMR* is less than one. This means that less than 100% of cohort deaths are occurring during period t, which is a sign that cohort deaths are being delayed, i.e., that mortality is declining. Conversely, the period *TMR* is greater than 1.00 during periods of increased mortality, when increased proportions of cohort deaths are occurring at the same time.

Figures 1 and 2 show long-term trends in the period *TMR* among French males and Swedish females, together with trends in period and cohort life expectancy. (The data come from the Vallin-Meslé database for France, and from Human Mortality Database for Sweden.) The period *TMR* is generally below 1.00, indicating mortality decline. However, *TMR*s above 1.00 were experienced by French males during WWI and WWII, and by Swedish females in 1918 during the influenza epidemic.

In Figures 1 and 2, changes in the period *TMR* can be attributed to changes in the timing of deaths from cohort to cohort. Because of these changes, the period *TMR* is a poor indicator of the "stationary-equivalent" *TMR*, i.e., the period *TMR* that would eventually be observed if current mortality conditions remained constant in the future. Indeed, under this constant-conditions scenario, one would expect the age distribution of deaths to be eventually identical for all cohorts, and the period *TMR* to reach a value of 1.00 eventually. The period *TMR* is also a poor indicator of the trend in the cohort *TMR*, which is constant at 1.00 for all cohorts. Making a parallel with fertility, it can be stated that the period *TMR* is affected by tempo changes, defined as changes in the timing of deaths within cohorts. Unlike the cohort *TFR*, however, there are no quantum variations in the cohort *TMR*, since it is constant at 1.00. This implies that deviations from 1.00 in the period *TMR* can be *entirely* attributed to tempo effects, and that a "tempo-adjusted" period *TMR* necessarily equals 1.00.

In mortality, the most important period indicator is not the *TMR*, but the period life expectancy at birth, e_0. In order to assess the presence of tempo effects in e_0, one may first examine the existence of situations in which e_0 has no relevance for actual cohorts. Figure 1 shows trends in period life expectancy in France, along with trends in cohort life expectancy (e_0^c), plotted at the time of birth. This figure illustrates the fact that in France, there are a few years – the WWI years – during which period e_0 levels have no relevance for any par-



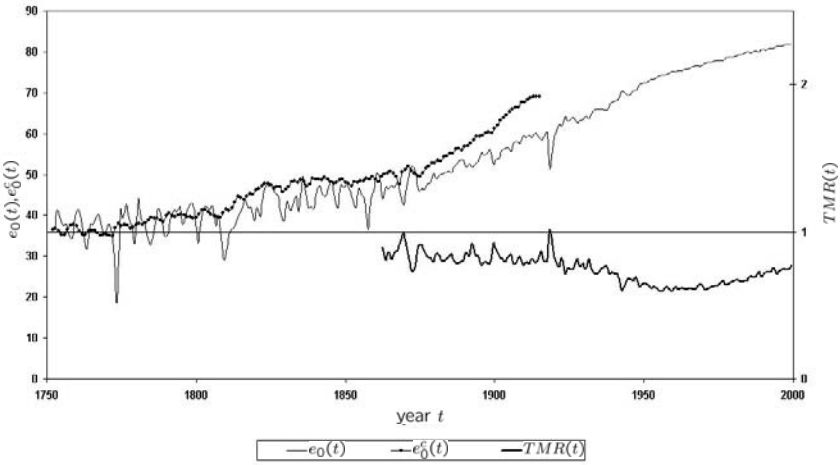
Note: Data source: Vallin-Meslé database.
http://www.ined.fr/publications/cdrom_vallin_mesle/contenu.htm

Note: $e_0^c(t)$ is plotted at time when the cohort was born.

Fig. 1. Period life expectancy at birth, $e_0(t)$; cohort life expectancy at birth, $e_0^c(t)$; and period total mortality rate, $TMR(t)$. France, males, 1806-1998.

ticular cohort. During these years, many cohorts had elevated mortality risks at the same time, resulting in period life expectancies as low as 27.2 years in 1915. But these elevated risks were relatively short-term, and no actual cohort contributing deaths during these years have experienced such low life expectancy levels (the lowest cohort life expectancy among contributing cohorts is 37.0 years for the cohort born in 1895). In a sense, the sudden decline in life expectancy in 1915 gives an exaggerated indication of mortality change occurring within cohorts. Changes in cohort mortality levels would have been poorly predicted on the basis of these large drops in e_0. This discussion of trends in period life expectancy has parallels with discussions of trends in the period TFR and the difficulty to use this measure as an indicator of real changes in cohort completed fertility.

It is less easy to tell if the period life expectancy at birth is a biased indicator of the "stationary-equivalent" life expectancy, or life expectancy under "current conditions". If today's mortality conditions remained constant, would the life expectancy at birth stabilize at the current period level or at some other level? Historical trends in cohort life expectancy are of little use for answering that question, because cohorts are exposed to constantly-changing period conditions.



Data source: Human Mortality Database. www.mortality.org.

Note: $e_0^c(t)$ is plotted at the time (t) when the cohort was born.

Fig. 2. Period life expectancy at birth, $e_0(t)$; cohort life expectancy at birth, $e_0^c(t)$; and period total mortality rate, $TMR(t)$. Sweden, females, 1752-1998.

3 Bongaarts and Feeney's tempo-adjusted life expectancy

The goal of Bongaarts and Feeney's alternative measure of survival is precisely to resolve potential discrepancies between period levels and stationary-equivalent levels of life expectancy. As said earlier, the goal of their tempo-adjusted measures is not to better track real changes in cohort life expectancy, so I will not discuss here how their approach performs this task. There are a number of papers in this volume and elsewhere which deal with this somewhat different issue (Guillot, 2003b; Schoen and Canudas-Romo, 2005; Goldstein, in this volume).

Bongaarts and Feeney (referred to as BF in the remainder of the chapter) compare three mortality indexes:

$$CAL(t) = \int_0^\infty p_c(x, t-x)\, dx \tag{3}$$

where $p_c(x, t-x)$ is the probability of surviving from birth to age x for the cohort born at time $t-x$.

$$MAD(t) = \frac{\int_0^\infty x \cdot d_c(x, t-x)\, dx}{\int_0^\infty d_c(x, t-x)\, dx} \tag{4}$$

$$M_4(t) = \int_0^{\infty} \exp\left\{ - \int_0^{x} \frac{\mu(a,t)}{TMR(t)}\, da \right\} dx \tag{5}$$

where $\mu(a,t)$ is the force of mortality at age a at time t.

The first index, $CAL(t)$ (= cross-sectional average length of life), sums actual proportions of cohort survivors at time t, rather than proportions of survivors in the synthetic cohort at time t as in the case of $e_0(t)$. Thus CAL takes into account all mortality rates previously experienced by cohorts whose survivors are present in the population at time t. This index, which is described in detail elsewhere (Brouard, 1986; Guillot, 1999, 2003a, 2005), has been used primarily for examining the impact of mortality change on population growth.

The second index, $MAD(t)$, is the mean age at death that would be observed at time t if the studied population, while subject to actual mortality trends, had experienced constant births per unit of time (constant-birth population) and had been closed to migration. MAD can be interpreted as the population mean age at death at time t, controlling for changes in the initial size of cohorts.

The third index, $M_4(t)$, is a period life expectancy at birth where all age-specific death rates are adjusted by a factor $1/TMR(t)$. If the TMR is equal to .8, each death rate will be adjusted upwards by a factor 1.25, and $M_4(t)$ will be lower than the actual $e_0(t)$.

An important feature of these summary indexes of mortality is that when mortality is constant over time, then $CAL(t) = MAD(t) = M_4(t) = e_0(t)$. If mortality varies, however, these indexes diverge. In particular, if age-specific mortality rates have been steadily declining, e_0 will be systematically higher than CAL, MAD or M_4.

Bongaarts and Feeney calculate these three indexes in populations where mortality has changed overtime. They demonstrate that $CAL = MAD = M_4$ under a specific pattern of mortality change, which they claim is a good approximation of the current situation in low-mortality populations. This quantity is then interpreted as a tempo-adjusted life expectancy at birth. These two propositions are examined successively in the following sections.

4 Evaluating Bongaarts and Feeney's "proportionality" assumption

The first assumption proposed by Bongaarts and Feeney involves a quantity described by Preston and Coale (1982) and Arthur and Vaupel (1984). This quantity may be called an age intensity, ν^*:

$$\nu^*(x,t) = \frac{-\partial p_c(x, t-x)/\partial x}{p_c(x, t-x)}. \tag{6}$$

In Equation (6), ν^* is the rate at which the proportion of cohort survivors in a population at time t varies from one age to the next. It also corresponds to

the age intensity of the constant-birth population. It is in fact a special case of Arthur and Vaupel's age intensity, ν, which applies to the more general case of populations with varying births and open to migration.

In their chapter, Bongaarts and Feeney (in this volume p. 11) demonstrate that $CAL(t) = MAD(t) = M_4(t)$ if at time t the age intensity $\nu^*(x,t)$ is proportional to $\mu(x,t)$, i.e., if the following equation holds:

$$\mu(x,t) = p(t)\nu^*(x,t) \tag{7}$$

They refer to this assumption as the "proportionality" assumption, and claim that this assumption is a good approximation of the current situation in Sweden, France and the US. As Wachter demonstrates in this volume, one situation which approximately produces proportionality is when all cohorts experience a Gompertz force of mortality and a constant, age-invariant rate of decline in age-specific death rates (Wachter, in this volume). More generally, the proportionality assumption is immediately met in a given year if, during that year, the proportions of cohort survivors shift along the age axis by an amount that is identical for all cohorts, i.e., if $p_c(x, t_2 - x) = p_c(x - F(t), t_1 - x + F(t))$, where $F(t)$ is the amount of the shift, in years, between t_1 and t_2 (Bongaarts and Feeney, 2002). For example, the proportionality assumption would be met if the proportion of cohort survivors at age 80 in 2000 was equal to the proportion of cohort survivors at age 78 in 1995 (i.e., a 2-year shift in 5 years), and if this correspondence could be established for all cohorts.

While it is true that if Equation (7) holds at time t, then $MAD(t) = CAL(t) = M_4(t)$, there are deviations from the proportionality assumptions in real populations which produce important discrepancies between the three indicators. This can be shown by calculating the three indicators in real populations, without making any assumption about the pattern of mortality change.

Figures 3 and 4 show that among French males and Swedish females, there are important differences between the three indicators. Typically, *CAL* has the lowest value, *MAD* has the highest value, and M_4 is somewhere in between. The difference between *CAL* and *MAD* is as large as 9.46 years in 1953 in France. Although the gap between the two measures has decreased over time, it is still 2.76 years for French males in 1998, and 2.08 years for Swedish females in 1997.

More importantly, Figure 3 and 4 also show that *CAL*, *MAD* and M_4 react very differently to period variations in mortality. In particular, *MAD* and M_4 are much more sensitive to variations in period mortality, with a trajectory somewhat parallel to that of the period life expectancy at birth, although at a lower level. In contrast, *CAL* is much less reactive to period variations in mortality. Since in real populations *CAL*, *MAD* and M_4 offer a different picture of changes in mortality over time, these three indexes should not be interpreted interchangeably. In particular, *CAL* should not be interpreted as a population mean age at death purged of changes in cohort size (*MAD*).

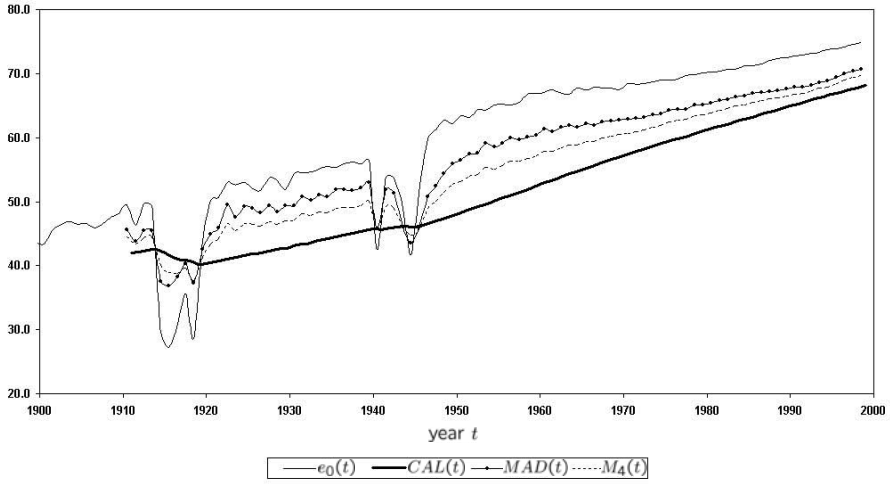


Fig. 3. Period life expectancy at birth, $e_0(t)$; cross-sectional average length of life, $CAL(t)$; mean age at death in the constant-birth population, $MAD(t)$; and Bongaarts and Feeney's $M_4(t)$. France, males, 1900-1998.

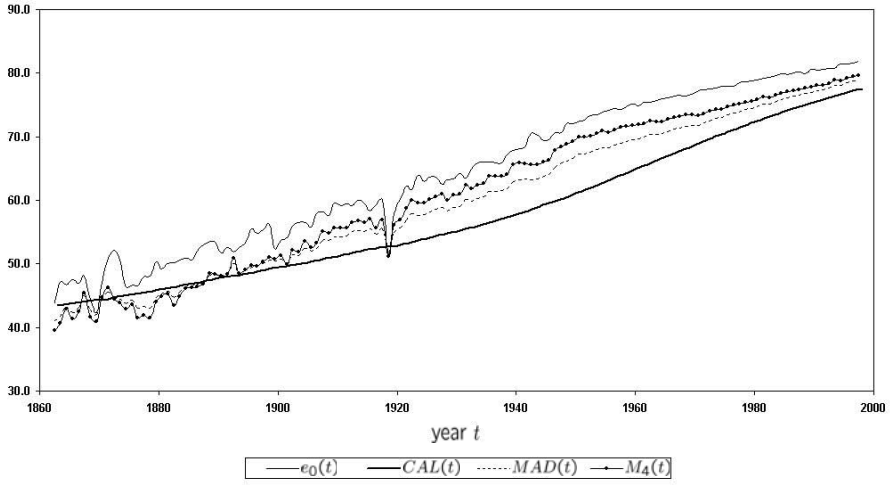
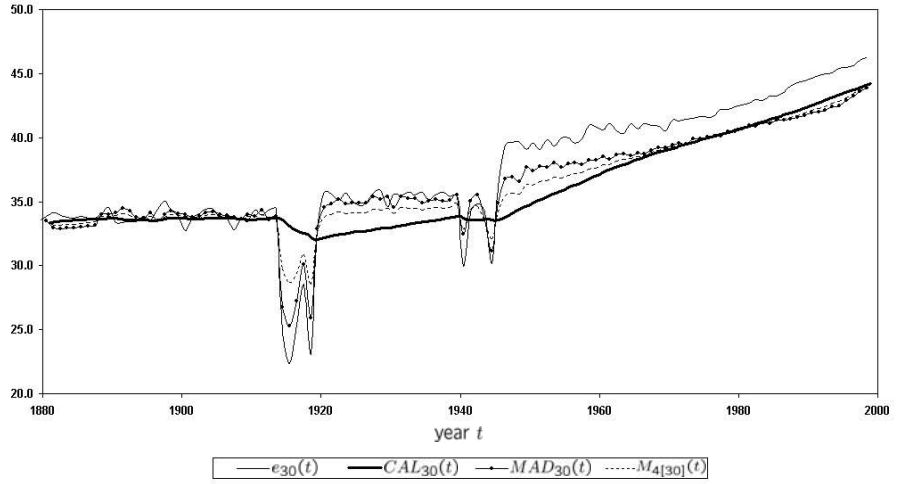


Fig. 4. Period life expectancy at birth, $e_0(t)$; cross-sectional average length of life, $CAL(t)$; mean age at death in the constant-birth population, $MAD(t)$; and Bongaarts and Feeney's $M_4(t)$. Sweden, females, 1862-1998.

Even if today, the difference between the two indexes is not as large as earlier (though still significant), they remain distinct conceptually.

The reason why BF do not find large differences between CAL, MAD and M_4 is that in their empirical examples, they make the additional assumption that there is no mortality below age 30 throughout the entire life time of all cohorts who have survivors at time t (i.e., since the early 20th century for current estimates of CAL, MAD or M_4). Indeed, if we discard mortality information below age 30 and estimate the mean number of years to be lived above age 30 only, the proportionality assumption is met in France and Sweden since the 1970s, and we obtain three indicators, CAL_{30}, MAD_{30} and $M_{4[30]}$ that are nearly equal for the recent period, as shown in Figures 5 and 6. (Note, however, that they still differed by about .75 years in the early 1990s in France.)



Note: Like $e_{30}(t)$, $CAL_{30}(t)$, $MAD_{30}(t)$, and $M_{4[30]}(t)$ represent a number of additional years expected to be lived above age 30, given survival to age 30.

Fig. 5. Period life expectancy at age 30, $e_{30}(t)$; cross-sectional average length of life, $CAL_{30}(t)$; mean age at death in the constant-birth population, $MAD_{30}(t)$; and Bongaarts and Feeney's $M_{4[30]}(t)$. France, males, 1880-1998.

In reality, mortality below age 30 is not negligible, especially when considering earlier decades of the twentieth century. Even in 1998 among French males, mortality below age 30 still produced a loss of 1.37 years of period life expectancy at birth. As a result, when all ages are taken into account, the proportionality assumption is not met, and this creates important discrepancies between CAL, MAD and M_4 which are not well addressed in BF's procedure.

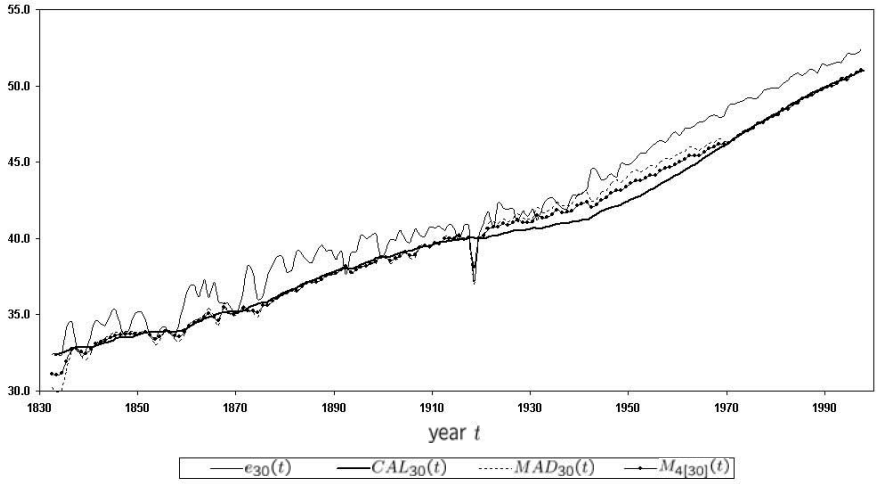


Fig. 6. Period life expectancy at age 30, $e_{30}(t)$; cross-sectional average length of life, $CAL_{30}(t)$; mean age at death in the constant-birth population, $MAD_{30}(t)$; and Bongaarts and Feeney's $M_{4[30]}(t)$. Sweden, females, 1832-1998.

So far, BF's procedure refers to mortality *above age 30 only* and does not permit the calculation of a life expectancy at birth that is consistent with their overall proposition. (In this volume, BF deal with mortality below age 30 differently. Instead of assuming that there is no mortality below age 30, as in their earlier work, they assume that there are no tempo effects below age 30. This allows them to calculate an adjusted life expectancy at birth which combines unadjusted rates below age 30 with adjusted rates above age 30. This assumption of no tempo effects below age 30, however, seems somewhat arbitrary.)

5 Bongaarts and Feeney's definition of changes in period mortality conditions

While departures from the proportionality assumption raises practical issues with the estimation of BF's adjusted life expectancy, there are more fundamental considerations to examine in order to evaluate the interpretation of CAL, MAD or M_4 as tempo-adjusted indicators. These considerations apply even if the proportionality assumption is met. Since $CAL = MAD = M_4$ under the proportionality assumption, this section focuses on the behavior of CAL only. I choose CAL, because unlike MAD or M_4, it has relevant properties (for example, Equation (8) later in this chapter) that do not require any assumption about the pattern of mortality change.

BF's approach relies on a particular definition of changes in period mortality conditions, which is different from the classic definition. Traditionally, demographers assume that particular period mortality conditions generate a set of age-specific mortality rates which completely reflect these conditions, as long as the population is homogeneous with respect to the risk of death. Therefore, it is assumed that changes in period age-specific mortality rates completely reflect changes in period mortality conditions. Similarly, it is assumed that when period mortality conditions stop changing, period age-specific mortality rates – or e_0 – become constant. Under this assumption, the period life expectancy at birth, as traditionally calculated, is an unbiased indicator of period mortality conditions, and no adjustment is needed.

As in the classic approach, BF assume that populations are homogeneous with respect to the risk of death, but they address mortality change differently. They define period mortality changes in terms of changes overtime in the $p_c(x, t-x)$ curve. According to them, a change in mortality conditions during a certain period is indicated by a change in $p_c(x, t-x)$, producing a change in the value of CAL. Conversely, they assume that mortality conditions stop changing whenever the curve $p_c(x, t-x)$ – or when $CAL(t)$ – becomes constant (Bongaarts and Feeney, 2002, p.17).

BF's definition of mortality change implies that, as a result of new mortality conditions appearing during a given period, all future cohort deaths are delayed by a certain amount of time. These delays in future cohort deaths accumulate over time as mortality conditions keep improving. When mortality conditions stop improving, no additional delay occurs, which implies that the delays in future cohort deaths, already accumulated by previous mortality change, remain unchanged.

This conception of mortality change is illustrated with Lexis diagrams in Figures 7a and 7b. The quantities in the Lexis areas refer to deaths in cohort life tables with a constant radix at age zero. (For simplicity, this illustration uses a starting age of zero, but a similar argument could be developed for any starting age.) In this illustration, mortality conditions are constant up to year $T-1$. As a result, up to year $T-1$, the age distribution of life table cohort deaths, D_x, is constant over time and the period TMR is equal to 1.00. This stationary situation changes as a new set of mortality conditions appear in year T (Diagram A). According to BF's definition of mortality change, these new mortality conditions generate postponements (or delays) in cohort deaths, and thus a certain proportion of deaths "migrate" to the following year. These delays are illustrated with arrows indicating the proportion of cohort deaths $\lambda(T)$ that are postponed to the following year as a result of the new mortality conditions appearing in year T. These proportions apply to the stationary deaths D_x that would have been observed during year T and subsequently if no change in mortality conditions had occurred during year T. $\lambda(T)$ also corresponds to the amount of delay (as fraction of a year) experienced by cohort deaths (Vaupel, in this volume p. 93, refers to these delays as δ). It also corresponds to the amount (in years) by which the curve

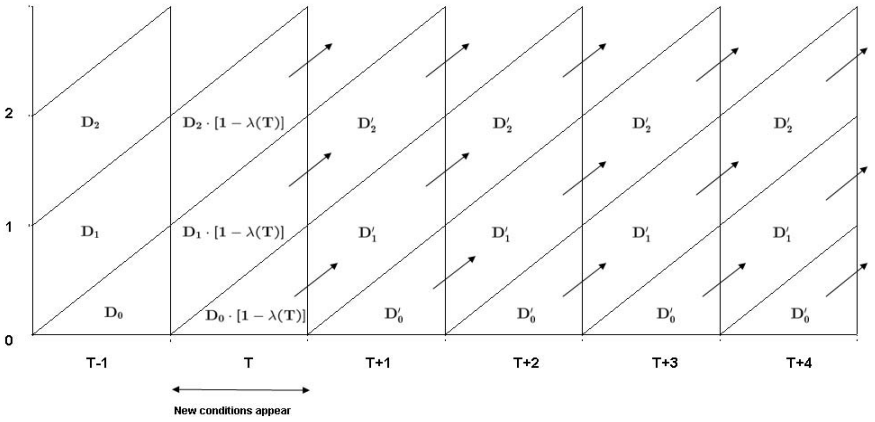
$p_c(x, t-x)$ shifts along the age axis. Note that the new conditions of year T do not only generate delays during year T, but during all future years. Delays resulting from new period mortality conditions can be experienced many years after the new conditions appeared. In the notation $\lambda(T)$, T refers to the time at which new conditions appear, generating delays in future cohort deaths. It does not refer to the time when these delays are actually experienced, because these delays can indeed be experienced many years later.

According to this scenario of mortality change, the *TMR* during year T is equal to $(1 - \lambda(T))$. However, if cohorts experience no additional delays in the timing of their future deaths, i.e., if mortality conditions stop changing according to BF's definition of mortality change, constant numbers of cohort deaths, D'_x, reemerge as early as the year $T + 1$. This implies that, starting in year $T + 1$, a *TMR* of 1.00 is reestablished, *CAL* becomes constant, and $e_0(t) = e_0^c(t) = CAL(t)$. The life expectancy at birth during year T will be higher than the new constant level starting at $T+1$, because unlike year $T+1$, less than 100% of cohort deaths (i.e., $1 - \lambda(T)$) are occurring during year T. The discrepancy is due to the fact that starting with year $T+1$, the number of additional deaths resulting from the previous year's delays equals the number of deaths postponed to the following year, while during year T, there are only "missed" deaths, postponed to the following year.

Mortality conditions, however, may not remain constant but be replaced by new mortality conditions appearing during year $T+1$ (Diagram B). These new conditions, according to BF, generate additional delays in cohort deaths, illustrated by a second set of arrows indicating the proportions of cohort deaths $\lambda(T + 1)$ that are postponed to the following year as a result of the new mortality conditions of year $T+1$. These proportions apply to the deaths D'_x that would have been observed during year $T + 1$ and subsequently if no further mortality change had occurred after time T (a counter-factual scenario that corresponds to the situation described in Diagram A).

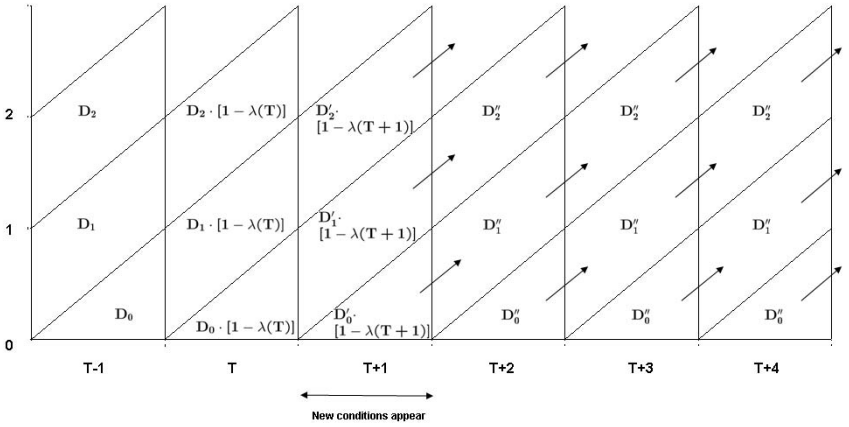
In Diagram B, the *TMR* during year $T + 1$ is equal to $(1 - \lambda(T + 1))$. Here also, if no new mortality conditions appear after year $T + 1$, starting at year $T + 2$, a *TMR* of 1.00 is reestablished, *CAL* becomes constant and $e_0(t) = e_0^c(t) = CAL(t)$. The life expectancy at birth during year $T + 1$ will be higher than the new constant level starting at $T + 2$, because fewer cohort deaths are occurring during year $T + 1$. This mechanism of mortality change could continue during following years, with new mortality conditions appearing every year and creating delays in cohort deaths which would come in addition to the delays already accumulated as a result of previous mortality change.

This example illustrates the implications of BF's conception of mortality change. The first implication is that changes in mortality conditions are entirely indicated by deviations from 1.00 in the *TMR*. When new period mortality conditions appear, the *TMR* deviates from 1.00, and the quantity $(1 - TMR)$ indicates the proportion of cohort deaths that are postponed to the following year as a result of these new conditions, or equivalently, the



Note: The quantities in the Lexis areas refer to deaths in cohort life tables with a constant radix at age zero. The arrows indicate the proportions of cohort deaths $\lambda(T)$ that "migrate" to the following year as a results of the new conditions appearing in year T. These proportions apply to the stationary deaths D_x of year $T-1$. $D'_x = D_x \cdot [1-\lambda(T)] + D_{x-1} \cdot \lambda(T)$

Fig. 7a. Lexis diagram illustrating Bongaarts and Fenney's scenario of mortality change. **Diagram A**: New conditions appear at time T.



Note: The quantities in the Lexis areas refer to deaths in cohort life tables with a constant radix at age zero. The arrows indicate the proportions of cohort deaths $\lambda(T+1)$ that "migrate" to the following year as a results of the new conditions appearing in year $T+1$. These proportions apply to the stationary deaths D'_x that would have been observed during the year $T+1$ and subsequently if no further mortality change had occurred after time T (as shown in Diagram A). $D''_x = D'_x \cdot [1-\lambda(T+1)] + D'_{x-1} \cdot \lambda(T+1)$

Fig. 7b. Diagram B: New conditions appear at time $T+1$.

amount of the delay. As period mortality conditions stop changing, a *TMR* of 1.00 is immediately reestablished. Similarly, changes in mortality conditions are entirely indicated by changes in *CAL*, because there is a direct connection between changes in *CAL* and levels of the period *TMR* (Guillot, 2003a, p.53):

$$TMR(t) = 1 - \frac{dCAL(t)}{dt} \tag{8}$$

(Note that unlike BF's similar equation (Bongaarts and Feeney, in this volume p. 11, Equation [8a]), Equation (8) does not require any assumption.)

The second implication of BF's conception of mortality change pertains to the interpretation of *CAL* as a stationary-equivalent life expectancy. BF's assumption about the effect of new mortality conditions on the timing of future cohort deaths produces a situation in which *CAL* better reflects current mortality conditions, because *CAL* corresponds to the life expectancy at birth that would eventually be observed in the population if mortality conditions stopped changing (i.e., if cohorts experienced no additional delays in the timing of their future deaths). In Diagram B of Figure 7, the period life expectancy at birth observed during year $T + 1$ does not reflect well the new mortality conditions emerging during that year, because it is different from the constant level of life expectancy at birth that would be observed starting in year $T + 2$ if mortality conditions remained constant. In reality, new mortality conditions may appear in year $T + 2$ and subsequently. Nonetheless, no matter what happens during year $T+2$, the level of *CAL* observed on January 1 of year $T + 2$ indicates this stationary-equivalent level of mortality.

BF's tempo-adjusted life expectancy is thus a stationary-equivalent period life expectancy that is consistent with *their* definition of mortality change, based on the behavior of $p_c(x, t-x)$. In general terms, if $p_c(x, t-x)$ becomes constant at time t, then $p(x, t) = p_c(x, t-x)$. Therefore, if $p_c(x, t-x)$ becomes constant, e_0 immediately adjusts to the corresponding *CAL* level and remains constant thereafter.

One can note here that this scenario of constant mortality conditions is possible only if the function $p_c(x, t - x)$ is monotonically decreasing. This assumption is less restrictive than BF's proportionality assumption, and allows for the proportion of postponed deaths, $\lambda(T)$, to vary with age. (Age-varying delays are also examined by Feeney in this volume). One assumption that must remain, however, in order to use *CAL* as a stationary-equivalent life expectancy, is that these age-specific delays in future cohort deaths generated by the new conditions of year T – which we can denote $\lambda(x, T)$ – must be identical for all cohorts. For example, new mortality conditions of year T must generate delays in deaths of age 80 for the cohort age 40 at time T that are equal to the delays in deaths of age 80 for the cohort aged 70 at time T. In other words, age-specific delays need to remain constant with time in the constant-condition scenario. In Figure 7a, the proportions of deaths transferred to the following year as a result of new mortality conditions of

year T, illustrated with the arrows, *may vary vertically, but must be constant horizontally.* This insures that e_0 adjusts to CAL in BF's scenario of constant mortality conditions.

6 Assessing indicators of period mortality conditions: e_0 vs. CAL

The assessment of BF's tempo-adjusted life expectancy (apart from discussing the adequacy of the proportionality assumption) comes down to determining whether new mortality conditions generate a new set of period age-specific death rates, as traditionally believed, or whether these new conditions generate delays in the timing of future cohort deaths, as illustrated in Figure 7. In particular, it comes down to determining whether cohorts would stop experiencing additional delays in the timing of their future deaths if mortality conditions stopped changing. In general terms, it comes down to determining whether levels and trends in period mortality conditions are better reflected by changes in life expectancy or changes in CAL.

In order to contrast these two views, one first needs to recognize that life expectancy and CAL are not independent of one another. In particular, it can be shown that variations in CAL depend in part on differences between proportions of survivors in the synthetic cohort at time t and proportions of survivors in real cohorts at time t (Guillot, 2003a, p.53):

$$\frac{dCAL(t)}{dt} = \int_0^{\omega} \mu(x,t)[p(x,t) - p_c(x,t-x)]\,dx \tag{9}$$

where ω is the age at which $p(x,t) = p_c(x,t-x) = 0$.

Under steady mortality decline, $p(x,t)$ tends to be greater than $p_c(x,t-x)$, and CAL tends to increase. In fact, if $p(x,t) \neq p_c(x,t-x)$ for any x in the interval $(0,\omega)$ (which happens for most years in France and Sweden), the direction of the change in CAL will be determined by the sign of the difference between e_0 and CAL:

$$\frac{dCAL(t)}{dt} = \bar{\mu}(t)[e_0(t) - CAL(t)] \tag{10}$$

where $\bar{\mu}(t)$ is a value, always positive, of the force of mortality $\mu(x,t)$ at an age in the interval $(0,\omega)$.

Figure 8 and 9 show trends in life expectancy and CAL among French males and Swedish females. In order to examine these trends in the context of BF's discussion of tempo effects, these figures use mortality information above age 30 only, but similar correspondences between CAL and life expectancy would be observed if all ages were taken into account. As expected, the direction of the change in CAL is related to whether life expectancy is

above or below the corresponding value of CAL. These figures also illustrate the relationship between CAL change and the TMR levels (Equation (8)).

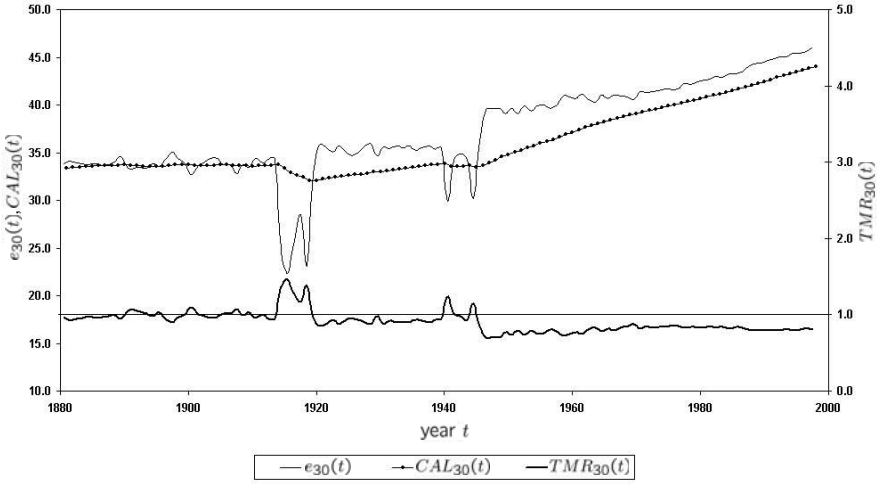


Fig. 8. Period life expectancy, $e_{30}(t)$; cross-sectional average length of life, $CAL_{30}(t)$; and period total mortality rate, $TMR_{30}(t)$. France, males, 1880-1998.

Equations (9) and (10), illustrated in Figures 8 and 9, allow us to contrast two different views of mortality change above age 30. The classic view implies that changes in mortality conditions at these ages is indicated by changes in e_{30}, and that CAL_{30} simply "reacts" to these variations, depending on whether e_{30} is above or below CAL_{30} during a given year. According to this view, if current conditions stopped changing, e_{30} would remain constant while CAL_{30} would gradually increase towards e_{30}, as expected from Equation (9). This view implies that CAL_{30} is a biased indicator of stationary-equivalent life expectancy, because if mortality conditions stopped changing, e_{30} would remain constant while CAL_{30} would continue changing.

On the contrary, BF consider that changes in mortality conditions are indicated by changes in CAL_{30}, and perceive variations in e_{30} as less meaningful, created by whatever trajectory CAL_{30} is taking. According to this view, e_{30} is a biased indicator of stationary-equivalent life expectancy, because CAL_{30} would remain constant while e_{30} would change if mortality conditions stopped changing.

Another way to contrast these two views is to examine the equation for the TMR. Equation (11) is a modified version of Equation (2) in which cohort deaths at time t are expressed in terms of cohort survivors exposed to the force of mortality at time t and in which only ages 30 and above are taken

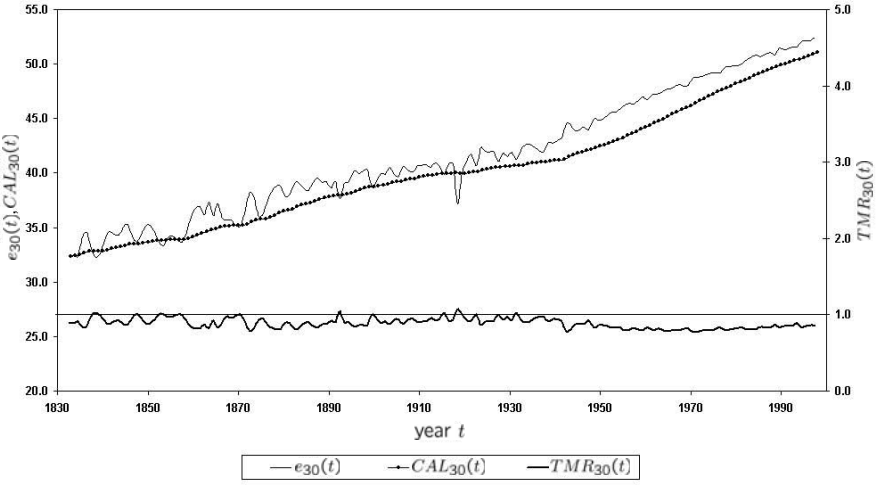


Fig. 9. Period life expectancy, $e_{30}(t)$; cross-sectional average length of life, $CAL_{30}(t)$; and period total mortality rate, $TMR_{30}(t)$. Sweden, females, 1832-1998.

into account (i.e., $p_c(30, t-30) = 1$):

$$TMR_{30}(t) = \int_{30}^{\infty} p_c(x, t-x) \cdot \mu(x,t)\, dx \tag{11}$$

As we saw earlier, the TMR will deviate from 1.00 whenever the timing of deaths is changing from cohort to cohort. No matter how we define mortality conditions, if period mortality conditions stopped changing, one would expect TMR_{30} to eventually reach the stationary value of 1.00. The stationary-equivalent period TMR_{30}, or $TMR_{30}(\infty)$, can thus be expressed for a given year as $TMR_{30}(t)$ divided by itself. This produces the following equation:

$$TMR_{30}(\infty) = \frac{1}{TMR_{30}(t)} \int_{30}^{\infty} p_c(x, t-x) \cdot \mu(x,t)\, dx \tag{12}$$

The conventional approach would attribute deviations in $TMR_{30}(t)$ to the fact that the proportions of cohort survivors, representing individuals exposed to past mortality levels, tend to be smaller than proportions of survivors in the synthetic cohort for year t, while $\mu(x,\ t)$ adequately represents current mortality conditions. If current mortality conditions stopped changing, the stationary-equivalent $TMR_{30}(\infty)$ of 1.00 would be reached through a progressive increase in $p_c(x, t-x)$, while $\mu(x,\ t)$ would stay constant at current levels. In contrast, BF assume that, if mortality conditions stopped changing, the stationary-equivalent $TMR_{30}(\infty)$ of 1.00 would be reached through

a change in the force of mortality by a factor $1/TMR_{30}(t)$, while $p_c(x, t-x)$ would stay constant at current levels. They are able to entirely attribute the correction factor of $1/TMR(t)$ in Equation (12) to $\mu(x,\ t)$ because of their assumption of cohort-invariant delays of future cohort deaths in the constant-condition scenario. (This adjustment of $\mu(x,\ t)$ appears in Equation (5) for M_4.) In sum, both views agree that $TMR_{30}(t)$ is biased an indicator of the stationary-equivalent TMR_{30} by a factor $1/TMR_{30}(t)$, but this correction factor is allocated to different components of Equation (12), yielding different estimates of the stationary-equivalent level of life expectancy.

It is difficult to tell with certainty whether mortality change above age 30 is indicated by e_{30} or by CAL_{30}, or equivalently, whether life expectancy would stabilize at $e_{30}(t)$ or $CAL_{30}(t)$ if mortality conditions stopped changing after time t. Bongaarts and Feeney rely on the existence of proportionality above age 30 as a key element in support of their view of mortality change. Proportionality, however, does not *per se* demonstrate the existence of cohort-invariant delays of future cohort deaths in the constant-condition scenario. Proportionality means that up to now, as a result of mortality change, successive cohorts have been delaying there deaths according to a specific pattern, but it does not allow to predict what would happen to the timing of *future* cohort deaths if mortality conditions stopped changing. In particular, the proportionality assumption does not demonstrate that cohorts will stop experiencing additional delays in the constant-condition scenario. Also, the proportionality assumption does not disprove the classic view assuming that if conditions stopped changing, mortality rates would remain constant at current levels. A hypothetical test (although perhaps not impossible for animal populations) would involve fixing the current epidemiological conditions (defined as all factors - technological, behavioral and environmental - affecting survival) at current levels and observing the resulting dynamics of CAL and life expectancy.

There are, however, several reasons to believe that period mortality conditions above age 30 are better reflected by e_{30}, and that CAL_{30} would not remain constant if mortality conditions stopped changing:

(1) In Sweden (Figure 9), periods during which CAL_{30} remained constant (or equivalently, when TMR_{30} reached a value of 1.00) seem to coincide with mortality crises (1870, 1892, 1900 and 1918, for example) rather than with periods during which mortality conditions remained constant.

(2) In Figures 8 and 9, $e_{30}(t)$ appears to have a dynamics of its own, as one would expect from an indicator reflecting changes in the epidemiological environment of a population. CAL_{30}, in comparison, appears as a "response" indicator, reacting to changes in e_{30} rather than generating them. (CAL reacts to changes in life expectancy somewhat like the temperature of a glass of water reacts to changes in ambient temperature.) For example, excess mortality during WWI in France appears as a short-term

deviation from an underlying trend in e_{30}. After the war, e_{30} quickly recovers this underlying trend, plausibly indicating that prewar epidemiological conditions were quickly recovered after the war. CAL_{30}, however, does not recover prewar levels until 1938, implausibly suggesting that pre-WWI epidemiological conditions were not reestablished until 20 years after the end of the war. Similarly, the relatively small decreases in CAL_{30} during WWII in France and during the 1918 Influenza epidemic in Sweden seem to understate the worsening of epidemiological conditions during these years. The independent nature of life expectancy is not as obvious today because of the absence of mortality crises, but this doesn't mean that CAL is now driving mortality change. (The sudden increase in e_{30} after WWII among French males, however, is somewhat puzzling. The level of e_{30} in 1946 is 3.9 years higher than in 1938, suggesting a sudden, substantial, and somewhat implausible improvement in mortality conditions relative to the pre-war period.)

(3) As stated earlier, the most important assumption of BF's approach is that new mortality conditions generate delays in future cohort deaths that may vary with age but are identical for all cohorts (or equivalently, that are constant with time). BF's approach thus does not address the fact that cohorts may react differently to new epidemiological conditions, with some cohorts benefitting more than others. In particular, younger cohorts – exposed to the new conditions for a longer period of time – may experience greater delays at older ages, as a result of these new conditions, than cohorts already old at the time when the new conditions appeared. It seems likely that many medical innovations, such as new drugs or new knowledge regarding health behaviors, have benefits that accumulate with time. For example, we expect delays in ages at death resulting from the 1964 US surgeon general's statement establishing smoking as a risk factor to be greater for smokers who were young in 1964 than for smokers who were older. The amount of delay generated by a medical innovation may thus depend to a large degree on how long before the innovation appeared. In other words, delays may very well be cohort-specific, implying that delays – and CAL – could continue changing even in the absence of further changes in conditions. (In fact, a scenario of constant e_{30} allows the occurrence of such cohort-specific delays.) It is true that certain medical discoveries apply only to individuals who are at the terminal stage of a disease, in which case the resulting delays in deaths may not depend on how long before the new technology appeared. However, mortality conditions encompass a broad range of factors, including some that likely have cumulative effects on survival.

These various points support the notion that current period conditions – and changes thereof – may be better described by life expectancy than by CAL. The above argumentation is imperfect because based on historical rather than

contemporary data, or on expectations regarding the cumulative effect of medical innovations on the timing of cohort deaths. The nature of mortality dynamics may well have changed, along with the nature of medical innovations, as Bongaarts and Feeney argue. Nonetheless, in the absence of direct evidence regarding the long-term impact of new epidemiological conditions on the timing of cohort deaths, it seems preferable to continue to believe in the classic view of mortality conditions, based on period age-specific death rates.

7 Conclusion

This chapter first makes the distinction between two different purposes for calculating tempo-adjusted indicators in demography. The first purpose is the estimation of stationary-equivalent demographic levels, i.e., the levels that would be eventually observed in the population if all factors affecting demographic behavior remained constant in the future. The second purpose is the estimation of changes in the behavior of real cohorts. Since these two purposes have different solutions, the various methodologies for dealing with tempo adjustments need to be distinguished according to their objectives.

This chapter then shows that the performance of Bongaarts and Feeney's adjusted life expectancy as an indicator reflecting current mortality conditions depends primarily on the assumption that new mortality conditions generate delays in future cohort deaths that may be age-specific but need to be cohort-invariant (or, equivalently, time-invariant). At present, there is no clear evidence about the existence of such effects, although this may just reflect a gap in the existing knowledge regarding the dynamics of mortality in contemporary populations. Nonetheless, until the existence of such effects can be demonstrated, I argue that it is preferable to continue using the conventional life expectancy as an indicator of period mortality conditions.

The assumption of homogeneity, necessary for simulating the synthetic cohort in classic period life table construction, presents a challenge to the interpretation of the period life expectancy as an indicator of current conditions that is better documented than BF's tempo effects. If mortality risks vary across individuals, and if the frailty composition of the actual population differs from that of the stationary-equivalent population, the conventionally-calculated period life expectancy will be biased (Vaupel et al., 1979; Yashin et al. 1985; Pollard, 1993). There is a body of evidence suggesting that age-specific mortality rates are affected by earlier life conditions (Wilmoth, 1990; Elo and Preston, 1992), and that consequently period age-specific mortality rates do not completely reflect period mortality conditions. Unlike BF's conclusion that conventional e_0 provides too high an estimate of the stationary-equivalent e_0 level, recent research in this area suggests that conventional e_0 is *too low*, because the prevalence of disability in the population is higher than in the stationary-equivalent population (Lièvre et al., 2004). Similarly, Avdeev et al. (1998) have suggested that low levels of life expectancy in Rus-

sia in the early 1990s may provide too negative a picture of period mortality conditions because of increases in the proportion of frail individuals resulting from the abrupt mortality decreases of the late 1980s. While heterogeneity and tempo effects are two separate issues, they both address discrepancies between life expectancy under current rates and life expectancy under current conditions. Our current knowledge on both issues suggests that there may be a more urgent need for developing period life expectancy estimates that take heterogeneity into account.

Acknowledgments

An earlier version of this chapter was presented at the Bay Area Colloquium in Population (BACPOP) series, University of California-Berkeley, November 4, 2004; and at the Mortality Tempo Workshop sponsored by the Max Planck Institute for Demographic Research and the Population Council, New York, November 18-19, 2004. The author wishes to thank John Bongaarts, Sam Preston, Ken Wachter, John Wilmoth, and anonymous reviewers, for their useful comments.

References

Arthur, W. B. and Vaupel, J. W. (1984). Some general relationships in population dynamics. *Population Index*, 50(2):214–226.

Avdeev, A., Blum, A., Zakharov, S., and Andreev, E. (1998). The reactions of a heterogeneous population to perturbation. *Population: An English Selection*, 10(2):267–302.

Bongaarts, J. (1999). The fertility impact of changes in the timing of childbearing in the developing world. *Population Studies*, 53:277–289.

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. The quantum and tempo of life-cycle events. *In this volume, also published in Vienna Yearbook of Population Research*, pages 115–151. 2006.

Bongaarts, J. and Feeney, G. (1998). On the quantum and tempo of fertility. *Population and Development Review*, 124(2):271–291.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Brouard, N. (1986). Structure et dynamique des populations. la pyramide des années à vivre, aspects nationaux et exemples régionaux. [Population structure and dynamics. The later life pyramid, national aspects and regional examples]*, Espaces, Populations, Sociétés*, (2):157–168.

Elo, I. and Preston, S. (1992). Effects of early-life conditions adult mortality: a review. *Population Index*, 58(2):186–212.

Feeney, G. Increments to life and mortality tempo. *In this volume, also published in Demographic Research*, 14(2):27–46. 2006.

Goldstein, J. Found in translation. *In this volume, also published in Demographic Research*, 14(5):71–84. 2006.

Goldstein, J. (2003). Late but not never: The tempo and quantum of first marriage in France. Unpublished manuscript, Office of Population Research, Princeton University.

Guillot, M. (1999). The period average life (PAL). Paper presented at the 1999 PAA meeting, New York City.

Guillot, M. (2003a). The cross-sectional average length of life (CAL): A cross-sectional mortality measure that reflects the experience of cohorts. *Population Studies*, 57(1):41–54.

Guillot, M. (2003b). Does period life expectancy overestimate current survival? an analysis of tempo effects in mortality. Paper presented at the PAA 2003 held in Minneapolis, Minnesota.

Guillot, M. (2005). The momentum of mortality decline. *Population Studies*, 59(3):283–294.

Keilman, N. (1994). Translation formulae for non-repeatable events. *Population Studies*, 48(2):341–357.

Kohler, H. and J.A.Ortega (2002). Tempo-adjusted period parity progression measures, fertility postponement and completed cohort fertility. *Demographic Research*, 6(6):92–144.

Lièvre, A., Brouard, N., and Heathcote, C. (2003). The estimation of health expectancies from cross-longitudinal surveys. *Mathematical Population Studies*, 10:211–248.

Pollard, J. (1993). Heterogeneity, dependence among causes of death and gompertz. *Mathematical Population Studies*, 4(2):117–132.

Preston, S. and Coale, A. (1982). Age structure, growth, attrition and accession: A new synthesis. *Population Index*, 48(2):217–259.

Ryder, N. B. (1956). Problems of trend determination during a transition in fertility. *ilbank Memorial Fund Quarterly*, 34:5–21.

Ryder, N. B. (1959). An appraisal of fertility trends in the United States. *Thirty Years of Research in Human Fertility: Retrospect and Prospect,* New York: Milbank Memorial Fund, pages 38–49.

Ryder, N. B. (1964). The process of demographic translation. *Demography*, 1(1):74–82.

Ryder, N. B. (1980). Components of temporal variations in american fertility. In: Hiorns RW, ed. *Demographic patterns in developed societies,* London, Taylor and Francis Ltd., 1980 (Society for the Study of Human Biology. Symposia, 19:15–54.

Ryder, N. B. (1983). Cohort and period measures of changing fertility. in Rodolfo A. Bulatao and Ronald D. Lee (eds.), *Determinants of Fertility in Developing Countries, New York: Academic Press*, 2:737–756.

Ryder, N. B. (1986). Observations on the history of cohort fertility in the United States. *Population and Development Review*, 12:617–643.

Schoen, R. and Canudas-Romo, V. (2005). Changing mortality and average cohort life expectancy. *Demographic Research*, 13(5):117–142. http://www.demographic-research.org/volumes/vol13/5/.

Vaupel, J. Lifesaving, lifetimes and lifetables. *In this volume, also published in Demographic Research*, 13(24):597–614. 2005.

Vaupel, J., Manton, K., and Stallard, E. (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography*, 16(3):439–454.

Wachter, K. Tempo and its tribulation. *In this volume, also published in Demographic Research*, 13(9):201–222. 2005.

Wilmoth, J. (1990). When does a cohort's mortality differ from what we might expect? *Population: An English Selection*, 2:93–126.

Winkler-Dworak, M. and Engelhardt, H. (2004). On the tempo and quantum of first marriages in Austria, Germany, and Switzerland. *Demographic Research*, 10(9):231–263.

Yashin, A., Manton, K., and J.Vaupel (1985). Mortality and aging in a heterogeneous population: a stochastic process model with observed and unobserved variables. *Theoretical Population Biology*, 27(2):154–175.

Increments to life and mortality tempo*

Griffith Feeney

9 Fairview Road, Scarsdale, NY 10583, USA. E-Mail: feeney@gfeeney.com

Summary. This chapter introduces and develops the idea of " increments to life." Increments to life are roughly analogous to forces of mortality: they are quantities specified for each age and time by a mathematical function of two variables that may be used to describe, analyze and model changing length of life in populations.

The rationale is three-fold. First, I wanted a general mathematical representation of Bongaart's "life extension" pill (Bongaarts and Feeney in this volume p. 11) allowing for continuous variation in age and time. This is accomplished in sections 3-5, to which sections 1-2 are preliminaries. It turned out to be a good deal more difficult than I expected, partly on account of the mathematics, but mostly because it requires thinking in very unaccustomed ways.

Second, I wanted a means of assessing the robustness of the Bongaarts-Feeney mortality tempo adjustment formula (Bongaarts and Feeney in this volume p. 11) against variations in increments to life by age. Section 6 shows how the increments to life mathematics accomplishes this with an application to the Swedish data used in Bongaarts and Feeney (in this volume p. 11). In this application, at least, the Bongaarts-Feeney adjustment is robust.

Third, I hoped by formulating age-variable increments to life to avoid the slight awkwardness of working with conditional rather than unconditional survival functions. This third aim has not been accomplished, but this appears to be because it was unreasonable to begin with. While it is possible to conceptualize length of life as completely described by an age-varying increments to life function, this is not consistent with the Bongaarts-Feeney mortality tempo adjustment.

What seems to be needed, rather, is a model that incorporates two fundamentally different kinds of changes in mortality and length of life, one based on the familiar force of mortality function, the other based on the increments to life function. Section 7 considers heuristically what such models might look like.

1 Time-discrete increments to life

Figure 1 shows cohort survival for two birth cohorts of Swedish females. In the usual way of thinking, the survival curve for the later cohort has moved

up because risks of death have declined, but we might equally well think of the curve for the later cohort as having moved to the right as a result of the prolongation of life.

To quantify this idea, consider the earlier cohort, choose a particular age ($x = 50$ years, say) and consider the horizontal distance between the two survival curves at the corresponding survival proportion, $l_c(50, t_1) = 0.6666$ (Figure 1), t_1 denoting the time of birth of the earlier cohort. To calculate this distance we need to know the age to which this proportion of persons survive in the later cohort. Interpolating on the values for the later cohort we find this age to be 60.65 years, i.e., $l_c(60.65, t) = 0.6666$. The horizontal distance between the two curves at the ordinate value $l_c(50, t_1) = l_c(60.65, t_2) = 0.6666$ is thus $\lambda_c^{t_1,t_2}(50) = 10.65$ years.

The difference between any two survival curves may be described as the collection of all such horizontal distances. These "increments to life" are plotted in Figure 2. The increment for any given age represents "how much longer" persons in the second cohort live in a rather special and formal sense. The persons in the second cohort who survive to age $x + \lambda_c^{t_1,t_2}(x)$ live $\lambda_c^{t_1,n}(x)$ years longer than the persons in the first cohort who survive to age x. Their advantage is retrospective, however, not prospective. The increment to life for older ages may be smaller, zero or negative.

The area under the increments to life curve is the difference between the areas under the survival curves. Since the area under the survival curves gives the expectation of life at birth for the two cohorts, we have the following decomposition of the difference between the expectations of life at birth in the two cohorts in terms of the increments to life values,

$$e_0^c(t_2) - e_0^c(t_1) = -\int_0^\infty \lambda_c^{t_1,t_2}(x)\, dl_c(x, t_1) \tag{1}$$

where the integral is taken with respect to the first survivorship function.

2 Empirical results: Swedish females, 1751-2002

Increments to life by single years of age may be calculated for successive pairs of annual birth cohorts for Swedish females using the data provided in the Human Mortality Database (http://www.mortality.org). The database provides period life tables by single years of age to age 110 years for Sweden for (as of September 2004) 252 years, from 1751 through 2002. The q_x values from these tables may be used to compute cumulative cohort survival for the birth cohorts of persons born at the beginning of each calendar year. Applying the calculation of the preceding section to each successive pair of cohorts gives increments to life by single years of age for successive pairs of cohorts. These values may be arranged in a table in which rows correspond to single years of age and columns to pairs of adjacent birth cohorts and therefore to calendar years.

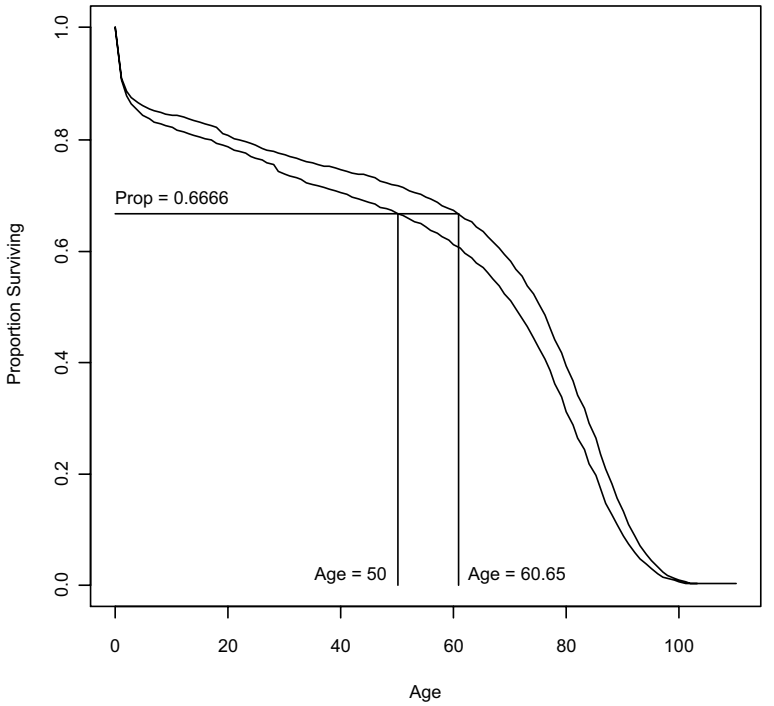


Fig. 1. Survivorship for Swedish female cohorts of 1890 and 1900.

Figure 3 shows increments to life averaged over successive pairs of birth cohorts for the period 1751-1760. It illustrates that increments to life may be negative as well as positive, corresponding to a rise in mortality risks and a decline in length of life. Figure 4 shows increments to life averaged over successive pairs of birth cohorts for the period 1891-1900. Values are positive here, and the age pattern quite different. The depression at young adult ages is notable.

3 Time-continuous cohort-indexed increments to life

Let $l_c(x,t)$ denote the proportion of persons surviving to age x in the cohort of persons born at time t. These values define a two-dimensional surface over the age-time plane of the Lexis diagram. This surface may be described by its contour lines, the lines on the age-time plane along which proportions surviving are constant. If length of life is constant, these contour lines will be straight lines parallel to the time axis. If length of life is increasing (decreasing), they will move to higher (lower) ages. The assumption that the population age

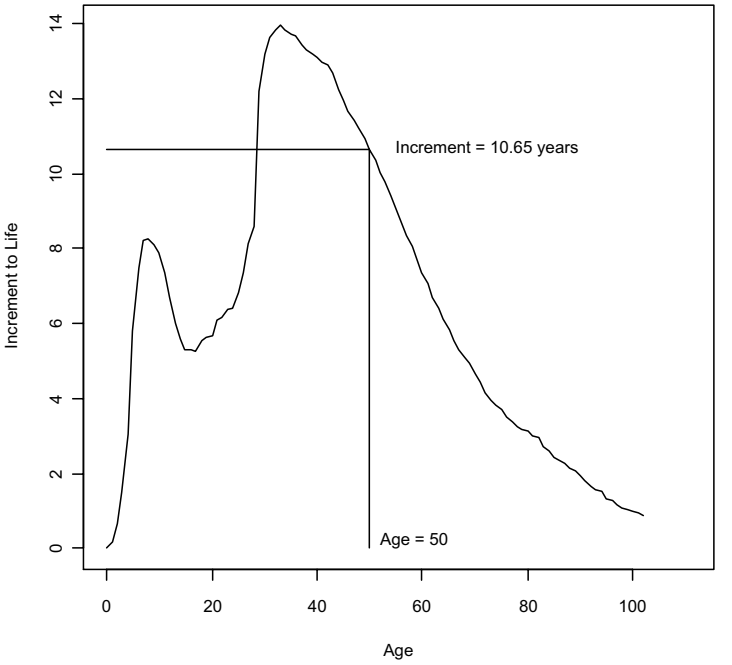


Fig. 2. Time discrete increments to life for Swedish female cohorts of 1890 and 1900.

distribution defined by $l_c(x,t)$ shifts to uniformly to higher ages (Bongaarts and Feeney 2002:16) is equivalent to the assumption that the rate of change of the contour lines with respect to age at any given time is invariant with respect to age.

Let the rate of change with respect to age of the contour line passing through the point (x,t) be $\lambda(x,t)$. The directional derivative of the surface defined by $(\lambda(x,t),1)$ in the direction $l_c(x,t)$ equals zero because the value of $l_c(x,t)$ does not change on the contour line. We therefore have

$$\frac{\partial l_c(x,t)}{\partial x}\lambda_c(x,t) + \frac{\partial l_c(x,t)}{\partial t} = 0\,, \tag{2}$$

where the constant factor in the definition of the directional derivative may be ignored since the value is zero. Formula (2) is equivalent to

$$\lambda_c(x,t) = -\left[\frac{\partial l_c(x,t)/\partial t}{\partial l_c(x,t)/\partial x}\right]\,, \tag{3}$$

which may be taken as the formal definition of the time-continuous cohort-indexed increment to life $\lambda_c(x,t)$ at age x and time . The partial derivative in the denominator shows that empirical increments to life values will tend

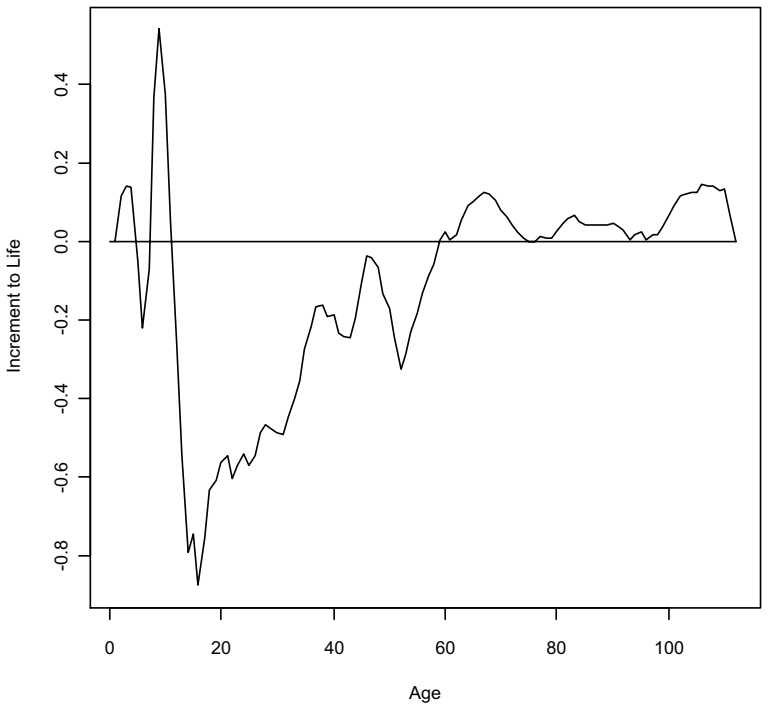


Fig. 3. Time-continuous cohort increments to life, Swedish females, average over cohorts of 1751-1760.

to be unstable over age intervals over which few deaths occur, since for these intervals $\partial l_c(x,t)/\partial x$ will be close to zero.

Dividing both sides of (2) by $l_c(x,t)$ and rearranging terms gives

$$\lambda_c(x,t)\mu(x,t) = r(x,t+x)\,, \tag{4}$$

where $\mu(x,t)$ denotes the force of mortality at age x and time t and $r(x,t)$ denotes the age-specific growth rate at age x and time t of the normalized population $l_c(\bullet,\bullet)$. This shows that values of the increments to life function vary inversely with the values of the force of mortality function for any given age and time.

The definition of increments to life by formula (3) supposes that the values $l_c(x,t)$ are given. If we assume instead that values $\lambda_c(x,t)$ are given, formula (2) defines a partial differential equation that may be solved for the values $l_c(x,t)$ given the boundary condition $l_c(x,t)$ for $x > 0$.

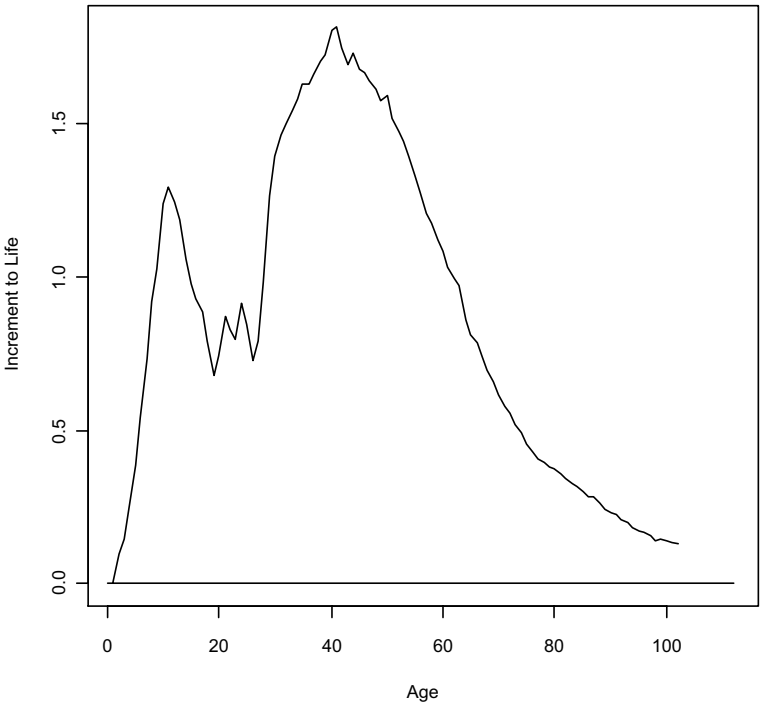


Fig. 4. Time-continuous cohort increments to life, Swedish females, average over cohorts of 1891-1900.

4 Time-continuous period-indexed increments to life

Let $l_p(x,t)$ denote the proportion of persons born at time $t-x$ who survive to age x. From this definition and that of $l_c(x,t)$ it follows immediately that

$$l_p(x,t) = l_c(x, t-x) \tag{5a}$$

and

$$l_c(x,t) = l_p(x, t+x) \tag{5b}$$

Compare Appendix 1 of Bongaarts and Feeney (1998), which states the same relation using slightly different notation. The subscripts refer to the cohort indexing of the preceding section and the period indexing of this section. Note that both $l_p(x,t)$ and $l_c(x,t)$ are survival proportions for cohorts; the difference is only in the time reference.

The apparently trifling difference between the two representations turns out to have non-trivial consequences. Proceeding as before, consider contour lines of the surface defined by the values $l_p(x,t)$. In the period case these contour lines may move backward as well as forward in time. Backward movement will occur whenever a later cohort experiences much lower survivorship than an earlier cohort.

Suppose for example that (a) for the cohort born at time , half of all persons survive to age 50 years, corresponding to the point $(50, t+50)$ and that (b) the cohort born at time $t+1$ experiences much higher infant mortality, with the result that the age to which half of all persons in the cohort survive is only 40 years, corresponding to the point $(40, t+41)$. The time coordinate of the point for the later cohort lies 9 years before the time coordinate of the point for the earlier cohort.

The time-continuous increment to life may still be defined as the direction for which the directional derivative equals zero, but this direction must now be specified as a vector rather than as a scalar. The period version of formula (2) is

$$\frac{\partial l_p(x,t)}{\partial x}\lambda_p^1(x,t) + \frac{\partial l_p(x,t)}{\partial t}\lambda_p^2(x,t) = 0\,, \tag{6}$$

where the vector $(\lambda_p^1(x,t), \lambda_p^2(x,t))$ gives the direction of the tangent to the contour line at the point (x,t). For consistency with the cohort formulation we may assume that $\lambda_p^2(x,t)$ assumes only the values $+1$ and -1, corresponding to movement forward and backward in time.

5 Relation between cohort and period increments to life

Figure 5 shows a Lexis diagram in which the diagonal line beginning at time t and ending at time $t+1+\lambda_c$ represents the tangent line to the contour line that passes through the point (x,t) of the surface $l_p(x,t)$. The slope of this line is by definition the period increment to life $\lambda_p = \lambda_p(x,t)$.

The corresponding rate of change between the cohorts born at times $t-x$ and $t-x+1$, represented by the dotted diagonal lines, is $\lambda_c = \lambda_c(x, t-x)$. From the similarity of the two right triangles,

$$\lambda_p(x, t-x) = \frac{\lambda_c(x, t-x)}{1+\lambda_c(x, t-x)}\,, \tag{7}$$

from which it follows that $\lambda_p(x,t) \to 1$ as $\lambda_c(x,t) \to \infty$ and $\lambda_p(x,t) \to -\infty$ as $\lambda_p(x,t) \to -1$. Values of $\lambda_c(x,t)$ less than correspond contour lines moving backward in time.

Zeng Yi and Land (2002) prove a special case of (7) for a model in which cohort fertility, period fertility, the shape of the age-schedule of fertility and the rate of change in the mean age at childbearing are all constant over time.

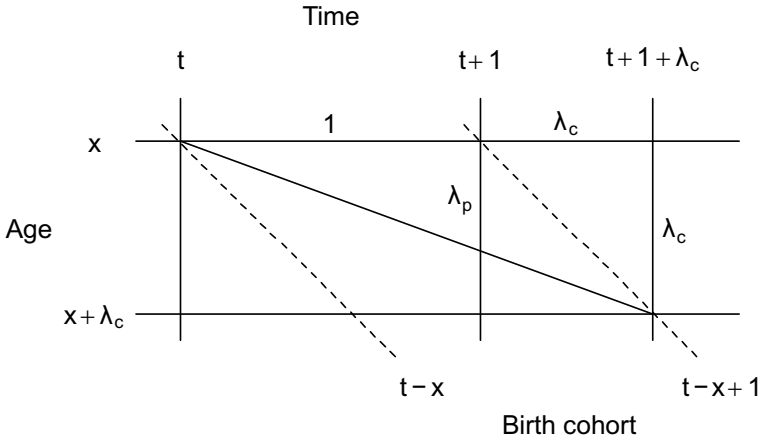


Fig. 5. Lexis diagram illustrating relation between cohort and period increment to life.

To obtain a more general formula, observe that the partial derivatives in (6) may be expressed as

$$\frac{\partial l_p(x,t)}{\partial x} = \frac{\partial l_c(x,t-x)}{\partial x} - \frac{\partial l_c(x,t-x)}{\partial t} \tag{8a}$$

and

$$\frac{\partial l_p(x,t)}{\partial t} = \frac{\partial l_c(x,t-x)}{\partial t}\,, \tag{8b}$$

these expressions being obtained by differentiating (5a). Substituting the right hand sides here in (6) and rearranging terms gives

$$\lambda_p^1(x,t) = \frac{-\partial l_c(x,t-x)/\partial t}{\partial l_c(x,t-x)/\partial x - \partial l_c(x,t-x)/\partial t} \tag{9a}$$

if $\lambda_p^2(x,t) = +1$ and

$$\lambda_p^1(x,t) = \frac{\partial l_c(x,t-x)/\partial t}{\partial l_c(x,t-x)/\partial x - \partial l_c(x,t-x)/\partial t} \tag{9b}$$

if $\lambda_p^2(x,t) = -1$. Dividing the numerator and denominator on the right hand sides of (9) gives

$$\lambda_p^1(x, t-x) = \frac{-\lambda_c(x, t-x)}{1+\lambda_c(x, t-x)} > -1\,, \tag{10a}$$

if $\lambda_p^2(x,t) = +1$ and

$$\lambda_p^1(x, t-x) = \frac{\lambda_c(x, t-x)}{1+\lambda_c(x, t-x)} < -1 \tag{10b}$$

if $\lambda_p^2(x,t) = -1$.Formula (10a) is the same as formula (7), but the graphical approach leaves it unclear how to cope with the case in which $\lambda_p^2(x,t) = -1$ or, equivalently, $\lambda_c(x,t) < -1$.

The relationship between ,$\lambda_c(x,t)$, $\lambda_p^1(x,t)$ and $\lambda_p^2(x,t)$ is shown in Figure 6. The curve to the right of the vertical at $\lambda_c(x,t) = 1$ shows the relation between $\lambda_c(x,t)$ and $\lambda_p^1(x,t)$ when $\lambda_p^2(x,t) = +1$ and the curve to the left of this vertical shows this relation when $\lambda_p^2(x,t) = -1$.

The relation displayed in Figure 6 is curious indeed. Discussion of tempo effects in the demographic literature has generally (always, so far as I am aware) been limited to values of λ_c and λ_p fairly close to zero (roughly, say, the unit square centered on the origin), and in this neighborhood the relationship is unremarkable. The Lexis diagram in Figure 5 shows that λ_p cannot exceed one, whereas λ_c may assume arbitrarily large values, so it is not surprising to see in Figure 6 that $\lambda_p \to 1$ as $\lambda_c \to \infty$. To see $\lambda_p \to -\infty$ as $\lambda_c \to -1$ is rather less comfortable (though obviously, from (10a), this is what happens), since this suggests that tempo effects in this case can have arbitrarily large magnitude. In demographic terms (Lexis diagram in Figure 5), events in successive cohorts are shifting to younger ages in such a way as to pile up events on the vertical line at time t.

The portion of Figure 6 to the left of the vertical (dotted line) at $x = -1$ is even more surprising. The idea that events occurring in successive cohorts may be moved to earlier ages so rapidly that the period effect is to "thin out" events and reduce period levels rather than to "bunching up" events and increase period levels has not, so far as I am aware, ever been considered in the demographic literature. Yet this is what happens when $\lambda_c < 1$. In demographic terms (Lexis diagram in Figure 5), events in subsequent cohorts are moved to earlier ages so rapidly that they occur earlier in time than events to earlier cohorts. The asymptotic approach to λ_p to the left of the vertical line (dotted) at $x = -1$ mirrors the asymptote on the other side, but with λ_c decelerating toward -1. Of course the value of λ_c is constrained on the left because events cannot be shifted to a time before the cohort's birth!

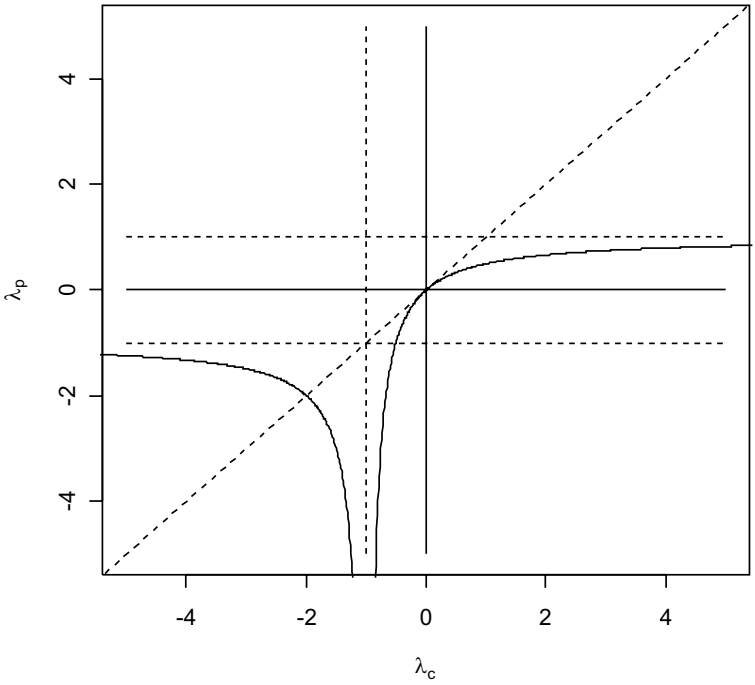


Fig. 6. Relation between cohort and period increments to life.

6 Robustness of the Bongaarts-Feeney tempo adjustment formula

The Bongaarts-Feeney mortality tempo adjustment formula (Bongaarts and Feeney 2002, in this volume p. 11) is based on the "constant shape assumption," which they show to be equivalent to the assumption that the normalized age distributions $l_p(x,t)$ are translated uniformly up or down the age axis with changing time. This is equivalent to the assumption that period increments to life $\lambda_p(x,t)$ are constant with respect to age for each time t, $\lambda_p(x,t) = \lambda(t)$ for all a. This suggests that tempo adjusted life expectancy at birth may be calculated more generally by replacing $\lambda(t)$ by $\lambda_p(x,t)$ in the Bongaarts-Feeney tempo adjustment formula (2003: formula 11, in which $\lambda(t) = \partial M_1(t)/\partial t$.

This adjustment may be applied to average of annual values of q_x for Swedish females for 1980-1995 with q_x set equal to zero for $x < 30$ years, the same Swedish data used in Bongaarts and Feeney (in this volume p. 11). Values of $\lambda_p(x,t)$ are obtained by first calculating $\lambda_c(x,t)$ using formula (3) and then applying formula (10) to obtain values of $\lambda_p(x,t)$. The resulting period increments to life by age $\lambda_p(x,t)$ are plotted in Figure 7, which suggests

that they are reasonably close to constant with respect to age from about age 35 onward.

Calculation of a tempo-adjusted e_0 using these values gives 79.5 years, as compared with an unadjusted value of $e_0 = 81.0$ years, for a tempo effect of 1.5 years. This is very close to the 1.6 years given in Bongaarts and Feeney (in this volume p. 11). I conclude that the simple, non-age-specific adjustment is robust against observed departures from the constant shape assumption in this application, and also that the increments to life concept has succeeded in providing a general method for assessing robustness.

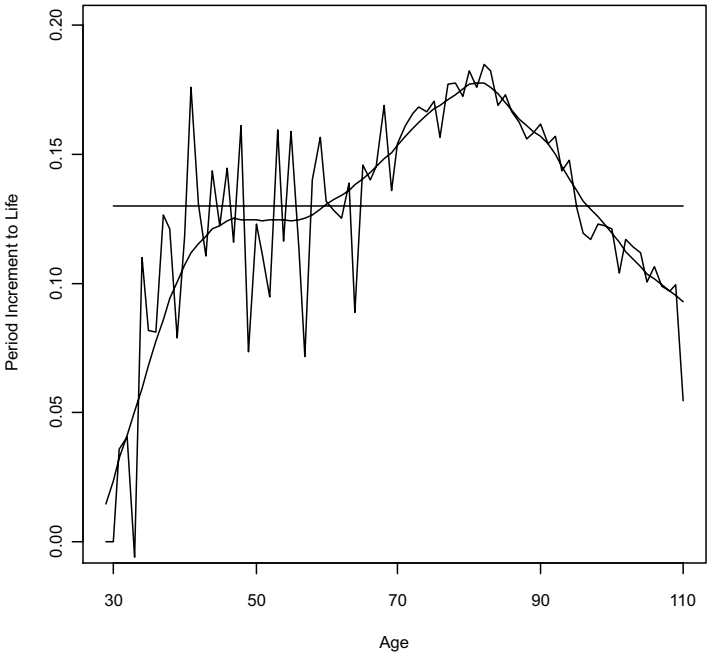


Fig. 7. Time-continuous period increments to life, Swedish females, 1980-1995 ($q_x = 0$ for $x < 30$ years).

7 Increments to life and mortality tempo: mixed models

What happens if the conditioning on survival to mid-adult ages is dropped and variable increments to life are substituted for the constant increment to life used in the Bongaarts-Feeney adjustment formula? The procedure described in the previous section gives in this case an expectation of life more than

5 years lower than the conventional expectation of life. The magnitude of the implied tempo effects is about three times larger than the tempo effects calculated by Bongaarts and Feeney.

The explanation for this discrepancy is evidently the age variation in increments to life shown Figures 3 and 4. The Bongaarts-Feeney mortality tempo adjustment is derived on the assumption that increments to life are constant with respect to age. When the survival function is conditional on survival to age 30 years, the Swedish increments to life 1980-1995 vary in a range of about ± 0.05, as shown in Figure 7. When the survival function is unconditional, increments are very far from constant. Figure 4 shows a variation of about ± 0.9. Conditioning on survival to age 30 has the effect of radically reducing the variability of increments to life by age.

Consistency with the Bongaarts-Feeney mortality tempo model therefore requires that increments to life be considered only for adult survival. The nature of mortality change at younger and older ages appears to be fundamentally different, so that the tempo model that makes sense at older ages does not make sense at younger ages.

This suggests that we need a "mixed" model in which mortality change at younger ages is modeled differently from mortality change at older ages. To suggest what such models might look like, consider the familiar graph of the force of mortality function with values (vertical axis) plotted against age (horizontal axis). Thinking heuristically, suppose that there are two kinds of mortality change, "up and down" change (movement in the vertical direction to higher or lower values), and "back and forth" change (movement of a fixed schedule of values in the horizontal direction, to the left or to the right). Suppose further than "up and down" change occurs in infancy, childhood and young adult ages, and that "back and forth" change occurs at older ages.

The force of mortality function may be most appropriate representation of "up and down" change, the increments to life function the most appropriate representation of "back and forth" change. The distinction may be captured mathematically by writing the Makeham force of mortality function as $\mu(x,t) = a(t)e^{bx} + c(t)$, where $c(t)$ represents "up and down" change and $a(t)$ represents "back and forth" change that may be equivalently expressed in terms of increment to life values $\lambda(t)$ representing the rate at which movement toward older or younger ages occurs.

So regarded, the Makeham defines a mixed model incorporating both forces of mortality and increments to life. Both components of the model could be generalized, to arrive at a more realistic model without changing the mixed nature of the model.

8 Conclusion

The study of mortality and length of life has been dominated by the concept of risks of death, to the point that mortality is sometimes regarded as

being *defined* by age-specific death rates and the force of mortality function. Empirically, however, survival functions are the theoretical structure closest to the empirical data (migration may be handled with product limit survival functions), and changing survival functions give rise to and may be modeled by both forces of mortality and increments to life.

When we think in terms of risks of death, life times are a residual. How long we live reflects how successful we are in escaping various risks of death. When we think in terms of increments to life, deaths are the residual. Death is what happens when we run out of life. As pointed out by Vaupel and Yashin (1987), physicians and health personnel tend to think more in the latter terms than the former. They suggest also that the two perspectives are complementary rather than contradictory. A better understanding of this complementarity may usefully advance the study of changing mortality and length of life.

Acknowledgements

This work has been supported by the Population Council. For comments on earlier drafts of this chapter I am grateful to John Bongaarts, to the participants in the *Workshop on Tempo Effects on Mortality* (18-19 November, New York City), especially to Jutta Gampe, and to two anonymous *Demographic Research* reviewers.

References

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population Development Review*, 28(1):13–29.

Vaupel, J. W. and Yashin, A. I. (1987). Repeated resuscitation: How lifesaving alters life tables. *Demography*, 4(1):123–135.

Yi, Z. and Land, K. C. (2002). Adjusting period tempo changes with an extension of ryder's basis translation equation. *Demography*, 39(2):269–285.

Mortality tempo versus removal of causes of mortality: Opposite views leading to different estimations of life expectancy*

Hervé Le Bras

École des hautes études en sciences sociales, 54 bd Raspail, 75006 Paris, France.
E-mail: lebras@ehess.fr

Summary. We propose an alternative way of dealing with mortality tempo. Bongaarts and Feeney have developed a model that assumes a fixed delay postponing each death. Our model, however, assumes that changes take place with the removal of a given cause of mortality. Cross-sectional risks of mortality by age and expectations of life therefore are not biased, contrary to the model of the two authors. Treating the two approaches as two particular cases of a more general process, we demonstrate that these two particular cases are the only ones that have general properties: The only model enjoying a decomposable expression is the removal model and the only model enjoying the proportionality property is the fixed delay model.

1 Introduction

A change in the timing of events does not exert the same influence on cross-sectional mortality indexes (i.e. life expectancy) as it does on fertility (i.e. the total fertility rate). The total fertility rate measures an intensity that is sensitive to the changing pace as well as delays or advances in events. In a life table, by contrast, the intensity remains equal to one because death occurs only once and all die in the end. Can we thus assert that timing has no effect on mortality indexes? In two recent papers, John Bongaarts and Griffith Feeney (2002, 2003 also published in this volume p. 11) have taken the opposite stance, showing that delays or advances in mortality modified cross-sectional life expectancy. They found that the delay observed led to an overestimation by 2.4 years in France and by 1.6 years in the US and Sweden.

Following a brief overview of the computations made by the two authors, we propose an alternative way of dealing with mortality changes ; using multiple decrement life-tables and removing different causes of death. In contrast to the delay model advocated by Bongaarts and Feeney, our model shows no discrepancy between cross-sectional and longitudinal indexes. We argue that

our model is more general than that of the above authors and more at pace with the true nature of mortality processes, the latter of which cannot be compared with nuptial or fertility processes. In brief, delays are a causal factor in the field of fertility and a consequential one in the field of mortality.

2 Decreasing mortality as a sign of delay in deaths

Let us suppose that on January 1st of year t, all deaths are suddenly postponed by a delay equal to a proportion u of the same year. When we follow this change on a Lexis diagram, the half plane at the right of vertical t is translated into a 45 degree direction by a vector (u, u). In the vertical strip stretching between t and $t + u$, there is no death at all. After $t + u$, the deaths reappear as before, this time, however, with a shift of u in age. Thus, instant life expectancy becomes infinite in the gap because no death occurs. After $t + u$, it reverts to its former value (i.e. the value before the change), increased by u. Despite the fact the changes occurred in $t = 0$, the cross-sectional value of life expectancy does not reflect the true conditions of mortality during a period of u until it resumes its actual longitudinal value in $t + u$. When we compute the mortality table on an annual basis during year 0 following the change, each number of deaths by age group is reduced by a proportion of u, independent of age. In the literature, such a change is called the "proportionality rule" and is in keeping with the observed data on modern countries. When we use these deaths to compute the forces (quotients) of mortality and to build a life table, we find an expectation of life that is higher than during the preceding and following years. This is an example of the discrepancy introduced between longitudinal and cross-sectional measures when delays occur.

The same discrepancy is found when the delay changes (increases) continually through time. Le $t = 0$ be the beginning of the process. The delay ending in t will be called $f(t)$, $s(x, t)$ denotes the survival function at age x and time t, and will be called $S(x)$ before $t = 0$, instead of $s(x, 0)$. With these notations, we get:

$$s(x, t) = S(x + t - f(t) - t) = S(x - f(t))$$

The deaths since time $t - f(t)$ or age $x - f(t)$ were not postponed by a delay greater than $f(t)$. From this we deduce an expression of deaths $d(x, t)dt$ between t and t+dt (and x and $x + dx$) and of the forces of mortality $q(x, t)$ by dividing these deaths by the survivors at that time and age:

$$\begin{aligned} d(x,t)\,dt = d(x,t)x &= s(x,t) - s(x+dt, t+dt) \\ &= S(x - f(t)) - S(x + dt - f(t+dt)) \\ &= S(x - f(t)) - S(x - t(t)) - (1 - f'(t))S'(x - f(t))\,dt \\ d(x,t) &= (1 - f'(t))D(x - f(t)) \end{aligned}$$

Consequently

$$q(x,t) = (1 - f'(t))\,\mu(x - f(t)) \tag{1}$$

where $\mu(x)$ is the force of mortality at age x and initial time $t = 0$. A simple case is that of a linear evolution of the delay at rate α which means that $f(t) = \alpha t$. It follows that:

$$d(x,t) = (1 - \alpha)D(x - \alpha t) \tag{2}$$

The same relationship holds for the forces of mortality. By integrating them, the relationship can be expressed in terms of survivors:

$$s(x,t) = (S(x - \alpha t))^{1-\alpha}$$

Cross-sectional life expectancy at time t, $e(t)$ is:

$$\begin{aligned} e(t) &= \int_0^w S(x,t)\,dx = \int_0^w (S(x - \alpha t))^{1-\alpha}\,dx \\ &= \int_0^w (S(x,t))^{1-\alpha}\,dx + \alpha t \end{aligned}$$

If the delay is stabilized just after t, then the longitudinal expectation of life $E(t)$ will become

$$\mathrm{e}(t) = \int_0^w S(x)\,dx + \alpha t \tag{3}$$

The more the computation of $e(t)$ overestimates the gain in life expectancy, the more rapidly the delay increases. For example, in Table 1 we computed the discrepancy value of the most recent life table for France for different values of the rate of increase (the delays were taken into account at age 36 and over).

Table 1. Overestimation of life expectancy at various rates of increase of the delay.

Annual rate of increase of the delay (%)	Overestimation(discrepancy)
5	0.44
10	0.86
15	1.26
20	1.65
25	2.02

At the rate of 25% we find a value not far from the one found by Bongaarts and Feeney (2002, in this volume p. 11). This is due to the actual rate of increase in life expectancy in France, which is approximately a quarter of a percent each year.

The entire process, in mathematical terms, results in a change in the time scale but not in the age scale, thus it is not necessary to go into further detail on the equations. The change in the time scale can be compared to a twisting of the life-lines on the Lexis diagram by a continuous deformation. In t, the deaths occurring during a small interval Δt can be written as: $d(x - f(t))(1 - \partial f(t)/\partial t)\Delta t$; the survivors, $S(x - f(t))$, and the life table corresponding to the forces computed by dividing the deaths by the survivors have the survivorship functions:

$$S(x,t) = S(x - f(t))^{\beta(t)} \tag{4}$$

with $\beta(t) = (1 - \frac{\partial h(t)}{\partial t})$

3 Decreasing mortality as a change in the causes of death

One can consider evolution by processes other than delays and advances of events in order to analyze more precisely how the mortality process works. When we compare the distribution of the actual causes of death, we see very significant differences in comparison to the past (for example, the oldest table published in 1661 by Petty and Graunt in *Natural Observations...*). Some causes, such as smallpox, have disappeared or have become negligible, (measles, infectious diseases and appendicitis are among these). This is the true process by which mortality diminishes. There are two reasons why this historical process can not be simulated with small delays added to the life of every individual in the population. Firstly, only those who contracted the fatal illness (before they could be cured) are affected (i.e. not everybody is affected), and secondly, after being cured, the delay or added expectation of life is quite large and does not receive a definite value. Let us start with a very simple situation: At time $t = 0$, a successful treatment for a certain cause of death is discovered and applied to every patient who is consequently saved from death. We assume, as is common in the computations of removed causes of mortality, that the individuals thus saved do not suffer the after-effects of the treatment, and they can enjoy the same mortality pattern as all other individuals of the same age who are not affected. This means, that since the first instant $t > 0$, all individuals are dying, following the law of mortality where all other causes are unchanged and one particular cause is removed. At any time after $t = 0$, the forces of mortality and the expectations of life are constant and correspond to the new life table, which is the longitudinal table if no other change occurs. In contrast to the results obtained by Bongaarts and Feeney when they introduce a delay, no temporary large increase in life expectancy is observed. No discrepancy arises between cross-sectional and

longitudinal indexes and no correction is needed. This result requires more detailed explanations and a mathematical proof.

4 Removing one cause of death: deeper insights

The method of suppressing causes of death leads to a delay in death, but there are different forms of delay. In the case postulated by Bongaarts and Feeney (we will call it the delay method or model), the delay applies to all individuals and is independent of age. As to the suppressed cause of death (we will call it the removal method or model), the delay affects only those struck by the particular cause, and this depends strongly on age. More precisely, let $(S, \mu, D), (S_1, \mu_1, D_1), (S_2, \mu_2, D_2)$, respectively, be the life table (survivors, forces of mortality, deaths), first before the change, second for the specified cause, and third after the removal of this cause. This results in two very simple relationships:

$$\mu_1 + \mu_2 = \mu(x) \tag{5a}$$

and

$$S(x) = S_1(x)S_2(x) \tag{5b}$$

Let us describe in greater detail the different stages of the decline in mortality: before $t = 0$, the population follows the first mortality pattern (S, μ, D). Since time $t = 0$, amongst those $\mu(x)S(x)$ who are assumed to die, $\mu_2(x)\mathrm{d}x$ do so and $\mu_1(x)\mathrm{d}x$ are saved and follow from this point the second pattern of mortality (S_2, μ_2, D_2). Therefore they have a probability density of $k(u) = D_2(x + u)/S_2(x)$ of dying after delay u. This is in line with the hypothesis of independence of the causes of mortality: After being cured, the probability of dying from another cause is the same as in the general population of the same age. From these remarks, we can compute the deaths $d(x, t)$ at age x and time t by taking in t at age x the endpoint of all delays (including 0):

$$d(x,t) = \int_0^t S(x-u)\mu_1 D_2(x)/S_2(x-u)\, du + \mu_2(x)S(x) \tag{6}$$

Similarly, the survivors $s(x, t)$ are those who have survived any cause of mortality at age x and $S(x)$ and those who were hit at a former time $t - u$ by the cause of death that was removed and whose delays are superior to u:

$$s(x,t) = S(x) + \int_0^t S(x-u)\mu_1(x-u)S_2(x)/S_2(x-u)\, du \tag{7}$$

When dividing the deaths by the survivors, one can compute the forces of mortality $q(x,t) = d(x,t)/s(x,t)$. As demonstrated in Appendix B, after some mathematical manipulations we arrive at: $q(x,t) = \mu_2(x)$. The forces are independent of t at any time after $t = 0$ and they are the same as those of the actual longitudinal pattern of mortality after $t = 0$.

A sudden change in mortality preceded and followed by complete stability is not very realistic. The method of introducing a sudden change was used here, and was in the paper by Bongaarts and Feeney as well, to introduce a change of delays through time and not only at a fixed point in time. The same generalization as in delay can be made for the removal of a cause of death. We can assume that this removal is made at intervals T/n in n stages, each accounting for $1/n$ of the force of mortality $\mu_1(x)$. Because the life table corresponding to the removal of each successive change is immediately followed by the population, the result, applicable separately for each elementary stage, holds for the whole change and can be made continuous by increasing n to infinity. For the same reason, the change does not need to be regular but can follow any time path. It is not even necessary to use the same age pattern; the only requirement is to have the table at the end without the removed cause or causes (S_2, μ_2, D_2).

5 A numerical example of the two methods

The preceding results are abstract ones. Let us be more concrete in comparing the two methods using the same example. We assume the law of mortality defined over five years with survivorship function $(100, 60, 30, 10, 0, 0)$ at age $(0, 1, 2, 3, 4, 5)$. First, let us assume that a delay by half a year is gained by some mean since the beginning of the process at $t = 0$ (Bongaarts and Feeney call this mean a " survival pill ") that automatically expands life by six months. If the deaths are spread regularly over each age, then we observe at each age only half of the deaths of the preceding years during the year following $t = 0$ (Table 2). Thereafter, the deaths will be as numerous as before but shifted to half a year later (under the assumption that the newborns also take the miracle pill). Table 2 can be extended indefinitely, but this is not necessary because the numerical values are stabilized as of the second year after the change in the number of deaths, as well as for the survivors and quotients. The total number of deaths is the same as the preceding year 0, but the expectation of life is extended by half a year.

Now, let us take the same life table as in Table 3, before (year -1) and after (year 1) the change, but suppose it results from a sudden change of pattern due to the removal of a cause of death at time $t = 0$. For that cause, the quotients of mortality Q_1 are such that

$$1 - Q_2(x) = (1 - Q_1(x))(1 - Q(x))$$

which gives the following tables:

Table 2. Quotients of the life table before and after the removal of one cause.

Age x	Final table $Q_2(x)$	Mortality cause removed: $Q_1(x)$	Initial table $Q(x)$
0-1	0.20	0.25	0.40
1-2	0.44	0.11	0.50
2-3	0.56	0.25	0.67
3-4	0.75	1.00	1.00
4-5	1.00		

Each year following $t = 0$, the survivors of the initial table are distributed in three groups: those who die from a cause of death in the final table, those who are facing death but survive, and those who will survive anyway. The second group dies according to the figures in the final table. For example, amongst the 40 foreseen deaths of the initial table at $t = 0$, 20 die as given by the quotient of the final table, and the other 20 follow the pattern of the second table. Similarly, of the 30 individuals of the initial table who are prone to die within one or two years, 26.25 (30 x 0.44) die and 3.75 survive according to the figures in the final table. The survival times of those cured from the removed cause of death are distributed by duration, as shown in Table 4.

Table 3. An example of delay in mortality.

	Survivors and deaths										**Quotients**			
Time	**-1**		**0**		**1**		**2**		**3**		**-1-0**	**0-1**	**1-2**	**2-3**
	Sur	**D**	**Sur**	**D**	**Sur**	**D**	**Sur**	**D**	**Sur**	**D**				
Age														
0	100		100		100		100		100					
		40		20		20		20		20	0.40	0.20	0.20	0.20
1	60		60		80		80		80					
		30		15		35		35		35	0.50	0.25	0.44	0.44
2	30		30		45		45		45					
		20		10		25		25		25	0.67	0.33	0.56	0.56
3	10		10		20		20		20					
		10		5		15		15		15	1.00	0.50	0.75	0.75
4	0		0		5		5		5					
		0		0		5		5		5			1.00	1.00
Deaths (Total)	100		50		100		100		100					
Life expectancy	1.5		2.5		2.0		2.0		2.0					

With this distribution, it is now possible to establish, at each successive year, the balance of deaths in the same way as in the preceding case of a given delay. We arrive at a similar table, in which the number of deaths is

Table 4. Distribution of durations after the given cause of death was suppressed.

Age when the given cause is cured	Survival duration (years) 1	2	3	4	Total surviving at the beginning
0-1	8.75	6.25	3.75	1.25	20
1-2	2.08	1.25	0.42		3.75
2-3	2.50	0.83			3.33
3-4	2.50				2.50

computed according to the delays ending in t at age x. For example, the 11.25 (7.5+2.5+1.25) dead during period 3-4 at age 3 in completed years come from 7.5 with delay 0 because they died from a cause other than the suppressed one, 2.5 with delay 1 from the preceding year and 1.25 with delay 2 coming from the preceding two years (see Table 4).

We see in Table 5 that the quotients and expectations of life reach directly and immediately their value in the second or final table where the given cause is removed. However, deaths and survivors rejoin the structure of the final table only after four periods, which is the length of the greatest delay. The overall structure displayed in Table 5 is more general than that of Table 2. The quotients of the given cause can take any value smaller than the quotient of the initial life table at the same age (the only restriction.). No proportionality hypothesis or rule is further required.

6 Which life table is the reference table?

Bongaarts and Feeney contrast three ways of computing life expectancy: firstly, summing the survivors over the life course, secondly, taking the mean age at death in the standardized distribution of deaths, and thirdly, starting with the forces or quotients to construct the life table. In the real world, the last method is by far the most common one applied by statistical offices. The distributions of survivors or deaths are seldomly handled directly since they reflect generational history and are distorted by migrations. A cross-sectional life table is not intended to represent a remote past but to capture the actual trend. A usual justification of the cross-sectional life table, the *fictitious generation*, is that it can be observed in a generation for which all the observed data at time t are frozen and reproduced in the future during the time span of a generation.

This pseudo-empirical definition is not very useful. It seems better to freeze the causes that command the data rather than the data itself. With this causal approach, a good cross-sectional life table at time t is one that would be observed in a generation if suddenly all the parameters that configurate mortality were instantaneously immobilized at time t, including of course the advances and delays or the removal of some causes of mortality. In the

Table 5. An example of the removal of a mortality cause.

Survivors and deaths												
Time	**-1**		**1**		**2**		**3**		**4**		**5**	
	Sur	**D**	**Sur**	**D**	**Sur**	**D**	**Sur**	**D**	**Sur**	**D**	**Sur**	**D**
Age												
0	100		100		100		100		100		100	
		40		20		20		20		20		20
1	60		60		80		80		80		80	
		30		26.25		35		35		35		35
2	30		30		33.75		45		45		45	
		20		16.67		18.75		25		25		25
3	10		10		13.33		20		20		20	
		10		7.50		10		11.25		15		15
4	0		0		2.50		3.33		5		5	
		0		0		2.5		3.33		3.755		5
Deaths (Total)		100		70.42		86.25		94.58		98.75		100
Life expectancy	1.5		2.0		2.0		2.0		2.0		2.0	

	Quotients			
Time	**-1-0**	**0-1**	**1-2**	**2-3**
Age				
0				
	0.40	0.20	0.20	0.20
1				
	0.50	0.25	0.44	0.44
2				
	0.67	0.33	0.56	0.56
3				
	1.00	0.50	0.75	0.75
4				
			1.00	1.00
Deaths (Total)				
Life expectancy				

preceding example where the two ways of change have been explored, the life table of reference is called the final life table. Its survivor and death functions emerged rapidly according to the delay method and progressively when a cause of mortality had been removed. However, with the removal method, the final table was immediately given by its quotients, in contrast to the delay method where fluctuations took place until the fixed delay was achieved. The issue, therefore, is not whether to choose between an estimation from the quotients, the survivors, or the deaths, but rather which life table constructed in the usual manner from the quotients provides an exact estimate of the

longitudinal table corresponding to the " frozen " causes. Herein lies the large discrepancy between the model of delay and the model of removal.

What is meant by " frozen " causes is clearer than what is meant by "fictitious" generation, but the former still needs to be explained in greater detail. To a certain extent, the assumed behavior depends on the model adopted. A difference can be noted between the two models of a single change in $t = 0$. In the case of removal, there is no direct transformation of the cross-sectional data to longitudinal data, but a continuation of the process until the last delay is completed and all the distributions stabilized, as shown in Table 5. In the delay process, the stabilization is more rapid, as shown occurring in the second period in Table 2. However, in each case, the longitudinal life-table does not exist on an empirical basis during the stabilization process. It is only defined as a process that converges more or less rapidly according to the model selected.

Continuous change of causes through time is more problematic. In the removal model, the quotients of the longitudinal "frozen" table are immediately reached, owing to instantaneous adjustment. But with a change in delay $f(t)$ through time t, the question arises: What is the behavior to be frozen at time t? Is it the behavior corresponding to the last (observed) delay ending just at t which began at $t - f(t)$ and consequently, the behavior in $t - f(t)$, or the behavior corresponding to the delay (non-observed) starting just in t ? The second solution is a more rational one, but the length of the delay beginning in t is unknown and will only be determined when it ends, by the mean age at the standardized deaths. The difference is not small, as the following example illustrates: if $f(t) = \alpha t$ (linear case), with t being the end point of the delay (see Appendix C), the delay beginning in t amounts to $\alpha t/(1-\alpha)$. The difference d to delay αt ending in t is quite high: $d = \alpha t/(1-\alpha) - \alpha t = \alpha^2 t/(1-\alpha)$. If the rate of increase is 25% and $t = 30$ (as in Europe), $d = 2.5$ years, and amusingly, equal to the overestimation computed by Bongaarts and Feeney. That is, there are no definite "frozen" causes in the delay model. Furthermore, there is naturally no empirical reference that can be used for building the hypothetical life-table during the process. If one needs to design a correction procedure, the reference must be defined prior to the correction, but this is not feasible in any circumstance.

7 Unifying the two views: the repartition function of the delays by age and duration

The only difference between the two methods rests in the way by which the delays are postulated in formulating the two methods. We propose a more general model that covers the two instances considered so far. By the same token, we will demonstrate that they have strong and unique properties. Let us call $\lambda(x - u, u)$ the density proportion of deaths foreseen to occur at age $x-u$ and delayed at time $t-u$ until age u is reached at time t, and let $\theta(x)$ be

the proportion of deaths at age x non-delayed. By counting all delays which end in t at age x , we arrive at the number of deaths[2] :

$$d(x,t) = \int_0^1 S(x-u)\lambda(x-u,u)\,du + \theta(x)S(x) \tag{8}$$

Using the same procedure, we recount the survivors as all those who have either never directly been threatened by death, or, if threatened, are cured and alive:

$$s(x,t) = S(x) + \int_0^t S(x-u)\left(\int_u^w \lambda(x-u,v)\,dv\right)du \tag{9}$$

The two methods analyzed can be rewritten as follows:

for the removal method:

$$\lambda(x,v) = (\mu(x) - \theta(x))D_2(x+v)/S_2(x)$$
$$\theta = \mu_2(x)$$

for the delay method:

$$\lambda(x,v) = \delta(v-T)\mu(x)$$

where δ stands for the Dirac function and T for the fixed delay, $\theta(x) = 0$.

Both methods have special properties. The removal method is the only decomposable method for which the forces of mortality by age adjust instantaneously to the final or longitudinal quotients of the table without the removed cause. The delay method assumes a fixed and common delay $f(t)$ for every member of the population at any time t.

It is not a difficult but rather a tedious task to demonstrate the existence of these two properties. They are developed in Appendixes B and C. The first property is a good justification for using cross-sectional life expectancy as an indicator of longitudinal tendencies. It reveals, again, a large difference to fertility, where the total fertility rate is an inappropriate indicator of the evolution of the total number of children ever born.

The delay method is quite restrictive. Appendix C shows that it implies a common duration in the delay for all individuals. At time $t - f(t)$, each death is delayed by $f(t)$ exactly. This is not evident at first sight because the method starts from the proportionality rule. Nevertheless, it is a necessary consequence of the assumptions made. We can illustrate this with a simple

[2] The formulae could be made simpler by working with Lebesgue measures instead of Riemann Integrals. All the terms would be put under the integral sign, $\theta(x)$ becoming $\lambda(x,0)\,\delta(x)$. However, we prefer to keep contact with the real process, distinguishing those who enjoy a delay from those who die directly.

model. In a single delay in $t = 0$, assume that delay T applies only to a proportion p of the foreseen deaths at each age instead of being universal. The other $1 - p$ deaths are in time according to the initial life-table. For $t < T$, the formulae (I) and (II) take the form:

$$d(x,t) = (1-p)\mu(x)S(x) = (1-p)D(x)$$
$$s(x,t) = S(x) + p(S(x-t) - S(x)) = (1-p)S(x) + pS(x-t)$$

The resulting force of mortality is: $q(x,t) = \mu(x)/(1+pS(x-t)/((1-p)S(x))$.

We see in the formula that the proportionality rule no longer exists, the denominator varying with age x. When delay T is over, the force of mortality becomes:

$$q(x,t) = ((1-p)D(x) + pD(x-T))/((1-p)S(x) + pS(x-T)). \qquad (10)$$

The shift in the survivorship function is no longer constant, and depends on age because of the varying slope of $S(x)$. The proportionality rule no longer applies. These remarks hold when the delay is not the same for all individuals but follows a probability distribution. A discussion of this issue is provided in Appendix C. The delay method is, therefore, restrictive. It supposes that at time $t - f(t)$ every death without exception is postponed by a fixed duration $f(t)$. From an empirical view, this seems unlikely. Below, we discuss the features and the likelihood of each method, delays or removal of the causes of death.

8 Which is the best model? A discussion of the two methods

The two models are now embedded in a common pattern of delays depending on age and time, yet the difference in the results is, so far, not suppressed. The delay model reveals an overestimation or an underestimation of life expectancy according to an increase or decrease in mortality. In the removal model, no overestimation, yet rather a correct estimation of the longitudinal trend, can be seen. Which method is the most appropriate one? Which model provides a more accurate representation of the process of changing mortality? The answers are found in the comparative handling of delays and risks. As its name indicates, the delay method moves the delays forward in time. The risks measured by the quotients and forces are the results of changing delays, which are the causal factor. The removal method, by contrast, sees the mortality change as a process of changing risks pertaining to certain causes. The delays are the consequence of changing risks through which the acting causes are channeled.

Let us begin with the simple model of a single change at $t = 0$. Can we make the assumption of a delay by a few months for each foreseen death? With very old and very sick people, the delay can depend on euthanasia. But most of the deaths are not imputable to extreme age or to severely deteriorated physical conditions. Infectious diseases (some of which are contracted in hospital), accidents, cancers and heart attacks, when cured, give a large and aleatory respite whose average is the expectation of life at the age of the patient. The word aleatory is important here: It is impossible to fix an individual delay for any individual, to assure the individual that he or she will stay alive for a specific time. A murder, an earthquake, or a new disease can hurt the individual. In fertility, the situation is different. If one wants to avoid birth during a given period, one can rely on contraception and abortion in case of unwanted conception. The delays play a major role in fertility and nuptial processes because they involve the will. One can postpone the wedding day, even postpone it indefinitely, but one cannot do the same for the day of death, which is only partially influenced by our will.

There is another difficulty with the delay model. As demonstrated, all individual delays $f(t)$ ending at time t are the same. Furthermore, they are postponed only once. If we enter the full details of the longitudinal process of mortality into the delay model, when a death is foreseen at age x, then the death is postponed until age $x + f(t)$ but at that age, it becomes certain. There is no second chance of delay. It means that the population is split into two groups; those who are subject to a known date of death and those whose death has never been delayed. The expectations of life differ considerably in the two groups. In the removal model, by contrast, this difference disappears. After removing the given cause, all individuals run the same risk of death at a given age x. As mentioned before, the delays do not matter; they are the result of the process, not its cause. The new or final life-table is computed by changing the overall risks at each age in substracting the force of mortality of the given cause from the overall force of mortality. In the delay model, the new life-table is computed by the mean of the added delays. If we take a very long term view of mortality, say, from the Cro Magnon Era onwards, the process of mortality in the delay model results in the sum of many small delays of survival, each certain and the same for all individuals. This does not concur with the data on mortality. In such a model, there is no way to differentiate between individuals and no room for chance, and if it existed, then the resulting curve of deaths according to age should be Gaussian.

The removal model seems to be the more realistic one. In the long term, it depicts mortality as a process of removing the causes of mortality one after the other. To a demographer, this corresponds well with the analysis of mortality by cause and with the techniques of the multiple decrement life-table. In summation, mortality appears to be more of a multiplicative process than an additive one. One could say, nevertheless, that the language of delays and the language of causes of mortality are two different expressions of the same reality. This is not true, however. Knowing the delays $T = f(t)$, we can

compute the corresponding gain in risks at each age using the same notations as before:

$$\mu_2(x) = \mu(x) - \mu_1(x) = \mu(x - T)$$
$$\mu_1(x) = \mu(x) - \mu(x - T)$$

If $\mu(x)$ follows the Gompertz law, $\mu(x) = Ae^{rx}$, $\mu_1(x)$ and $\mu_2(x)$ also follow this law with the same exponent but with a different scale factor:

$$\mu_2(x) = Ae^{rx}(1 - e^{-rT})$$
$$\mu_1(x) = Ae^{rx}(e^{-rt})$$

This property seems to be an empirical argument in favor of the delay method, because in the developed countries since the 1970s, the reduction of mortality has followed this pattern. Yet, as the preceding equations show, the same pattern can be generated with the removal of a cause of mortality displaying the same Gompertzian slope as the general mortality. Moreover, there is a one-to-one correspondence between delays and risks. In general, a given profile of risk by age has no equivalent in terms of delay. The removal method is more general. It allows for any risk profile, with $\mu_1(x) < \mu(x)$ being the only condition, whereas the delay method imposes a specific age profile of the risks.

In brief, the removal method is better suited to the process of mortality:

- Life expectancy after a postponed death is likely large, and not limited to a few months. However, if the delay is long, it provides a long duration with no risk of mortality. This is an unrealistic assumption, considering the nature of mortality.
- The delay is the same at any age in the delay method, yet it varies according to the expectation of life at age x in the removal method. This variation seems more realistic.
- The reference to the initial and final life tables is straightforward in the removal model, but not well defined during the course of the delay in the delay model.
- In the removal model, there is always one single population with every individual at age x who is threatened at any time by the same force of mortality, either $\mu(x)$ or $\mu_2(x)$. In the delay model, some individuals experiencing delay are exposed to high risks and are near death, whereas the other individuals remain exposed to the usual risks.
- The removal model is coherent with the analysis of mortality in terms of multiple decrements.

Until now, a comparison was drawn only in the simple case of a unique and sudden change in $t = 0$. Does this result hold when mortality varies

continuously? In these circumstances, a comparison between the two methods shows further advantage of the removal method. All preceding remarks still apply. One additional remark sharpens the difference, where the delay method raises the problem of reference life tables. One cannot take a reference table that was computed in a former epoch and that can embody bias, as found by Bongaarts and Feeney, and counterbalance its effect. What former life table brings the guarantee of having been computed under stationary conditions? As discussed, the longitudinal reference life table cannot be well defined in this respect. It cannot be computed at t, that since $t - f(t)$ the delays were held constant, and the delays beginning after $t - f(t)$ are not known because they can be determined only when they come to their end. The removal method does not raise such problems. At each point in time, the observed life table is the reference table.

It does not follow from the discussion that any change of mortality pertains to the removal method. Only the long term changes or the trend in mortality obey such a model. In the short term, many causes of fluctuation are at work, suffice it to say seasonal variations related to atmospheric conditions, cold or hot weather, and influenza more severe than usual. They can delay or advance some deaths, but their effect is negligible on the average at a medium term range. The following statement could stem from Solomon: For the short term, take the delay method and for the long term, take the removal method. For short term fluctuations, the proportionality rule which is crucial for the working of the delay method, is not observed. Influenza, hot or cold weather, humidity or dry weather conditions, have a negative effect on very young and very old persons. A good example was provided by the heat wave that hit France in August 2003. The rates of mortality following the wave and computed for the age groups are reported in Table 6. Here, we can see how late and accelerated the increase of the probability of death was, and how far we are from proportionality when we compare these rates to the overall quotients at the same ages provided by the most recent French tables (2001).

The best way to tackle seasonal accidents remains the multiple decrement life table. It allows computing the decrease of life expectancy. The interest focuses on $\mu_1(x)$ and not on $\mu_2(x)$. This is because it is clear that the change is not a permanent but an accidental one. In any case, the removal method should be preferred to the delay method and no correction is needed.

Table 6. Rates of death caused by the heat wave (France, August 2003) compared to the general quotients of mortality.

Age group	Heat wave mortality rates (for one million)		Overall quotients (for one thousand)	
	Men	Women	Men	Women
60-64	115.000	50.000	65	27
65-69	244.000	138.000	99	41
70-74	396.000	281.000	149	69
75-79	786.000	673.000	226	122
80-84	1.901	1.923	356	227
85-89	2.759	2.821	528	400
90-94	5.702	6.696	712	620
95 and +	9.900	12.431	809	780

References

Avdeev, A., Blum, A., Zakharov, S., and Andreev, E. (1998). The reaction of a heterogeneous population to perturbation. *Population*, 10(2):267–302. (Eng. sel).

Beaumel, C., Doisneau, L., and Vatan, M. (2003). *La situation dmographique de la France en 2001.* INSEE, Paris.

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Graunt, J. (1661). *Natural and political observations...* John Martyn, London.

Le Bras, H. (1993). Simulation of change to validate demographic analysis. In Reher, D. and Schofield, R., editors, *Old and new methods in historical demography*, pages 259–280.

Ryder, N. B. (1964). The process of demographic transition. *Demography*, 1:74–82.

Appendix A: Deaths and survivors of age x in t after the removal of a mortality cause in $t = 0$

The aim of the computation is to show that the force of mortality in t and x is independent of t and equal to its final value $\mu_2(x)$. We have seen that the distribution of delays u in $t = 0$ at age x was $u = 0$ in $\mu_2(x)$ cases and $f(u) = D_2(x + u)/S_2(x)$ in $\mu_1(x)$ cases. Conversely, the deaths at age x in t can be computed by adding up all the delays terminating in t at age x:

$$d(x,t) = \int_0^t \frac{S(x-u)\mu_1(x-u)D_2(x)}{S_2(x-u)}\,du + \mu_2(x)S(x)$$
$$= D_2(x)\int_0^t \mu_1(x-u)S_1(x-u)\,du + \frac{D_2(x)S(x)}{S_2(x)}$$

because $S(x) = S_1(x)S_2(x)$ et $\mu_1(x-u) = -S_1'(x-u)/S_1(x-u)$. This results in:

$$D_2(x) = D_2(x)\int_0^t -S_1'(x-u)\,du + D_2(x)S_1(x)$$
$$= D_2(x)(S_1(x-t) - S_1(x)) + D_2(x)S_1(x) = D_2(x)S_1(x-t) \quad \text{if } t < x$$

and

$$= D_2(x)(1 - S_1(x)) + D_2(x)S_1(x) = D_2(x) \quad \text{if } t > x.$$

We get the survivors $s(x,t)$ at age x in t with the same kind of computation:

$$d(x,t) = \int_0^t \frac{S(x-u)\mu_1(x-u)D_2(x)}{S_2(x-u)}\,du + \mu_2(x)S(x)$$
$$= D_2(x)\int_0^t \mu_1(x-u)S_1(x-u)\,du + \frac{D_2(x)S(x)}{S_2(x)}$$

because $S(x) = S_1(x)S_2(x)$ et $\mu_1(x-u) = -S_1'(x-u)/S_1(x-u)$. This results in:

$$s(x,t) = S(x) + \int_0^t \frac{S(x-u)\mu_1(x-u)S_2(x)}{S_2(x-u)}\,du,$$
$$= S(x) + \int_0^t S_1(x-u)\mu_1(x-u)S_2(x)\,du,$$
$$= S(x) + \int_0^t S_1(x-u)\mu_1(x-u)S_2(x)\,du,$$
$$= S(x) + S_2(x)\int_0^t -S_1'(x-u)\,du,$$
$$= S(x) + S_2(x)(S_1(x-t) - S_1(x)) = S_2(x)S_1(x-t) \quad \text{if } t < x$$

and

$$= S(x) + S_2(x)(1 - S_1(x)) = S_2(x) \quad \text{if } t > x.$$

The mortality force $q(x,t) = d(x,t)/s(x,t)$ follows:

$$q(x,t) = \frac{D_2(x)S_1(x-t)}{S_2(x)S_1(x-t)} = \frac{D_2(x)}{S_2(x)} = \mu_2(x) \quad \text{if } t < x,$$

and

$$= \frac{D2(x)}{S2(x)} = \mu_2(x) \quad \text{if } t > x.$$

Therefore, for any $t > 0$, the force of mortality at age x is the force of mortality $\mu_2(x)$ of the final life table (the initial table from which the given cause was removed under the assumption of independence).

Appendix B: Demonstrating the strong properties of the two methods

Let (S, D, μ) be the life table of reference, $d(x,t)$ the density of deaths at age x in $t > 0$, $s(x,t)$ the survivors and $\lambda(x-u,u)$ the probability for a death foreseen in $t-u$ at age $x-u$ to be delayed until age t (the delay is u). In summing all deaths in t at age x by considering the end of the delays and those who experience no delay, we get the same results as in Appendix A:

$$d(x,t) = \int_0^t S(x-u)\lambda(x-u,u)\,du + \theta(x)S(x), \tag{1}$$

$$s(x,t) = S(x) + \int_0^t S(x-u)\left(\int_u^w (x-u,v)\,dv\right)du. \tag{2}$$

A third relation results from the fact that the sum of the probability of the different situations for the foreseen death is the overall force of mortality $\mu(x)$:

$$\int_0^w \lambda(x+u,u)\,du + \theta(x) = \mu(x). \tag{3}$$

As examples:

$$-\lambda(x,v) = (\mu(x) - \theta(x))\frac{D_2(x+v)}{S_2(x)},$$

when a cause of mortality is removed (S_2, D_2, μ_2) denotes the final life table).

$$-\lambda(x,v) = \mu(x)f(v) \tag{4}$$

when the delays v do not depend on age and have a probability distribution $f(v)$.

We will demonstrate that the removal method is the only decomposable method that keeps the force of mortality unchanged after $t = 0$ and in this case gives $\theta(x) = \mu_2(x)$.

Let use define:

$$\varphi(x) = \frac{d(x,t)}{s(x,t)}.$$

The derivative in t of the left part of this equation needs to be 0 for any $t > 0$. Using the formulae (I) and (II), this can be written as:

$$\frac{\partial d(x,t)}{\partial t} s(x,t) = d(x,t) \frac{\partial s(x,t)}{\partial t}.$$

$$S(x-t)\lambda(x-t,t)s(x,t) = d(x,t)S(t-x)\left(\int_t^w (x-t,v)\,dv\right),$$

so:

$$\lambda(x-t,t)s(x,t) = d(x,t)\left(\int_t^w \lambda(x-t,v)\,dv\right),$$

and:

$$\lambda(x-t,t) = \varphi(x)\left(\int_t^w \lambda(x-t,v)\,dv\right). \tag{4a}$$

If we put the expression $\lambda(x-t,t)$ in the formula (I) giving the deaths, we get:

$$\begin{aligned} d(x,t) &= \varphi(x)\int_0^t S(x-u)\left(\int_u^w \lambda(x-u,v)\,dv\right)du + \theta(x)S(x), \\ &= \varphi(x)(S(x,t) - S(x)) + \theta(x)S(x) \end{aligned}$$

according to (2)

$$= d(x,t) + S(x)(\theta(x) - \varphi(x)),$$

thus:

$$\varphi(x) = \theta(x),$$

from what:

$$\lambda(x-t,t) = \theta(x)\left(\int_t^w \lambda(x-t,v)\,dv\right). \tag{4b}$$

Now take the decomposability assumption for $\lambda(x-t,t)$:

$$\lambda(x-t,t) = A(x-t)B(x).$$

Putting it in (4b), it becomes:

$$A(x-t)B(x) = \theta(x)\int_t^w A(x-t)B(x+v-t)\mathrm{d}v.$$
$$B(x) = \theta(x)\int_t^w B(x+v-t)\,dv.$$
$$B(x) = \theta(x)\int_x^w B(u)\,du.$$

This formula is that of a life table, and more precisely, the final life table (S_2, μ_2, D_2), already encountered when looking at the removal of a cause of mortality. Effectively:

$$B(x) = D_2(x); \quad S_2(x) = \int_x^w B(u)\,du = \int_x^w B_2(u)\,du; \quad \mu_2(x) = \theta(x).$$

There remains to determine the possible values for $A(x-t)$. To that end, we need the equation (3):

$$\int_0^w \lambda(x+u,u)\,du + \theta(x) = \mu(x),$$

which becomes:

$$\int_0^w D_2(x+u)A(x)\,du + \mu_2(x) = \mu(x).$$
$$A(x)\int_x^w D_2(v)\,dv = \mu(x) - \mu_2(x).$$
$$A(x)S_2(x) = \mu(x) - \mu_2(x),$$
$$A(x) = \frac{\mu(x) - \mu_2(x)}{S_2(x)}.$$

Knowing that $\mu_1(x) = \mu(x) - \mu_2(x)$: $A(x) = \mu_1(x)/S_2(x)$, we can now give the expression $\lambda(x-t,t)$:

$$\lambda(x-t,t) = A(x-t)B(x),$$
$$= \frac{D_2(x)\mu_1(x-t)}{S_2(x-t)}.$$

This is exactly the expression obtained when a cause of mortality described by the table (S_1, μ_1, D_1) is removed in $t = 0$. The method of removal, therefore, is the only decomposable model for which the forces of mortality at any age

x and any time $t > 0$ catch up with their value in the actual longitudinal life table and does not depend on the time passed since the removal of the cause of mortality.

When the delays are independent of age, $\lambda(x-t,t)$ can be written as:

$$\lambda(x-t,t) = \mu(x-t)g(t).$$

Putting this expression in the equation (4b) for the equality of the cross-sectional and longitudinal life tables after the change in $t = 0$, we get:

$$\mu(x-t)g(t) = \varphi(x)\left(\int_t^w \mu(x-t)g(v)\,dv\right),$$
$$g(t) = \varphi(x)\int_t^w g(v)\,dv.$$

It imposes $\varphi(x) = k$ constant and $g(t) = Ce^{-kt}$. Substituting in equations (I) and (II) for deaths $d(x,t)$ and the survivors $s(x,t)$, we arrive at a contradiction for their ratio that depends on t:

$$\frac{d(x,t)}{s(x,t)} = k\frac{s(x,t) - S(x)}{s(x,t)}.$$

Appendix C: Fixed and variable delays

First, let us go back to the evaluation of deaths and forces of mortality from the value of the delays $g(\theta)$ taken at θ, the time of their beginning (and not $f(t)$ taken at their end). Let θ be the time at the departure of the delay that ends at t. We get: $t = \theta + g(\theta) = \theta + f(t)$. Let us take a small interval $\Delta\theta$ after θ. The delay beginning in $\theta + \Delta\theta$ will end in $t1 = \theta + \Delta\theta + g(\theta + \Delta\theta)$ which is equivalent to $\theta + \Delta\theta + g(\theta) + g'(\theta)\Delta\theta$. The interval Δt between t and t_1 is as follows:

$$\Delta t = (1 + g'(\theta))\Delta\theta.$$

At the end time in t, the density of delayed deaths is therefore at the ratio $1/(1 + g'(\theta))$ with the density of the deaths delayed at the departure between θ and $\theta + \Delta\theta$. Consider now the increase of the delay at the point of arrival of the delay in t: between t and t_1 , the delay $f(t)$ grows by $g'(\theta)\Delta\theta$. Its derivative is:

$$f'(t) = g'(\theta)\frac{\Delta\theta}{\Delta t} = \frac{g'(\theta)\Delta\theta}{1 + g'(\theta)}; \quad \Delta\theta = \frac{g'(\theta)}{1 + g'(\theta)}$$

.

This gives the formula for the deaths in case of delay:

$$d(x,t) = (1 - f'(t))D(x - f(t)) = \frac{1}{1 + g'(\theta)} D(x - g(\theta)),$$

because

$$1 - f'(t) = 1 - \frac{g'(\theta)}{(1 + g'(\theta))} = \frac{1}{1 + g'(\theta)}.$$

It is necessary to have the formula at the departure of the delay and not only at its arrival if we want to write the case where delay u varies according to the law of probability $k(u)\mathrm{d}u$. Let us demonstrate in these circumstances that the proportionality rule no longer holds. In consequence, the delay method necessarily rests on the particular hypothesis of a fixed delay. There is at a time t only one value for the delay for any individual threatened by death. We can begin the demonstration with the simple case of a sudden and unique change in $t = 0$. The deaths at age x and time t will be:

$$d(x,t) = \int_{\Xi}^{\infty} k(u)D(x - u)\, du, \tag{5}$$

and the survivors $s(x,t)$:

$$s(x,t) = S(x) + \int_0^t G(u)D(x - u)\, du, \tag{6}$$

where:

$$K(u) = \int_u^w k(v)\, dv. \tag{7}$$

If the proportionality property holds, it is necessary that:

$$d(x,t) = p(t)D(x - l(t)). \tag{8}$$

Integrating in the whole age range the two formulae of the deaths (6) and (8) leads to:

$$\int_0^t k(u)\, du \int_{-\infty}^w D(x - u)\, dx = p(t) \int_{-\infty}^w D(x - l(t))\, dx,$$

according to (7):

$$1 - K(t) = p(t).$$

Under these conditions, we would get:

$$D(x - l(t)) = \int_0^t \frac{k(u)}{1 - K(t)} D(x - u)\, du.$$

With $(k(u)/(1-K(t)))$ being a probability distribution on the interval of time $0, t$, the distribution of deaths should be at each age a weighted average of itself with a constant shift (playing the role of the delay). This is impossible *a priori* and *a posteriori* because the distributions of delays u, $(k(u))$, and of deaths x, $(D(x))$, have no relationship.

If the proportionality property is lost in the case of a unique and sudden change, when a fixed duration of the delay is replaced by a distribution of durations, it is also the case, *a fortiori*, when the change is continuous through time. In making the assumption of proportionality as a way of defining delays, Bongaarts and Feeney are postulating a very strong structure and a questionable one, because it means that all those who had to die in $t - f(t)$ in absence of delay would have the same duration $f(t)$ of the delay.

Tempo effect on age-specific death rates *

Shiro Horiuchi

CUNY Institute for Demographic Research, and School of Health Science, Hunter College of the City University of New York, 425 East 25th Street, New York, NY 10010-2590, USA. E-mail: Shiro.Horiushi@hunter.cuny.edu

Summary. It is widely known that shifts of cohort fertility schedule can produce misleading trends in period TFR. This note shows that such a "tempo bias" can occur in age-specific mortality as well: if the age distribution of cohort deaths shifts toward older (younger) ages, the period age-specific death rate is biased downward (upward).

1 Introduction

Relationships between "quantum" and "tempo" of demographic behavior are crucial for understanding population dynamics, in particular, discrepancies between demographic profiles of periods and cohorts. In this note, tempo measures are defined as indicators of the location and shape of the age curve of the given demographic behavior. Thus the first and higher moments of the age curve are tempo measures. Quantum measures are based on the area under the age curve, either over the entire life span or for a finite age range. For example, the number of deaths is a function of age, the mean and variance of age at death are tempo measures of the age curve, and the total number of deaths and the crude death rate are quantum measures.

Changes in tempo and quantum of demographic behavior among cohorts and over periods can produce trends that are misleading, apparently inconsistent, or difficult to interpret. Such trends may be considered biased or distorted, even though the concept of the true value is not always clear. It is widely known that shifts of cohort fertility schedule can produce misleading trends in period TFR (Ryder 1956).

Bongaarts and Feeney (2002, in this volume p. 11) argue that tempo biases occur in mortality as well. Using an artificial example, Feeney (2003, Figure 4) has demonstrated that cohort changes in the death distribution within an age interval can distort the period death rate for the age interval. The

example is essentially a straightforward conversion of their previous fertility example (Bongaarts and Feeney 1998, Figure 2) from birth to death. It has been developed for a special case that all deaths occur only at one point in the age range and the point shifts linearly among cohorts.

The purpose of this note is to show that tempo effects can operate in mortality, using a more general assumption about the shape and shift of death distribution than Feeney's hypothetical example. Sections 2 and 3 give a mathematical proof that if the age distribution of cohort deaths within an age interval shifts toward older (younger) ages, the period number of deaths in the age interval and, in turn, the age-specific death rate are biased downward (upward). Section 2 discusses main points of the proof in an intuitive and visually oriented way and Section 3 presents the inference in a formal manner. In addition, two hypothetical illustrations of mortality tempo effect by Bongaarts and Feeney are examined in Appendix, with focus on their implications for age-specific survival ratios.

2 Intuitive visual explanation

Two notions, which are familiar to demographers, are essential to the proof. The first is the split of Lexis square into two triangles. Figure 1 shows a Lexis diagram for the age interval between x and $x+1$ over the time period from $t-1$ to $t+2$. Time-age coordinates of six important points in Figure 1 are as follows: $A(t, x+1)$, $B(t+1, x+1)$, $C(t+2, x+1)$, $D(t-1, x)$, $E(t, x)$ and $F(t+1, x)$. We compare the number of deaths in the square ABFE (the estimation period), that in the parallelogram ABED (the earlier cohort) and that in BCFE (the later cohort). If both the number and the distribution of deaths in the age interval are identical for the two cohorts and age-specific deaths are evenly distributed over time within each cohort, the square ABFE also has the same number of deaths as each parallelogram has.

Suppose that the number of deaths that occur between x and $x+1$ is identical for the two cohorts, but the distribution of those deaths within the age interval is older in the later cohort. Then, at relatively young ages between x and $x+1$, more deaths occur in the earlier cohort than in the later cohort; but at relatively older ages in the range, more deaths occur in the later cohort than in the earlier cohort. Therefore, more deaths occur in the triangle AED than in BFE, and more deaths occur in the triangle BCF than in ABE. Because the square ABFE can be split into two triangles BFE and ABE, both of which have fewer deaths than their corresponding triangles have, the number of deaths in ABFE is smaller than that in ABED and that in BCFE. Because usually the number of person-years does not differ significantly among ABFE, ABED and BCFE, this leads to a paradoxical result that the age-specific death rate for the period is lower than that for either one of the two cohorts that pass through the age interval during the period.

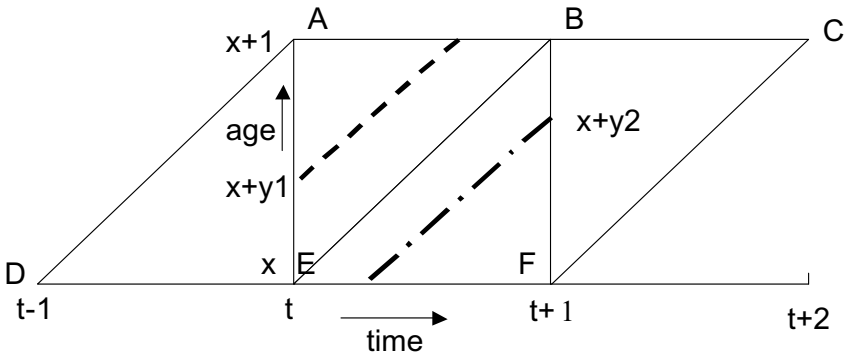


Fig. 1. Lexis Diagram for one period (ABFE) and two cohorts (ABED and BCFE)

The second main point of the proof is the definition of "shift of age distribution of deaths toward older ages (or more briefly, aging of death distribution) within a given age range." This issue is essential when the continuous cohort (instead of two discrete cohorts) is considered. There are possibly at least several different definitions of the concept, including those based on central tendency measures (e.g., rise in the mean age at death). In this note, the shift is defined as an overall rise of survival curve, as illustrated by the three curves in Figure 2. If the age distribution of deaths in population A is older than that in population B, then for any age (excluding the both ends of the age range), the proportion of all deaths above the age is greater in A than in B, and equivalently, the proportion of all deaths below the age is smaller in A than in B.

This may seem to be a strong condition, because the inequality has to hold at any age. However, to my knowledge, in any of widely used model life table systems, survival curves within the system do not cross over with each other, as illustrated in Figure 3. This means that in the model life system, the age distribution of deaths over the entire life span shifts toward older ages in the manner defined above.

Figure 2 shows survival curves for three cohorts over the one-year age range from x to $x + 1$. It can be viewed as a part (for example, the small rectangle on the highest curve) of Figure 3, which covers the entire life span. Thus the survival curves in Figure 2 are for only those who died in the age interval, excluding all those who died outside the interval. It is assumed that all of the three cohorts have the same number of survivors at age x and the same number of deaths between x and $x+1$, but different death distributions within the one-year age range.

Now, for further discussion, the definition of "cohort" needs to be changed from discrete (the two parallelograms in Figure 1) to continuous (infinitely many 45-degree diagonal lines in the parallelogram ACFD). Let the cohort aged x at t (line EB) be called the mid-cohort, which splits the rest into

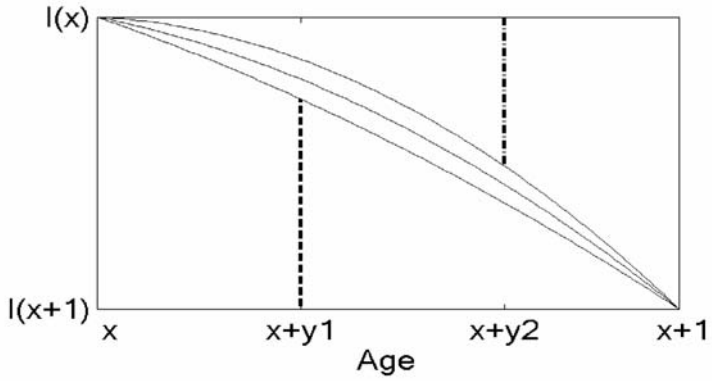


Fig. 2. Survival curves for those who died within the one-year age interval.

earlier cohorts and later cohorts. Suppose that the number of deaths[2] in the age interval is same for all cohorts, but the age distribution of deaths shifts toward older ages as defined above. Then the three survival curves in Figure 2, from high to low, can be considered to represent mortality experiences of a later cohort, the mid-cohort, and an earlier cohort in Figure 1. Obviously, for any age $x + y$ in the age range (y is between 0 and 1), the number of deaths above age $x + y$(corresponding to $l(x + y) - l(x + 1)$, the dashed line in Figure 2) in an earlier cohort is lower than that in the mid-cohort, and the number of deaths below age $x + y$ (corresponding to $l(x) - l(x + y)$, the dash-dot line in Figure 2) in an later cohort is lower than that in the mid-cohort.

Figure 1 indicates, however, that for an earlier cohort, deaths above a certain age occur during the period from t to $t + 1$ (e.g., on the dashed line in Figure 1), and for a later cohort, deaths below a certain age occur in the period (e.g., on the dash-dot line in Figure 1). (Note that the vertical dashed (dash-dot) line at age $x + y1$ (age $x + y2$) in Figure 2 corresponds to the number of deaths occurred on the diagonal dashed (dash-dot) line in Figure 1.) Thus, for any cohort of the both earlier and later groups, the number of deaths that occur between t and $t + 1$ is smaller than the number of deaths that would occur to the cohort during the period if the cohort has the same death distribution as that of the mid-cohort. This means that if the death distribution shifts toward old ages, the total number of deaths in ABFE is smaller than the total number of deaths in ABFE that would occur if the

[2] It is more accurate to call this "the single-year cohort equivalent of the density of death" rather than just "the number of deaths," but for simplicity, this lengthy expression is not used in this note.

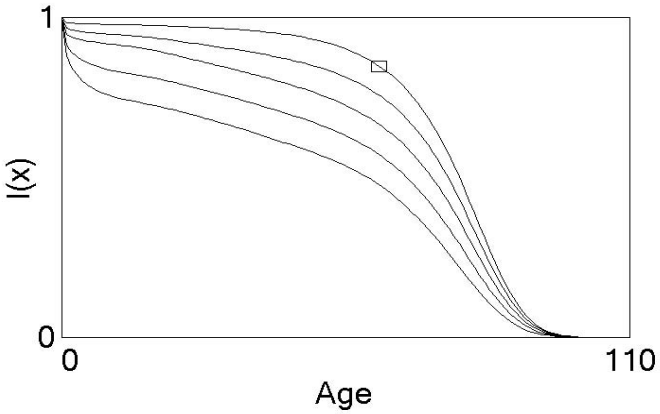


Fig. 3. Survival curves over the human life span.

death distribution remains same as that of the mid-cohort (or actually that of any cohort because the number of deaths for each cohort was set to be equal).

Therefore, a cohort shift of death distribution toward older ages seems to downwardly bias the age-specific number of period deaths. In the next section, this intuitive explanation is presented in a more formal manner.

3 Mathematical presentation

We use the regular continuous-variable Lexis framework. Let $d(x,t)$ be the number (density) of deaths at age x and time t, and let $d_c(x,u)$ be the number of deaths at age x for the cohort born at time u:

$$d_c(x,u) = d(x,u+x). \tag{1}$$

The cumulative death function from age x to $x+y$ is given by

$$F(x,y,t) = \int_0^y d(x+z,t)\,dz \quad \text{for time } t \tag{2}$$

and

$$F_c(x,y,u) = \int_0^y d(x+z,u)\,dz \quad \text{for cohort born at time } u \tag{3}$$

We consider a Lexis square for the age interval between x and $x+1$ and the time period from t and $t+1$ (ABFE in Figure 1). The number (density) of deaths that occur in the square is:

$$D(x,1,t,1) = \int_t^{t+1} \int_x^{x+1} d(y,u)\, dy\, du \tag{4}$$

Now, it is assumed that the cumulated death function from age x and $x+1$ is constant for all cohorts:

$$F_c(x,1,u) = g \tag{5}$$

for any u between $t-x-1$ and $t-x+1$. This assumption is needed in order to examine effects of cohort changes in the age distribution of deaths, independently of effects of cohort changes in the number of deaths. Obviously, if the age distribution of age at death remain constant among cohorts, i.e., if

$$F_c(x,y,u_1) = F_c(x,y,u_2) \tag{6}$$

for any y between 0 and 1 and any $u1$ and $u2$ between $t-x-1$ and $t-x+1$, then the total number of deaths in the Lexis square is

$$D(x,1,t,1) = g. \tag{7}$$

Suppose that the distribution of age at death within the age interval shifts toward older ages among cohorts. As described earlier, this means, by definition,

$$F_c(x,y,u_1) < F_c(x,y,u_2) \quad \text{if } u_1 > u_2 \tag{8}$$

for any y between 0 and 1 (excluding 0 and 1) and any $u1$ and $u2$ between $t-x-1$ and $t-x+1$.

Inequality (8) concerns deaths below age x+y. As for deaths above age x+y, we have

$$g - F_c(x,y,u_1) < g - F_c\,(x,y,u_2) \quad \text{if } u_1 < u_2. \tag{9}$$

Cohorts that pass through the Lexis square were born between $t-x-1$ and $t-x+1$ and reached age x between $t-1$ and $t+1$. Let the cohort born at $t-x$ be called the mid-cohort. It follows from (8) and (9) that for a cohort born after the mid-cohort, i.e. for $u > t-x$

$$F_c(x,y,u) < F_c(x,y,t-x), \tag{10}$$

and for a cohort born before the mid-cohort, i.e., for $u < t - x$,

$$g - F_c(x, y, u) < g - F_c(x, y, t - x). \tag{11}$$

By separating deaths during the period into deaths to cohorts born before and after the mid-cohort and using (10) and (11), the total number of deaths in the Lexis square is given by

$$\begin{aligned} D(x, 1, t, 1) &= \int_t^{t+1} \int_x^{x+1} d(y, u)\, dy\, du \\ &= \int_0^1 \int_{1-u}^1 d_c(x + y, t - x - 1 + u)\, dy\, du \\ &\quad + \int_0^1 \int_0^{1-u} d_c(x + y, t - x + u)\, dy\, du \\ &= \int_0^1 \{g - F_c(x, 1 - u, t - x - 1 + u)\}\, du \\ &\quad + \int_0^1 F_c(x, 1 - u, t - x + u)\, du \\ &< \int_0^1 \{g - F_c(x, 1 - u, t - x)\}\, du + \int_0^1 F_c(x, 1 - u, t - x)\, du \\ &= g. \end{aligned} \tag{12}$$

4 Discussion

As indicated above, if the number of deaths in an age range remains constant among cohorts but the death distribution within the age interval shifts toward older ages, the number of deaths in the age range for the estimation period is smaller than the corresponding number of cohort deaths. Similarly, a cohort shift of death distribution toward younger ages makes the number of period deaths higher than the corresponding number of cohort deaths.

The proof was given for the age-specific number of deaths, but essentially the same effect on the age-specific death rate is expected, because the relative effect on the number of person-years (the denominator of age-specific death rate) is smaller than the effect on the number of deaths (the numerator) (Feeney 2003). This is mainly because the shift does not significantly change the number of person-years of those who do not die in the age interval. In most one-year age intervals, a vast majority of persons survive through the interval.

In addition, the number of person-years for the period is likely to be very close to the number of person-years that would be obtained if the death distributions of all cohorts are identical to that of the mid-cohort, because losses in ABE and gains in BFE cancel each other to some extent.[3] Thus, it can be concluded that a shift of death distribution toward older (younger) ages is likely to bias the age-specific death rate downward (upward).

A few points about the assumptions adopted in this analytical study may be noteworthy. The age-specific number of deaths was assumed constant among cohorts. Admittedly, this is not realistic for two reasons. First, when the age-specific death rate changes, usually both the number and distribution of deaths within the age range change. When the overall mortality level declines, the number of deaths tends to increase above the modal age of adult deaths and decrease below it, shifting the mode to the right. Second, when the distribution of deaths moves toward older or younger ages, the shift occurs over a wide age range, thereby changing the number of deaths in each age group. Furthermore, in practice, the tempo effect will be numerically small if the distributional change is restricted to a narrow age range.

The purpose of this note, however, is not to produce a realistic and comprehensive picture of mortality change. Probably there are different pathways through which mortality changes bias period measures, and this investigation is an attempt to clarify the logical mechanism of one of those pathways. Thus the cohort number of deaths was assumed constant in order to investigate effects of cohort changes in the distribution of deaths independently of other effects that may confound the analysis.

Concerning the shift of age distribution, this analytical study is less restrictive than some previous studies of tempo effects, in which linear parallel shifts of the age curves were assumed (Ryder 1956, Inaba 1986, Bongaarts and Feeney 1998, Feeney 2003). The assumed pattern of shift in this study allows changes to occur in both the location and shape of distribution.

[3] If l(x) is same for all of the cohorts, then for any y between 0 and 1, l(x+y) of an earlier cohort, which passes through ABE, is smaller that of the mid-cohort. Similarly, l(x+y) of a later cohort, which passes through BFE, is larger that of the mid-cohort.

Acknowledgments

This research was supported by grants R01-AG14698 and R01-AG11552 from the National Institute on Aging, U.S.A.

References

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. (1998). On the quantum and tempo of fertility. *Population and Development Review*, 24(2):271–291.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Feeney, G. (2003). Mortality tempo: a guide for the skeptic. unpublished.

Inaba, H. (1986). On the relationship between the period total fertility rate and cohort fertility. *Journal of Population Problems*, 178:48–53. In Japanese.

Ryder, N. (1956). Problems of trend determination during a transition in fertility. *Milbank Memorial Fund Quarterly*, 34.

Appendix

An implication of transition between two stationary age distributions for age-specific death rates

This appendix examines two artificial examples of mortality tempo effects presented by Bongaarts and Feeney (2002: Figure 3; in this volume p. 11: Figure 5) and discusses an implication for age-specific death rates of the population dynamics assumed in the examples. In both of the examples, the hypothetical population shifts from a stationary age distribution to another stationary distribution through a one-year transition period. Thus there are three different periods (first stationary period, transition period, and second stationary period), and the number of births remains unchanged throughout these three periods. The mortality level in the second stationary period is slightly lower than that in the first stationary period.

It seems reasonable to expect that the mortality level for the transition period falls between those for the two stationary periods. However, the hypothetical computations show that the total number of annual deaths and the crude death rate for the transition period are substantially lower and the life expectancy at birth is considerably higher than those for either stationary period. For example, in one of the hypothetical illustrations, the life expectancy rises suddenly from 70.0 years in the first stationary period to about 73 in the transition period, and then falls to 70.25 in the second stationary period. This anomalous trend was interpreted to show the tendency for the life expectancy to be distorted when the mortality pattern is changing.

However, it is important to note that these examples were produced under the special scenario of shift between two stationary age distributions. In the hypothetical populations, tempo effects of mortality change seem to be confounded with effects on mortality trend of this particular type of population dynamics. This appendix will explain why the special scenario leads to the anomalous mortality trend.

Suppose that a population is stationary before time T and after time $T+1$ and the age distribution shifts between T and $T+1$. The number of individuals in the age interval between x and $x+1$ at time t is given by

$$N(x,t) = N_1(x) \quad \text{if } t \leq T \quad \text{and} \quad N(x,t) = N_2(x) \quad \text{if } t \geq T+1 \qquad \text{(A.1)}$$

where $N_1(x)$ and $N_2(x)$ are the number of individuals in the age interval between x and $x+1$ during the first stationary period and that during the second stationary period, respectively.

It is assumed that the number of births remains constant and the force of mortality at any age is lower in the second stationary period than in the first stationary period. Then

$$N_1(x) < N_2(x) \quad \text{for any } x > 0 \tag{A.2}$$

if x is not greater the highest age of the second stationary population.

The age-specific survival ratio from the age interval between x and $x+1$ to the next age interval between $x+1$ and $x+2$ is $N_1(x+1)/N_1(x)$ for the first stationary period, $N_2(x+1)/N_2(x)$ for the second stationary period, and $N_2(x+1)/N_1(x)$ for the one-year transition period. The survival ratio for the transition period is higher than that for the first stationary period because of the inequality of the numerator, i.e., $N_2(x+1) > N_1(x+1)$. It is higher than that for the second stationary period as well, because of the inequality of the denominator, i.e., $N_1(x) < N_2(x)$.

The above results can be generalized to any length u of transition period by considering the survival ratio from the age interval between x and $x+1$ to the age interval between $x+u$ and $x+u+1$, as far as $x+u$ is under the highest age of the population. Obviously, high age-specific survival ratios imply low age-specific death rates. Thus it can be claimed that if the population shifts between two stationary age distributions and the mortality level in the later stationary period is lower (higher) than that in the earlier stationary period, then age-specific death rates in the transition period tend to be lower (higher) than those in either stationary period.

This anomalous mortality trend is due to the very special type of age structure change, i.e., shift from a stationary population to another. Suppose that the mortality pattern remains constant for a while, then changes in a short period of time, and remains constant again thereafter. Usually, it will take many years for the population to eventually become stationary. (The number of births is assumed unchanged in this population.) However, the two simulations adopt an unusual scenario that the population becomes stationary immediately after some mortality change. Therefore, the high life expectancy during the transition period in the artificial examples may be attributable mainly to this unusual scenario, i.e., shift between two stationary age distributions. It does not seem to be a typical tempo bias.

Mortality tempo-adjustment: Theoretical considerations and an empirical application*

Marc Luy

University of Rostock, Institute for Sociology and Demography Ulmenstrasse 69, 18057 Rostock, Germany. E-Mail: email@marc-luy.de

Summary. The number of scholars following the tempo approach in fertility continues to grow, whereas tempo-adjustment in mortality generally still is rejected. This rejection is irrational in principle, as the basic idea behind the tempo approach is independent of the kind of demographic event. Providing the first empirical application to a substantial problem, this chapter shows that mortality tempo-adjustment can paint a different picture of current mortality conditions compared to conventional life expectancy. An application of the Bongaarts and Feeney method to the analysis of mortality differences between western and eastern Germany shows that the eastern German disadvantages still are considerably higher and that the mortality gap between the two entities began to narrow some years later than trends in conventional life expectancy suggest. Thus, the picture drawn by tempo-adjusted life expectancy fits the expected trends of changing mortality and also the self-reported health conditions of eastern and western Germans better than that painted by conventional life expectancy.

1 Introduction

One of the main goals of quantitative demography is the derivation of period measures with a clear and distinct meaning to analyze demographic developments in time as well as current demographic conditions in different populations. Since more than a century demographers have been assuming to know how to provide correct calculations and interpretations of period measures, such as the total fertility rate (TFR) or life expectancy at birth (e_0). Both are summary measures and have the purpose to represent current fertility and mortality conditions respectively, standardized for the given age composition of populations driving the number of observed events and thus the values of crude rates.

* Great part of this chapter is reprinted from "Mortality tempo-adjustment: An empirical application" by Marc Luy, Demographic Research Vol. 15, 2006.

In a series of studies, Bongaarts and Feeney (1998, 2002, in this volume p. 11 and p. 29) recently have claimed that summary measures such as these should not only be standardized for age but also for tempo effects that arise whenever demographic conditions are changing. In their most recent study, Bongaarts and Feeney (in this volume p. 29) define a tempo distortion as an "inflation or deflation of a period quantum or tempo indicator of a life-cycle event, such as birth, marriage, or death, resulting from a rise or fall in the mean age at which the event occurs". Introducing the idea with corresponding formulae for tempo-adjustment, Bongaarts and Feeney stirred the world of demographers and divided their community into tempo supporters and tempo opponents. Despite existing critics (e.g., Van Imhoff and Keilman, 2000; Kim and Schoen, 2000; Van Imhoff, 2001; Smallwood, 2002; Schoen, 2004; Keilman, 2006), the number of scholars following the tempo approach in fertility continues to grow (see e.g., Lesthaeghe and Willems, 1999; Kohler and Philipov, 2001; Philipov and Kohler, 2001; Zeng Yi and Land, 2001, 2002; Goldstein et al., 2003; Sobotka, 2003, 2004a, 2004b; Winkler-Dworak and Engelhardt, 2004)[2]. However, Bongaarts and Feeney's successive work on mortality tempo effects still is generally rejected (see Guillot, 2003b, in this volume; Le Bras, in this volume; Wachter, in this volume; Wilmoth, 2005; Rodríguez, in this volume).

The rejection is irrational in principle, as the basic idea behind the tempo approach is independent of the kind of demographic event[3]. The idea of adjusting period life expectancy for tempo effects is as follows. When deaths are postponed to increasingly later ages, the number of deaths occurring in a given period is thinning out. For example, if every death in a given year were to be postponed by six months, there would be only half as many deaths observed in that year as one would have expected if there had been no postponement at all. Because death rates would decline at all ages, life expectancy would in-

[2] Winkler-Dworak and Engelhardt (2004) applied the tempo approach to the analysis of first marriage. From a methodological point of view, their work belongs to the papers dealing with tempo effects in fertility since the demographic logic behind the analysis of first marriages is identical to the analysis of first births.

[3] Bongaarts and Feeney's paper on mortality tempo is based on a paper published by them earlier and entitled "On the quantum and tempo of fertility". Following the idea presented in that paper, the most common critcisms of the mortality tempo approach holds that the quantum of mortality is necessarily one, and that life expectancy itself is a pure tempo measure and thus cannot be adjusted for tempo effects (Guillot, 2003b; Wachter, in this volume; Wilmoth, 2005; Rodríguez, in this volume). Note, however, that tempo effects are not necessarily connected to a quantum. Tempo generally affects period rates and the quantum is impacted by tempo effects only if the period rates are used to estimate the demographic quantum, such as is done with the TFR. When period rates are used to derive any other demographic measures, then these measures are affected by tempo distortions. It does not matter whether they contain a quantum component or not, as is the case in period life expectancy.

crease by a larger amount, many times the half a year that was actually added to the length of life in that period. Tempo-adjustment produces a period measure of longevity that changes only by the amount of lifetime by which deaths were postponed, and as such is a potentially useful tool for demographers[4]. It has already been shown that in a given situation of mortality decline, i.e. the mean age at death increases, life expectancy calculated from age-specific death rates during the period of changing mortality conditions is higher than life expectancy calculated from age-specific death rates under stationary mortality conditions at the end of this transition (Bongaarts and Feeney, 2002, in this volume p. 29; Feeney, 2003; Horiuchi, in this volume). An illuminating paper about the consequences of such biases on the interpretation of period data was written by Vaupel (2002), who called for a distinction between "life expectancy at current rates" and "life expectancy at current conditions"[5].

It seems that tempo effects impact current period measures for mortality significantly, as they do with fertility measures. In the actual discussion on mortality tempo, this aspect is given no consideration: the published papers solely deal with theoretical and technical questions, while empirical applications are used exclusively to compare different measures of period mortality conditions and to demonstrate their properties against the background of historical mortality trends (besides the Bongaarts and Feeney papers mentioned above, see e.g. Vaupel, 2002, in this volume p. 93; Feeney, 2003, in this volume; Guillot, 2003b, in this volume; Bongaarts, in this volume; Le Bras, in this volume; Wachter, in this volume; Wilmoth, 2005; Goldstein, in this volume; Rodríguez, in this volume). Empirical applications to a substantive problem of mortality differentials are, however, missing so far. An interesting aspect - although not explicitly mentioned by the authors - of the initial mortality tempo paper of Bongaarts and Feeney (2002) is that the variance in life expectancy between the US, Sweden, Japan, and France decreases from 3.4 years according to conventional life expectancy to only 1.7 years according to tempo-adjusted life expectancy. Applying the Bongaarts and Feeney method to mortality differences between eastern and western Germany, I will show that adjusting period life expectancy for tempo effects paints a different picture of mortality trends and of differences between these two regions than conventional tempo-unadjusted calculations. I will conclude that the results of tempo-adjusted life expectancy provides a better fit to the expected trends

[4] The author thanks an anonymous referee of this paper for his or her suggestion to include this example in order to describe the basic idea of mortality tempo-adjustment.

[5] In the paper mentioned, Vaupel (2002) regarded the distortions inherent in the current mortality rates as a consequence of the changed timing of death resulting from the effects of heterogeneity rather than from the effects of mortality tempo. Regardless of the different views on the origin of distortions in period mortality rates, Vaupel's message applies universally to all kinds of demographic period measures.

of changing mortality, and also to self-reported health conditions of eastern and western Germans.

First, however, I will demonstrate how tempo effects impact period life tables and why they should be seen as distortions.

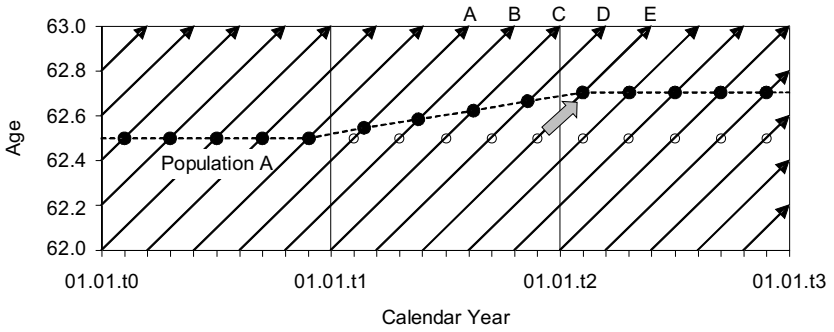
2 How mortality tempo affects period life expectancy

Inspired by an example in an unpublished paper of Feeney (2003) with the apt title "Mortality tempo: a guide for the skeptic", I use a simple illustration to demonstrate the idea of tempo effects in mortality. Consider a population A in which all births occur intermittently at intervals of 0.2 years and in which all deaths taking place during some base year occur at exactly the midpoint of a single year of age. Suppose that, at the end of the base year, age at death within a certain age group begins to increase linearly at the rate of 0.2 years per year for all persons, and cedes increasing at the end of the year. The Lexis diagram in Figure 1a shows this scenario for age 62 as an example. The life lines of each cohort are represented by an arrow moving through time and age. In base year t0, all deaths at age 62 happen exactly at age 62.5. During year t1, the age at death increases linearly with the given annual rate, from 62.5 to 62.7. The latter level is reached in year t2 and remains constant from then on. Assume further that the annual numbers of births in the population have been constant and that the proportion of deaths at a certain age is constant over all cohorts (meaning unchanged mortality conditions until base year t0). The two assumptions imply that each dot in Figure 1a represents the same number of deaths and that each arrow represents the same number of persons surviving until age 62.5. Let us assume that 20,000 individuals of each cohort reach age 62 and that 1,000 of them die at this age. Thus, according to the old mortality conditions until year t0 there are 5,000 annual deaths at age 62. The age-specific death rate for age 62 in year t0 is then given by 5,000 deaths divided by 97,500 risk years lived:

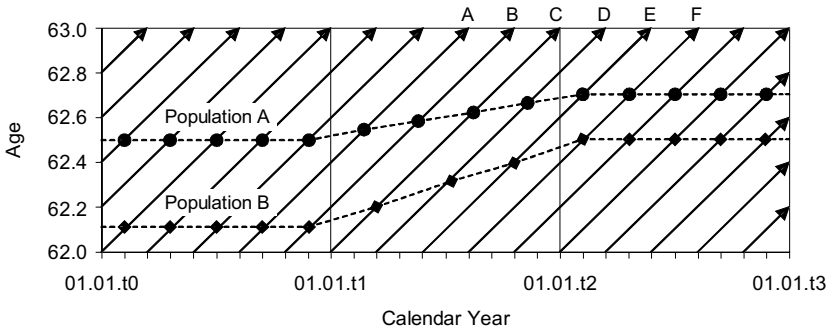
$$M^{A}_{62,t0} = \frac{5,000}{97,500} = 0.05128\,.$$

The number of risk years lived can easily be derived. If all individuals survived until age 63, the number of risk years lived was 100,000 since each individual lived exactly 1 person year at age 62. Since the 5,000 deceased individuals live only 2,500 person years at age 62, the total number of risk years lived reduces to 97,500.

Now it is important to see what happens with the number of deaths in year t1, the year of changing mortality. The five cohorts in t1 reaching age 62.5, the exact age at which those who do not survive the given age group die according to the old conditions, are marked with the letters A to E. Thus, cohort A is the oldest cohort reaching age 62.5 in year t1 and cohort E is the youngest. Due to assumed changes in mortality conditions during year t1, the



(1a) a population with decreasing mortality in year t1



(1b) two populations with differently decreasing mortality in year t1

Fig. 1. Mortality tempo effect illustrated in the Lexis diagram.

age at death of cohorts A to E increases steadily and cohort E is the first to reach the new age at death level of 62.7 years. Since each of the five cohorts lives longer than the preceding one, the intervals between the deaths are longer than they are between the births (both intervals are identical before the year of changing mortality conditions t1). Consequently, the deaths to the five cohorts that reach age 62.5 during year t1 are spread over a period exceeding one year. As a result, the deaths of persons belonging to cohort E are shifted to year t2, shown by the thick grey arrow in Figure 1a. Following Vaupel (in this volume p. 93), the shift can also be interpreted as "saved lives" in year t1. The number of deaths in year t1 declines by 1,000 when compared to the scenario before the mortality conditions changed. This is demonstrated in Figure 1a: Only four black dots are seen in year t1. Had mortality not changed during that year, there would have been five dots in year t1, as demonstrated by the unfilled dots representing age at death to cohorts according to the old mortality conditions until year t0. Due to the reduced number of deaths and

the risk years gained in year t1, the age-specific death rate for age 62 decreases to:

$$M^{A}_{62,t1} = \frac{4,000}{98,400} = 0.04065\,.$$

The mortality decline is reflected in the decreasing age-specific death rate. If such mortality change occurred similarly at the other ages, this would lead to an increase in life expectancy that is derived from the age-specific death rates. Figure 1a shows that the decline in the annual number of deaths is transitory in that it disappears when the age at death stops rising. From year t2 on, the intervals between births and deaths are, again, identical and there are 5,000 deaths in each subsequent calendar year, leading to the age-specific death rate:

$$M^{A}_{62,t2} = \frac{5,000}{98,500} = 0.05076\,.$$

Since the number of deaths in the numerator increases relatively steeper than the number of risk years lived in the denominator, the period death rate for year t2 will be higher than that for year t1, although the average age at death is higher in t2 than it is in t1. As a consequence, life expectancy would decrease between t1 and t2. Since period life expectancy is generally seen as an indicator for period mortality conditions, such a decrease is likely to be interpreted as an increase in mortality. However, Figure 1a shows that no cohort experiences any increase in mortality. In Bongaarts and Feeney's approach, such a discrepancy between the development of mortality conditions and its representation in period death rates (or measures derived from them, such as life expectancy) represents a tempo effect. The logic behind this argument is neither limited to the simple assumptions of this example (constant number of births, birth intervals of 0.2 years) nor is it restricted to one single age group. If we increased the number of age groups and assumed that the ages at death in these groups rise at different rates, then different numbers of deaths would be shifted and the magnitude of the tempo effect would vary from one age group to another (for more details, see Feeney, 2003).

When the trend in period life expectancy is used in order to analyze the changing mortality conditions of a specific population, such tempo effects may not be a problem as long as there is no sudden change or stagnation of mortality. However, tempo effects may lead to a distorted picture when mortality conditions of two populations are compared that experience mortality changes that differ in their patterns of change. This is demonstrated by introducing an additional population B to the illustration in Figure 1b. As with population A, all births in population B occur intermittently at intervals of 0.2 years, mortality remains unchanged until year t0 and at the end of year t0, the age at death begins to increase linearly and cedes increasing at the end of the year. Compared to population A, the cohorts of population B show lower ages at death at any time. Until year t0 all deaths at age 62 occur at the exact age

of 62.1 and the change during year t1 leads to an increased age at death of 62.5 years, thus being equivalent to the mortality conditions of population A until year t0. Using the same logic of calculating the age-specific death rates for population B, it follows for the years t0, t1, and t2 that:

$$M^B_{62,t0} = \frac{5,000}{95,500} = 0.05236\,,$$
$$M^B_{62,t1} = \frac{3,000}{97,900} = 0.03064\,,$$
$$M^B_{62,t2} = \frac{5,000}{97,500} = 0.05128\,.$$

Although no cohort of population B experiences lower mortality than the cohorts of population A, the death rate for population B in year t1 is lower than the death rate for population A. This is due to the fact that during year t1 mortality changes in population B are stronger than they are in population A. The higher increase of the age at death in population B leads to higher tempo distortions in t1 compared to population A.

Despite the confusing outcome, the examples represented in Figures 1a and 1b show that there is no mistake in the calculation of period death rates. There might be research questions where the conventional period rates are interesting, e.g. to analyze and forecast the annual number of deaths. However, the examples also show that the conventional way of calculating period measures may convey the wrong message whenever demographic conditions are changing in the year or period analyzed. As to life expectancy, one can make generalizations as already described by Bongaarts and Feeney (2002): If mortality declines, then life expectancy overestimates current conditions; if mortality rises, then life expectancy underestimates current survival conditions. The bias is the more marked, the more intensive the changes are during the period observed.

All demographic period measures are hypothetical estimates to standardize for current demographic conditions. Since different populations have different experiences of changes in the mean age at death, tempo effects impact them differently, as do different age compositions of the populations. Thus, tempo effects should generally be seen and treated as a distortion of period measures, as the effects of population age composition. Bearing in mind the simple examples above, the question arises as to the meaning that period measures based on current rates have in a world of continuous demographic change. This holds especially when populations with entirely different demographic developments are compared, such as the populations of West and East Germany. To assure that conventional period measures do not point into the wrong direction, it is necessary to look at tempo-adjusted measures regardless of whether fertility, mortality, or any other demographic process is analyzed.

3 Why life expectancy differences between western and eastern Germany call for tempo-adjustment

The demographic changes and developments in eastern and western Germany are generally seen to present a unique opportunity to understand the interaction between societal, social, and economic conditions on the one hand, and population processes on the other. The German experience thus is used to understand the reasons behind recent mortality changes. The two pre-war German regions were characterized by a demographic composition and behavior that was almost identical until 1945, followed however by 45 years under different political and socioeconomic structures and resulting in demographic developments that were entirely different (Dinkel, 1992, 1994, 1999; Gjonça et al., 2000). With unification in 1990, East Germany adopted the western societal and economic system, causing sudden changes in all of its demographic processes. These conditions - leading some scholars to describe the eastern German population as a kind of "natural experiment" (Dinkel, 1999; Vaupel et al., 2003) - generated a large number of studies on the changes in eastern German demography. Of central interest in the field of mortality research has been the rapid convergence of life expectancy since 1990 following roughly two decades of continuous divergence. The former widening and the subsequent closing in the life expectancy gap between western and eastern Germany were mainly caused by the age groups 60-80, leading to the central message that "it's never too late" to increase one's length of life (Vaupel et al., 2003).

Figure 2 shows the trends in period life expectancy at birth e_0, using standard life table techniques for western and eastern German women and men for each single calendar year from 1950 to 2004. The life table calculations are based on official population statistics, i.e. data for the living population and deaths for each calendar year and single age groups (for a more detailed description of these data, see Luy, 2004a)[6].

Regarding mortality differences between western and eastern Germany, the time span presented can be subdivided into five central phases:

- The first phase, from 1950 to roughly 1960, is characterized by irregular fluctuations, with several years of mortality crossing over. These trends correspond with the waves of influenza that swept East and West Germany

[6] Because of a reform to Berlin's district borders in the year 2000 it has been impossible since then to divide the population and the demographic events of Berlin into eastern and western Berlin according to the former borders of separated Germany. Thus, from the year 2000 onwards, the Statistical Office of Germany divides population data into western Germany without Berlin, eastern Germany without Berlin, and Berlin. In order to get a complete time series of life tables for eastern Germany (former GDR) and western Germany (former FRG) from 1950 to 2004, I divided the data on Berlin's living population and deaths of the years 2000 to 2004 into east and west according to the sex-specific share of inhabitants of eastern and western Berlin of the year 1999, assuming identical mortality for both parts of the city from the year 2000 onwards.

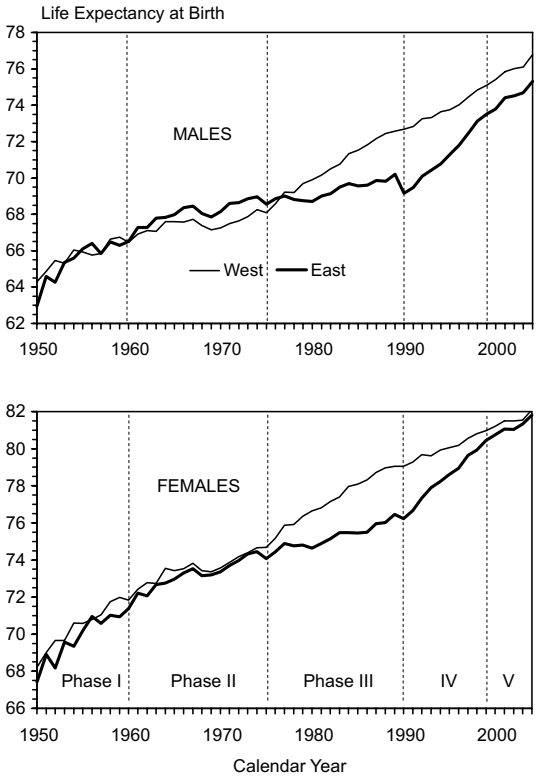


Fig. 2. Trends in conventional life expectancy at birth for western and eastern Germany, 1950-2004.

in different years (Luy, 2004a). No mortality differences can be detected between the two Germanys, neither for men nor for women.

- In the second phase, roughly covering the period 1960 to 1975, the developments in life expectancy in West and East Germany assumed more regularity, with mortality slightly higher among East German women and significantly lower among East German men. The differences in favor of East German males rose until the first half of the 1970s and reached a maximum of roughly one year in life expectancy at birth. However, the disadvantage of West German men arose mainly from different definitions of live birth in East and West Germany, thus causing lower infant mortality rates in the former GDR on statistical[7] grounds. An analysis of age-specific differences between West and East German mortality shows

[7] In West Germany, the result of childbirth is defined as live birth if one of three signs of life, namely heart-beat, natural respiration, or a pulsating umbilical cord, is evident. In East German statistics, live birth was defined only by heartbeat and simultaneous natural respiration (Müller, 1976). Consequently, all deaths of newborns showing only one of the three signs of life are registered as live births

that the higher life expectancy among East German men mainly (but not only) resulted from statistically lower infant mortality (Luy, 2004a).

- The third phase, starting in the middle of the 1970s, is characterized by the continuous divergence for both sexes in the development of mortality conditions in favor of West Germany. This development corresponds to the general divergence in mortality trends between all western and eastern European countries (e.g. Caselli and Egidi, 1980; Bourgois-Pichat, 1985; Bobak and Marmot, 1996a, 1996b; Hertzman et al., 1996; Meslé and Hertrich, 1997; Vallin and Meslé, 2001; Meslé and Vallin, 2002). Figure 1 shows that the widening of the life expectancy gap was caused by the fact that East German life expectancy at birth increased at a lower pace for both sexes, whereas life expectancy in West Germany rose more rapidly (Höhn and Pollard, 1991; Scholz, 1996; Gjonça et al., 2000; Nolte et al. 2000a). The differences peaked in 1988 for women (almost 3 years) and in 1990 for men (roughly 3.5 years).
- These peaks - virtually concurring with German unification - were followed by the continuous narrowing of the gap in west-east German mortality differences until the end of the 1990s, when the difference in e_0 reached about 0.5 years for women and about 1.6 years for men. As can be seen in Figure 2, as the two Germanys entered this phase the differences in life expectancy trends between them started to reverse compared to the trends in the third phase. The convergence of mortality levels now observable is due to the fact that since the beginning of the 1990s life expectancy has been rising much faster in eastern Germany than in the west. Based on these observations, German demographers assumed that the west-east mortality gap will fully close during the first two decades of the 21st century, as reflected in one of the most recent population forecasts of the Statistical Office of Germany (Statistisches Bundesamt, 2003).
- However, around the year 2000, this trend changed again. The differences in life expectancy between eastern and western German men now stagnate on a level around 1.5 years. Eastern German women, by contrast, seem to further approximate the western German level, however with a decline in the pace of approximation and with the difference now being around 0.25 years.

Figure 2 shows a striking decrease in life expectancy among eastern German men in 1990, a phenomenon described as the eastern German "mortality crisis" (Dorbritz and Gärtner, 1995; Riphan, 1999; Nolte et al., 2000a, 2000b) and as characteristic of a "demographic shock" in connection with the changes in eastern Germany resulting from unification (Eberstadt, 1994). However, long-term trends in life expectancy question the aptness of this description and call for an explanation of the rapid closing of the gap. The decisive question is: Which factor or which factors are responsible for the trend reversal in

and thus as infant deaths only in West Germany, whereas in East Germany such cases were registered as stillbirths and did not enter mortality statistics.

mortality differences between western and eastern Germany, a trend reversal that has occurred within one or two years only? The factors discussed most in search of an answer are the same that are assumed to be responsible for the general mortality gap between western and eastern European countries (e.g. Bobak and Marmot, 1996a, 1996b; Hertzman et al., 1996; for the discussion on the mortality differences between eastern and western Germany, see Luy, 2004a): East German working conditions, environmental conditions, the consequences of uranium mining and storage, the effects of the ongoing immigration of a more healthy foreign population to West Germany, selective internal east-west migration, psychological reactions to the political suppression, economic conditions, medical technology, lifestyles, and cardiovascular risk factors.

The similarity to the general European west-east divergence makes the search for the reasons behind the mortality trends in eastern Germany a subject of major interest that reaches beyond the borders of Germany. Additionally, it seems that finding the main cause(s) for the mortality differences between western and eastern Germany will be an important step forward in gaining a deeper understanding of general mortality differentials. A large and continuously increasing number of studies follow this path based on trends in life expectancy such as shown in Figure 2 (e.g. Chruscz, 1992; Dinkel, 1994, 1999; Schott et al., 1994; Becker and Boyle, 1997; Gjonça et al., 2000; Bucher, 2002; Nolte et al., 2002; Luy, 2004a, 2004b, 2005b; Mai, 2004). Although many researchers are working on this subject, the rapid approximation of life expectancy is still not explained in full.

However, following Bongaarts and Feeney's tempo approach, we must conclude that period life expectancy based on annual age-specific death rates is an imperfect solution for the reflection of period mortality conditions. As has been shown in the previous chapter, this is because death rates are biased downward with rising mean age at death (mortality decline) and they are biased upward when the mean age at death declines (mortality increase). Since different populations experience changes in the mean age at death with different paces, tempo effects impact them differently. Phases of mortality decline and phases of mortality increase set in in eastern and western Germany in different years and with different pace, coinciding with observed trends in life expectancy differences between the two parts of Germany: during Phase 3, life expectancy increased continuously in West Germany, whereas it rose only slightly or remained constant in the east. During Phase 4, life expectancy rose more steeply in eastern Germany than in the west. We can assume that the sudden improvements in eastern Germany after unification, for instance in economic conditions and medical technology, caused postponement of deaths in almost all age groups to an extent that was not possible in western Germany, where these conditions have already been on a high level. Similarly, for the years preceding unification we can expect that more deaths among western German women and men have been postponed as a consequence of increasing advantages in living conditions and medical standards. If these different

trends are causing tempo distortions in the sense of Bongaarts and Feeney's approach, then the studies on the causes of eastern German excess mortality are based on data leading to a distorted picture of mortality conditions in the two German regions and thus to the differences between them. In the final consequence, this may be the reason why the factor(s) mainly responsible for the impressive improvement of mortality conditions in eastern Germany is (are) still undetected.

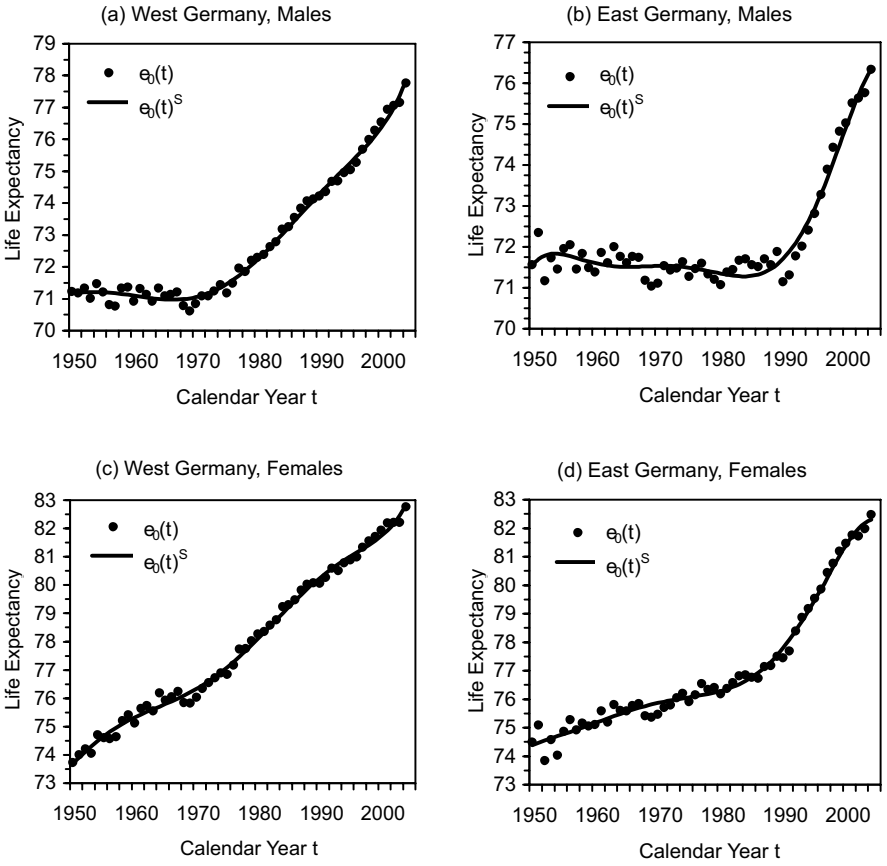


Fig. 3. Conventional life expectancy at birth $e_0(t)$ and smoothed estimates $e_0(t)^S$ (sixth degree polynomial), western and eastern Germany, 1950-2004 (no mortality under age 30).

In the following, tempo-adjusted life expectancy, denoted by $e_0^*(t)$, for western and eastern Germany, is estimated by using the indirect technique proposed by Bongaarts and Feeney (2002) in their initial paper on tempo

effects in mortality, assuming no mortality under age 30. Figure 3 shows the smoothed trends in conventional life expectancy at birth with mortality below age 30 set to 0 for each of the four populations observed (i.e., $e_0(t) = e_{30}(t) + 30$). These functions form the base for the method used to calculate tempo-adjusted life expectancy $e_0^*(t)$ in eastern and western Germany. The formula used for tempo-adjusted life expectancy is

$$e_0^*(t) = e_0(t) + \frac{1}{b} \ln \left(1 - \frac{de_0^*(t)}{dt} \right),$$

with b denoting the "Gompertz parameter" estimated by fitting a Gompertz model to annual age-specific death rates. The methods of estimation and the calculations done for western and eastern Germany are provided in the Appendix.

4 Trends in tempo-adjusted life expectancy in western and eastern Germany

Figure 4 shows the trends in conventional and tempo-adjusted life expectancy at birth (no mortality under age 30 in both cases) from 1975 to 2004 for western and eastern German females and males. The graph for western German females (Figure 4c) is very similar to the figures for US and Japanese women presented by Bongaarts and Feeney (2002: 24). As can be seen in Figure 3(c), western German females represent the only of the four populations analyzed with an observed life expectancy at birth that has been increasing almost constantly since 1950. Thus, the tempo distortion $S(t)$ (defined as the difference between conventional and tempo-adjusted life expectancy) is relatively constant among western German females during the observation period. Since improvements in life expectancy developed later (western and eastern German males) or at a changing pace (eastern German females) among the other three populations, tempo distortions must vary when compared to western German females. This is well reflected by the results gained for $e_0^*(t)$ and $S(t)$, as can be seen in Figure 4.

In all cases, the estimated tempo distortions agree with the logic of mortality tempo effects. This becomes clear when comparing Figures 3 and 4. The tempo distortion $S(t)$ was very low among West German men in 1975 and then increased steadily until the second half of the 1980s, when the difference between conventional and tempo-adjusted life expectancy reached an almost constant level (Figure 4a). It can be seen in Figure 3(a) that life expectancy among West German men remained more or less unchanged between 1950 and 1970, thus one cannot expect a noticeable tempo distortion in the mid 1970s. Rising life expectancy after 1970 is caused a by shift in the average age at death and thus the tempo-adjusted life expectancy also starts to increase,

although at a lower pace than does conventional life expectancy. Among eastern German males, life expectancy remained constant or even declined slightly until the end of the 1980s and then started to rise at a higher pace than in any phase of life expectancy trends in western Germany (Figures 3a and 3b). Consequently, tempo-adjusted life expectancy $e_0^*(t)$ did not differ from conventional life expectancy until the beginning of the 1990s and then began to increase at a considerably lower rate compared to $e_0(t)$. The extent of tempo distortions in conventional life expectancy grew during the observed period among eastern German females, too. From Figure 3(d) we know that their life expectancy rose in the period preceding unification, although it did so at a lower pace than among their West German counterparts (see also Figure 2). As a result, tempo distortions, i.e. the difference between tempo-adjusted and conventional life expectancy, remained at an almost constant level between 1975 and 1990. However, the difference between $e_0(t)$ and $e_0^*(t)$ started to increase at the end of the 1980s when conventional life expectancy rose at a higher rate - a phenomenon similar to what has been observed among men in the eastern part of Germany (Figures 4b and 4d).

The most important question is the way in which the differences in life expectancy between western and eastern Germany developed in the observation period 1975 to 2004 when adjusted for tempo distortions. The corresponding results are given in Figures 5 and 6 for males and females respectively; the single values can be found in Table 1. The thinner lines in the two graphs represent the absolute difference between western and eastern Germany in conventional life expectancy and the bold lines show the difference in tempo-adjusted life expectancy. Figure 5 again depicts the rapid decrease in conventional life expectancy differences after 1990, following a continuous increase since the beginning of the observation period. Whereas West German males enjoyed a higher life expectancy according to conventional calculation methods since 1976, East German men showed a higher tempo-adjusted life expectancy until 1981. Note that the different definitions of live birth do not affect the results presented in this section; thus there seems to be a real East German mortality advantage among men in the 1970s. Only after 1981 did the differences in tempo-adjusted life expectancy switch to an advantage for West German males, although much less so than the results based on conventional life expectancy. The graph demonstrates that the trend in increasing west-east differences occurred at a lower pace once life expectancy is adjusted for tempo effects. In 1990, when the difference in conventional life expectancy between West and East German men reached a peak of 3.08 years, the difference in tempo-adjusted life expectancy was only 1.07 years, i.e. two years less. The finding that the latter differences did not decrease since unification but continued to increase until the end of the 1990s is even more interesting. While the difference in conventional life expectancy between western and eastern German males declined to roughly one and a half years in 2004, those in tempo-adjusted life expectancy are now even higher, with a difference of about 1.6 years. Only at the end of the 1990s did the trend in increasing

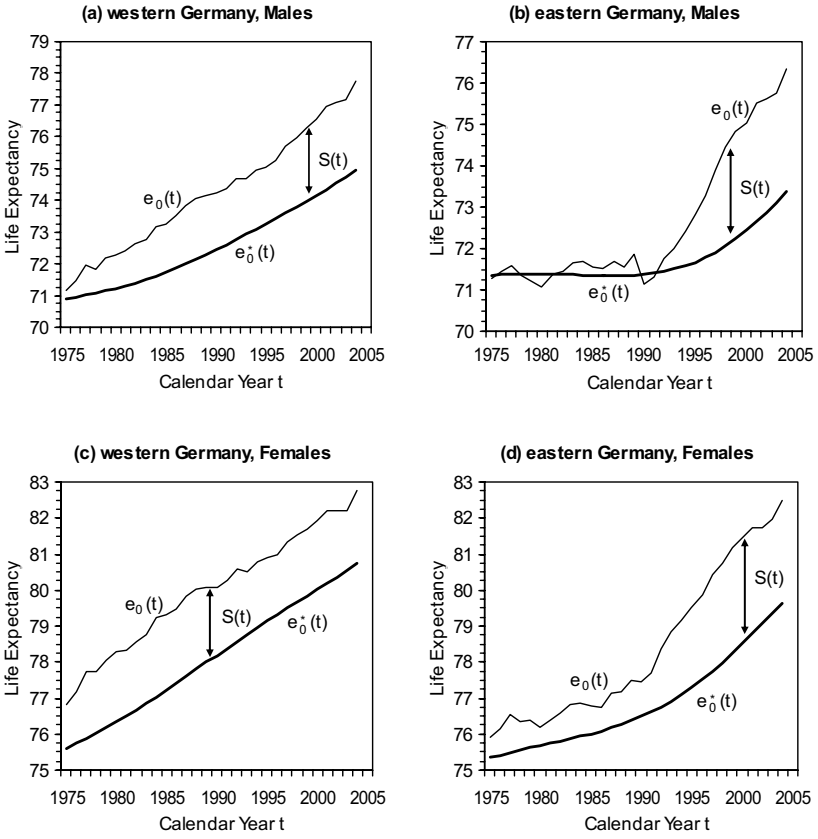


Fig. 4. Conventional life expectancy at birth $e_0(t)$ and estimated tempo-adjusted life expectancy at birth $e_0^*(t)$ with tempo distortion $S(t)$, western and eastern Germany, 1975-2004 (no mortality under age 30).

differences in tempo-adjusted life expectancy lower in speed, pointing to slow convergence solely in the last years of the observation period.

The results for the west-east German differences among females are similar to those just described for males. Until unification, the advantage in mortality conditions of West German females is lower when tempo-adjusted life expectancy is used instead of conventional life expectancy. Whereas the difference in conventional life expectancy increased to 2.85 years in 1988, those in tempo-adjusted life expectancy did never exceed 1.9 years. Similar to the situation among men, the differences in tempo-adjusted life expectancy between western and eastern German females did not decline with unifica-

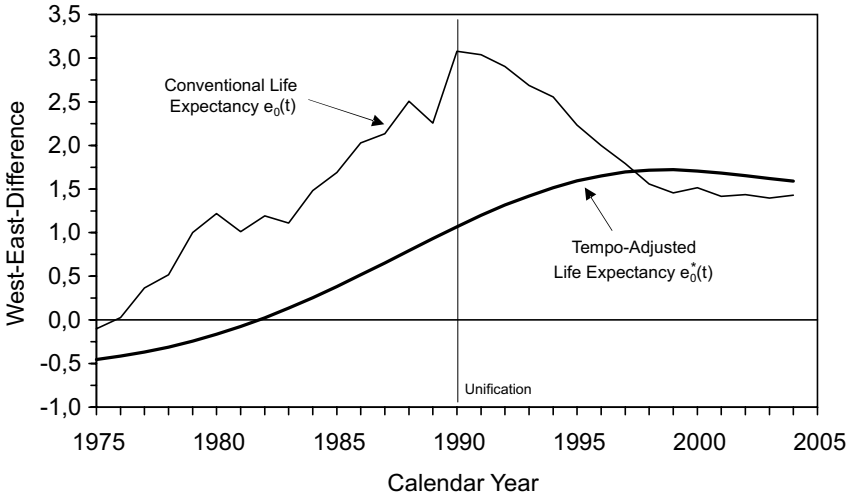


Fig. 5. West-east German difference in life expectancy at birth for conventional life expectancy $e_0(t)$ and tempo-adjusted life expectancy $e_0^*(t)$, males 1975-2004 (no mortality under age 30).

tion parallel to conventional life expectancy but instead rose until the mid 1990s. The trends in conventional and tempo-adjusted life expectancy crossed over between 1992 and 1993. From then on, the mortality advantage of western German females measured with tempo-adjusted life expectancy is higher when compared to the results based on tempo-unadjusted values. Although a decreasing trend in mortality differences between western and eastern German females is also evident with tempo-adjusted life expectancy since the mid 1990s, the remaining differences in favor of western German women are still considerably higher. While the disadvantage of eastern German women decreased to 0.28 years in the year 2004 according to conventional life expectancy, the tempo-adjusted difference still shows 1.1 years.

5 Discussion

The idea of mortality tempo effects is derived directly from the idea of fertility tempo effects; the latter have been known since more than half a century and they experience growing acceptance. Following similar approaches of Ryder (1956, 1964) or Ward and Butz (1980), Bongaarts and Feeney (1998) proposed a new method to estimate the tempo bias in period fertility rates and they provided a formula to adjust the TFR for these distortions. A few years later, Bongaarts and Feeney (2002) extended this approach to the analysis

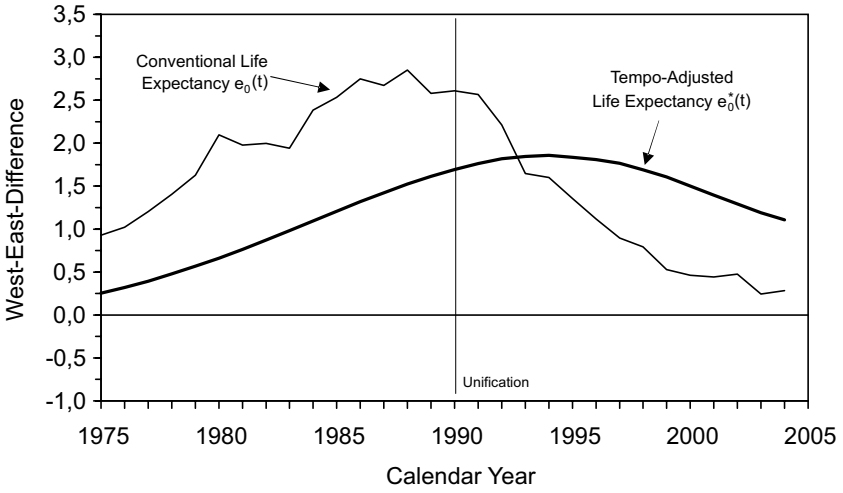


Fig. 6. West-east German difference in life expectancy at birth for conventional life expectancy $e_0(t)$ and tempo-adjusted life expectancy $e_0^*(t)$, females 1975-2004 (no mortality under age 30).

of mortality and claimed that tempo effects bias period life expectancy, too. In contrast to the situation in the field of fertility, the tempo approach in mortality generally still is rejected. However, if we accept the need for tempo-adjustment in the period TFR, we equally have to accept the need for this adjustment in period life expectancy. The basic idea of the TFR is to estimate the fertility quantum under current fertility conditions as a standardized indicator for current fertility conditions. Changes in the mean age at childbirth cause tempo effects which, in turn, affect age-specific fertility rates and thus the TFR that is based on them. The same holds for period life expectancy. The basic idea of life expectancy is to estimate the average length of life under current mortality conditions as a standardized indicator for current mortality conditions. Changes in the mean age at death are causing tempo effects, which affect age-specific death rates and thus life expectancy that is based on them.

There seems to be a misunderstanding in that Bongaarts and Feeney are assumed to have intended to estimate period measures with a certain cohort meaning. This is, however, not exactly so. As to their basic application of their method, they instead used cohort experiences in order to estimate current changes in age-specific mortality conditions. Thus, Bongaarts and Feeney's tempo approach has to be distinguished from methods in order to translate

Table 1. West-east German difference in life expectancy at birth according to conventional life expectancy $e_0(t)$ and tempo-adjusted life expectancy $e_0^*(t)$, 1975-2004 (no mortality under age 30).

Years before unification					Years after unification				
	Males		Females			Males		Females	
Year	$e_0(t)$	$e_0^*(t)$	$e_0(t)$	$e_0^*(t)$	Year	$e_0(t)$	$e_0^*(t)$	$e_0(t)$	$e_0^*(t)$
1975	-0.10	-0.45	0.93	0.25	1990	3.08	1.07	2.61	1.69
1976	0.03	-0.42	1.02	0.32	1991	3.04	1.20	2.57	1.76
1977	0.37	-0.37	1.20	0.39	1992	2.90	1.32	2.21	1.82
1978	0.52	-0.31	1.41	0.48	1993	2.69	1.42	1.65	1.85
1979	1.00	-0.24	1.63	0.57	1994	2.55	1.51	1.60	1.86
1980	1.22	-0.17	2.10	0.66	1995	2.23	1.60	1.36	1.84
1981	1.01	-0.08	1.98	0.76	1996	2.00	1.65	1.12	1.81
1982	1.19	0.02	2.00	0.87	1997	1.79	1.69	0.90	1.76
1983	1.11	0.14	1.94	0.98	1998	1.56	1.72	0.79	1.69
1984	1.48	0.25	2.38	1.10	1999	1.46	1.72	0.53	1.61
1985	1.69	0.38	2.53	1.21	2000	1.52	1.71	0.46	1.50
1986	2.03	0.52	2.75	1.32	2001	1.42	1.68	0.44	1.39
1987	2.13	0.65	2.67	1.42	2002	1.44	1.65	0.48	1.29
1988	2.51	0.79	2.85	1.52	2003	1.40	1.62	0.24	1.19
1989	2.25	0.93	2.58	1.61	2004	1.43	1.59	0.28	1.11

period information into cohort information[8]. The misunderstanding possibly originates from several sources. The first may be the title of their original paper "How long do we live?" since the term "we" does only make sense in the cohort perspective and does not exist in the logic of pure period measures. Another reason may be the similarity of Bongaarts and Feeney's tempo-adjusted life expectancy to other period measures that have clearly defined cohort components, such as the "cross-sectional average length of life" (CAL) introduced by Brouard (1986) and Guillot (2003a) or the "mean length of life" proposed by Sardon (1993, 1994).

In principal, the Bongaarts and Feeney adjustment formulae for the TFR and for life expectancy follow the same basic idea in that they assume that period effects influence all currently living cohorts identically. In the case of fertility, the tempo-adjustment formula is based on a shift of the age-specific fertility schedule; in the case of mortality, the original tempo-adjustment formula is based on a shift of the age-specific mortality schedule. However, since the TFR and life expectancy are fundamentally different in their structural

[8] Goldstein (in this volume) showed that in conditions of steady mortality change, tempo-adjusted life expectancy with the Bongaarts and Feeney formula can indeed be used as a measure of cohort life expectancy. However, since the formula contains no direct cohort component - as is shown in the Appendix of this chapter - the primary interpretation should be made in a period context.

designs, the adjustment formulae must include fundamental differences. The tempo-adjusted TFR depends only on age-specific fertility rates within a small neighborhood of the analyzed calendar year. This does not hold for the Bongaarts and Feeney formula for the tempo-adjusted life expectancy used in this chapter. The major difference to the fertility procedure is that the proposed adjustment method for life expectancy uses a series of previous period life tables. Consequently, it is evident that the Bongaarts and Feeney formula reflects past mortality conditions in a certain way. But in the logic of tempo distortions, this does not necessarily represent an inconsistency, especially when past changes in mortality conditions are steady and continuous, which approximately holds for adult ages in developed populations and in the last decades. Since these are the restrictions that Bongaarts and Feeney (2002) have made to the applicability of their tempo-adjustment formula for life expectancy, we should not see it as problematic that it leads to values close (but not exactly) to a weighted moving average of past period life expectancy, as Wachter (in this volume) has shown. Just the contrary, in restricting the application to the industrialized countries of the recent past, this property of the Bongaarts and Feeney formula is consistent with the theoretical idea of tempo distortions in life expectancy.

However, we cannot see the Bongaarts and Feeney formula providing a perfect measure for tempo-adjusted period mortality conditions. As already shown by several scholars, their formula is based on assumptions which cannot be met in full by reality (e.g. Goldstein, in this volume). Thus, we should see the Bongaarts and Feeney formula as an attempt to standardize for tempo effects in period life expectancy to obtain a better measure for comparing period mortality conditions. It is, however, not clear to which extent the Bongaarts and Feeney method catches factual tempo effects and it is not possible to assess whether it presents a maximum distortion in the sense that the truth lies somewhere between conventional and tempo-adjusted life expectancy, as discussed by Vaupel (in this volume p. 93). While the constant shape assumption turned out to be robust against moderate deviations (Feeney, in this volume), several scholars described specific characteristics of the Bongaarts and Feeney formula that under certain conditions may be partly inconsistent with the general idea of tempo-adjustment (e.g. Wachter, in this volume; Wilmoth, 2005; Guillot, in this volume). Nevertheless, it is important to separate these methodological aspects from the question of the general existence of tempo effects in period life expectancy in order to do justice to Bongaarts and Feeney's tempo approach.

The empirical results presented in this chapter are striking and may be important for the general understanding of several phenomena connected with changing mortality: once life expectancy is adjusted for tempo effects, the differences between western and eastern Germany do not decrease immediately after unification and ten years later they still are higher when compared to the differences in conventional life expectancy. Taking into consideration what we know about mortality trends and their driving factors, the following question

arises: Which of the two stories describes better what we can expect the mortality differences between western and eastern Germany to look like? We know that mortality differences such as these are caused by a number of different factors that cannot be separated empirically and that may work in opposite directions. This group of factors certainly includes some long-term effects on the mortality conditions of a population, such as the health consequences of environmental conditions or uranium mining and storage, or other long-term effects that are still working against life expectancy improvements in eastern Germany, such as of the continuing immigration of a more healthy foreign population to western Germany or selective internal east-west migration. But also the factors that are able to cause sudden changes in mortality conditions will possibly take at least a few years to display their maximum effects. For instance, qualitative improvements in medical technology cannot be reached within the short span of a year, and even if eastern German's lifestyles changed immediately after 1990, the impact on health and mortality possibly develops slowly and steadily. Consequently, following more than two decades of continuous divergence, it seems unlikely that mortality improvements in eastern Germany could reach beyond the improvements made in West Germany in the first year after unification. From this point of view, the picture drawn by tempo-adjusted life expectancy fits better than that painted by conventional life expectancy the expected trends and to the fact that the disadvantages in self-reported health - known to be a good predictor of mortality conditions - of eastern German women and men decreased only slightly during the 1990s (see Luy, 2004a, 2005b). Thus, tempo-adjusted life expectancy seems to be a more realistic indicator of the level and changes in current mortality conditions than conventional life expectancy.

These aspects indicate that the discussion on the reasons for the trends in mortality differences between western and eastern Germany of the last years might have been based on inappropriate measures and thus possibly have pointed into the wrong direction. It is puzzling that no factor was found that could explain the observed trends in conventional life expectancy at birth, despite the fact that several scholars have been conducting research on the subject. However, according to the trends in west-east German differences in tempo-adjusted life expectancy, the explanatory factors obviously do not necessarily narrow the gap in life expectancy by more than two and a half years among females and by more than 1.5 years among males within ten calendar years, and they do not necessarily change trends in mortality differences immediately after unification from one year to the next year. Research should rather focus on finding the factors responsible for producing immediate and continuous postponement of deaths that are causing these tempo distortions in life expectancy but do not necessarily increase the average length of life to the extent indicated by conventional life expectancy. Besides the standard of medical technology and economic conditions, one of these factors may be the availability of nursing care that shows a similar development in differences between western and eastern Germany as does conventional life expectancy in

the first years after unification (see Luy, 2004a, 2005b). Obviously, a comparable tempo-distorted picture is drawn by conventional life expectancy in the phase of rising differences prior to unification under changed conditions, with higher tempo distortions in period life expectancy among the West German population.

To sum up, the results of the empirical application of mortality tempo-adjustment presented in this chapter indicate that the extent and the trend of the differences in mortality conditions between western and eastern Germany are not what we thought they were. It is not surprising, then, that none of the explanatory variables usually stated to explain the west-east German mortality gap fit the observed mortality trends when measured by conventional life expectancy at birth. To come back to the central statement made by Vaupel et al. (2003) on the closing west-east mortality gap in Germany: it may never be too late to increase one's length of life, but changing mortality conditions seems to take longer than trends in conventional life expectancy suggest, and the reasons for such changes may be of different kind than generally expected.

Moreover, since life expectancy without adjustment for tempo effects is one of the demographic tools most used in order to analyze mortality, we may have to reconsider our knowledge on the basis of this measure in several other aspects:

- What about the opening and the recent closing of the mortality gap between women and men in the developed world?
- What about the linear increase in record life expectancy at birth, described by Oeppen and Vaupel (2002), especially regarding the impressive slope of this increase?
- What about the increasing mortality gap between eastern and western Europe?

This chapter has shown that tempo-adjustment of life expectancy might provide a different picture of current mortality conditions than does conventional life expectancy. We can expect that tempo effects distort the analysis in all cases where the compared populations experienced different trends in changing mortality. Consequently, we should not doubt the existence of tempo effects in period life expectancy and the distortions they possibly cause. As discussed above, it is the method proposed by Bongaarts and Feeney (2002) that has to be improved since it is based on a number of assumptions that will never be satisfied in full. Having accepted the existence of tempo effects, however, this method should be preferred to using tempo-unadjusted estimates for period life expectancy as long as there are no better solutions. Thus, the main goal of the future work of formal demographers should be the development of methods for tempo-adjusted life expectancy based on less restrictive assumptions that can be applied to all contemporary and past populations, as claimed similarly by Vaupel (in this volume p. 93) and Feeney (in this volume).

References

Becker, N. and Boyle, P. (1997). Decline in mortality from testicular cancer in West Germany after reunification. *The Lancet*, 350:744.

Bobak, M. and Marmot, M. (1996a). East-west mortality divide and its potential explanations: proposed research agenda. *British Medical Journal*, 312:421–425.

Bobak, M. and Marmot, M. (1996b). East-west health divide and potential explanations. In Hertzman, C., Kelly, S., and Bobak, M., editors, *East-west life expectancy gap in Europe. Environmental and non-environmental determinants*, pages 17–44. Dordrecht et al.: Kluwer.

Bongaarts, J. Five period measures of longevity. *In this volume, also published in Demographic Research*, 13:547–558. 2005.

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Science*, 100:13127–13133. 2003.

Bongaarts, J. and Feeney, G. The quantum and tempo of life-cycle events. *In this volume, also published in Vienna Yearbook of Population Research*, pages 115–152. 2006.

Bongaarts, J. and Feeney, G. (1998). On the quantum and tempo of fertility. *Population and Development Review*, 24:271–291.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28:13–29.

Bourgeois-Pichat, J. (1985). Recent changes in mortality in industrialized countries. In Vallin, J., Lopez, A. D., and Behm, H., editors, *Health policy, social policy and mortality prospects*, pages 507–539. Liege: Ordina Editions.

Brouard, N. (1982). Structure et dynamique des populations. La pyramide des années vivre, aspects nationaux et examples régionaux. *Espaces, Populations, Sociétés*, 2:157–168.

Bucher, H. (2002). Die Sterblichkeit in den Regionen der Bundesrepublik Deutschland und deren Ost-West-Lücke seit der Einigung. In Cromm, J. and Scholz, R. D., editors, *Regionale Sterblichkeit in Deutschland*, pages 33–38. Augsburg and Göttingen: WiSoMed, Cromm.

Caselli, G. and Egidi, V. (1980). New trends in European mortality. Strasbourg, Council of Europe.

Chruscz, D. (1992). Zur Entwicklung der Sterblichkeit im geeinten Deutschland: die kurze Dauer des Ost-West-Gefälles. *Informationen zur Raumentwicklung*, 9-10:691–700.

Dinkel, R. H. (1992). Kohortensterbetafeln für die Geburtsjahrgänge 1900 bis 1962 in den beiden Teilen Deutschlands. *Zeitschrift für Bevölkerungswissenschaft*, 18:95–116.

Dinkel, R. H. (1994). Die Sterblichkeitsentwicklung der Geburtsjahrgänge in den beiden deutschen Staaten. Ergebnisse und mögliche Erklärungshypothesen. In Imhof, A. E. and Weinknecht, R., editors, *Erfüllt*

leben - in Gelassenheit sterben: Geschichte und Gegenwart, pages 155–170. Berlin: Duncker & Humblot.

Dinkel, R. H. (1999). East and West German mortality before and after reunification. Unpublished manuscript. University of Rostock.

Dorbritz, J. and Gärtner, K. (1995). Bericht 1995 über die demographische Lage in Deutschland. *Zeitschrift für Bevölkerungswissenschaft*, 20:339–448.

Eberstadt, N. (1994). Demographic shocks after Communism: Eastern Germany, 1989-93. *Population and Development Review*, 20:137–152.

Feeney, G. Increments to life and mortality tempo. *In this volume, also published in Demographic Research*, 14:27–46. 2006.

Feeney, G. (2003). Mortality tempo: a guide for the sceptic. To be downloaded from http://www.gfeeney.com.

Gjonça, A., Brockmann, H., and Maier, H. (2000). Old-age mortality in Germany prior to and after Reunification. *Demographic Research*, 3(1).

Goldstein, J. R. Found in translation? A cohort perspective on tempo-adjusted life expectancy. *In this volume, also published in Demographic Research*, 14:71–84. 2006.

Goldstein, J. R., Lutz, W., and Scherbov, S. (2003). Long-term population decline in Europe: the relative importance of tempo effects and generational length. *Population and Development Review*, 29:699–707.

Guillot, M. Tempo effects in mortality: an appraisal. *In this volume, also published in Demographic Research*, 14:1–26. 2006.

Guillot, M. (2003a). The cross-sectional average length of life (CAL): a cross-sectional mortality measure that reflects the experience of cohorts. *Population Studies*, 57:41–54.

Guillot, M. (2003b). Does period life expectancy overestimate current survival? An analysis of tempo effects in mortality. Minneapolis, Minnesota, USA. Paper presented at the annual meeting of the PAA.

Hertzman, C., Kelly, S., and Bobak, M., editors (1996). *East-west life expectancy gap in Europe. Environmental and non-environmental determinants*. Dordrecht et al.: Kluwer.

Höhn, C. and Pollard, J. (1991). Mortality in the two Germanies in 1986 and trends 1976-1986. *European Journal of Population*, 7:1–28.

Horiuchi, S. Tempo effect on age-specific death rates. *In this volume, also published in Demographic Research*, 13:189–200. 2005.

Keilman, N. (2006). Demographic translation: from period to cohort perspective and back. In Caselli, G., Vallin, J., and Wunsch, G., editors, *Demography: analysis and synthesis*, pages 215–225. Amsterdam et al.: Elsevier.

Kim, Y. J. and Schoen, R. (2000). On the quantum and tempo of fertility: limits to the Bongaarts-Feeney adjustment. *Population and Development Review*, 26:554–559.

Kohler, H.-P. and Philipov, D. (2001). Variance effects in the Bongaarts-Feeney formula. *Demography*, 38:1–16.

Le Bras, H. Mortality tempo versus removing of deaths: opposite views leading to different estimations of life expectancy. *In this volume, also published in Demographic Research*, 13:615–640. 2005.

Lesthaeghe, R. and Willems, P. (1999). Is low fertility a temporary phenomenon in the European Union? *Population and Development Review*, 25:211–228.

Luy, M. (2004a). Mortality differences between Western and Eastern Germany before and after Reunification: a macro and micro level analysis of developments and responsible factors. *Genus*, 60:99–141.

Luy, M. (2004b). Verschiedene Aspekte der Sterblichkeitsentwicklung in Deutschland von 1950 bis 2000. *Zeitschrift für Bevölkerungswissenschaft*, 29:3–62.

Luy, M. (2005a). The importance of mortality tempo-adjustment: theoretical and empirical considerations. MPIDR Working Paper wp-2005-035. To be downloaded from http://www. demogr.mpg.de/papers/working/wp-2005-035.pdf.

Luy, M. (2005b). West-Ost-Unterschiede in der Sterblichkeit unter besonderer Berücksichtigung des Einflusses von Lebensstil und Lebensqualität. In Gärtner, K., Grünheid, E., and Luy, M., editors, *Lebensstile, Lebensphasen, Lebensqualität - Interdisziplinäre Analysen von Gesundheit und Sterblichkeit aus dem Lebenserwartungssurvey des BiB*, pages 333–364. Wiesbaden: VS-Verlag für Sozialwissenschaften.

Mai, R. (2004). Regionale Sterblichkeitsunterschiede in Ostdeutschland. Struktur, Entwicklung und die Ost-West-Lücke seit der Wiedervereinigung. In Scholz, R. and Flöthmann, J., editors, *Lebenserwartung und Mortalität, Materialien zur Bevölkerungswissenschaft*, volume 111, pages 51–68. Wiesbaden: BiB.

Meslé, F. and Hertrich, V. (1997). Mortality trends in Europe: the widening gap between east and west. In *23rd International Population Conference, Beijing 1997*, pages 479–508. Liège: IUSSP.

Meslé, F. and Vallin, J. (2002). Mortality in Europe: the divergence between east and west. *Population-E*, 57:157–198.

Müller, C. K. E. (1976). *Die Säuglingssterblichkeit in der Bundesrepublik Deutschland und in der Deutschen Demokratischen Republik*. Bonn: Schwarzbold.

Nolte, E., Scholz, R., Shkolnikov, V., and McKee, M. (2002). The contribution of medical care to changing life expectancy in Germany and Poland. *Social Science & Medicine*, 55:1905–1921.

Nolte, E., Shkolnikov, V., and McKee, M. (2000a). Changing mortality patterns in East and West Germany and Poland: I. Long-term trends. *Journal of Epidemiology and Community Health*, 54:890–899.

Nolte, E., Shkolnikov, V., and McKee, M. (2000b). Changing mortality patterns in East and West Germany and Poland: II. Short-term trends during transition and in the 1990s. *Journal of Epidemiology and Community Health*, 54:899–906.

Oeppen, J. and Vaupel, J. W. (2002). Broken limits to life expectancy. *Science*, 296:1029–1031.

Philipov, D. and Kohler, H.-P. (2001). Tempo effects in the fertility decline in Eastern Europe: evidence from Bulgaria, the Czech Republic, Hungary, Poland and Russia. *European Journal of Population*, 17:37–60.

Riphahn, R. T. (1999). Die Mortalitätskrise in Ostdeutschland und ihre Reflektion in der Todesursachenstatistik. *Zeitschrift für Bevölkerungswissenschaft*, 24:329–363.

Rodríguez, G. Demographic translation and tempo effects: an accelerated failure time perspective. *In this volume, also published in Demographic Research*, 14:85–110. 2006.

Ryder, N. B. (1956). Problems of trend determination during a transition in fertility. *Milbank Memorial Fund Quarterly*, 34:5–21.

Ryder, N. B. (1964). The process of demographic translation. *Demography*, 1:74–82.

Sardon, J.-P. (1993). Un indicateur conjoncturel de mortalité: l'exemple de la France. *Population (French Edition)*, 48:347–368.

Sardon, J.-P. (1994). A period measure of mortality: the example of France. *Population: An English Selection*, 6:131–150.

Schoen, R. (2004). Timing effects and the interpretation of period fertility. *Demography*, 41:801–819.

Scholz, R. D. (1996). Analyse und Prognose der Mortalitätsentwicklung in den alten und neuen Bundesländern - Ergebnisse des Ost/West-Vergleiches der Kohortensterblichkeit. In Dinkel, R. H., Höhn, C., and Scholz, R. D., editors, *Sterblichkeitsentwicklung - unter besonderer Berücksichtigung des Kohortenansatzes*, pages 89–102. München: Boldt.

Schott, J., Wiesner, G., Casper, W., and Bergmann, K. E. (1994). Entwicklung der Mortalität des alten Menschen in Ost- und Westdeutschland in den zurückliegenden Jahrzehnten. In Imhof, A. E. and Weinknecht, R., editors, *Erfüllt leben - in Gelassenheit sterben: Geschichte und Gegenwart*, pages 171–182. Berlin: Duncker & Humblot.

Smallwood, S. (2002). The effects of changes in timing of childbearing on measuring fertility in England and Wales. *Population Trends*, 109:36–45.

Sobotka, T. (2003). Tempo-quantum and period-cohort interplay in fertility changes in Europe. Evidence from the Czech Republic, Italy, the Netherlands and Sweden. *Demographic Research*, 8:152–214.

Sobotka, T. (2004a). Is lowest-low fertility in Europe explained by the postponement of childbearing? *Population and Development Review*, 30:195–220.

Sobotka, T. (2004b). *Postponement of childbearing and low fertility in Europe*. Amsterdam: Dutch University Press.

Statistisches Bundesamt (2003). *Bevölkerung Deutschlands bis 2050. 10. koordinierte Bevölkerungsvorausberechnung*. Wiesbaden: Statistisches Bundesamt.

Vallin, J. and Meslé, F. (2001). Trends in mortality in Europe since 1950: age-, sex- and cause-specific mortality. In *Trends in mortality and differential mortality*, pages 31–186. Strasbourg: Council of Europe Publishing. (Population Studies No. 36).

Van Imhoff, E. (2001). On the impossibility of inferring cohort fertility measures from period fertility measures. *Demographic Research*, 5:23–64.

Van Imhoff, E. and Keilman, N. (2000). On the quantum and tempo of fertility: comment. *Population and Development Review*, 26:549–553.

Vaupel, J. W. Lifesaving, lifetimes and lifetables. *In this volume, also published in Demographic Research*, 13:597–614. 2005.

Vaupel, J. W. (2002). Life expectancy at current rates vs. current conditions: a reflexion stimulated by Bongaarts and Feeney's 'How Long Do We Live?'. *Demographic Research*, 7:365–377.

Vaupel, J. W., Carey, J. R., and Christensen, K. (2003). It's never too late. *Science*, 301:1679–1681.

Wachter, K. Tempo and its tribulations. *In this volume, also published in Demographic Research*, 13:201–222. 2005.

Ward, M. P. and Butz, W. P. (1980). Completed fertility and its timing. *Journal of Political Economy*, 88:915–940.

Wilmoth, J. R. (2005). On the relationship between period and cohort mortality. *Demographic Research*, 13:231–280.

Winkler-Dworak, M. and Engelhardt, H. (2004). On the quantum and tempo of first marriages in Austria, Germany, and Switzerland: Changes in mean age and variance. *Demographic Research*, 10:231–263.

Zeng, Y. and Land, K. C. (2001). A sensitivity analysis of the Bongaarts-Feeney method for adjusting bias in observed period total fertility rates. *Demography*, 38:17–28.

Zeng, Y. and Land, K. C. (2002). Adjusting period tempo changes with an extension of Ryder's basic translation equation. *Demography*, 39:269–285.

Appendix

In order to estimate tempo-adjusted life expectancy for western and eastern Germany, I followed the approach of Bongaarts and Feeney (2002), who defined the tempo effect $S(t)$ in life expectancy in a year t as the absolute difference between the conventional life expectancy at birth $e_0(t)$ and the tempo-adjusted life expectancy at birth $e_0^*(t)$ (which Bongaarts and Feeney called the "average age at death"), thus

$$S(t) = e_0(t) - e_0^*(t). \tag{1}$$

Measure $e_0^*(t)$ is defined as the average age at death in a population with a constant number of births. This measure is closely related to the "cross-sectional average length of life" (CAL) but it is not identical (see Guillot 2003b). In subsequent studies, Bongaarts and Feeney (in this volume p. 11 and p. 29) presented further possibilities to estimate in a similar manner tempo-adjusted period life expectancy from complete cohort data on births, deaths, and migration respective cohort life tables in order to reconstruct empirically a constant birth population for a certain period. Detailed data such as these do not exist on the West and East German populations, however. When such cohort data is not available (at least for a time span long enough), $e_0^*(t)$ can be estimated by solving the equation

$$e_0(t) = e_0^*(t) - \frac{1}{b} \ln \left(1 - \frac{de_0^*(t)}{dt} \right) \tag{2}$$

for $e_0^*(t)$ from conventional life table estimates, based on the assumptions that mortality under age 30 can be neglected and that the annual changes in the force of mortality follow a shifting Gompertz function[9]. For a detailed derivation of this formula, see Bongaarts and Feeney (2002). As proposed by Bongaarts and Feeney (2002), value b is estimated by fitting a Gompertz model to annual age-specific death rates for ages 30-90[10]. Although cohort experiences are generally connected with age-specific period death rates and thus with the estimates of the Gompertz parameter b, Equation (2) does not

[9] The application of a Gompertz model requires the assumption that mortality under age 30 is negligible since the model does not fit the pattern of mortality in ages below 30. As this assumption is close to reality in modern populations with high life expectancy, it can be accepted as being applicable to western and eastern Germany from 1975 to 2004. However, this method cannot be used in populations with high mortality in infancy, childhood, and young adult ages.

[10] Bongaarts and Feeney (2002) fitted the age-specific death rates until age 100. For western and eastern Germany, however, official population and death data for the time series analyzed in this chapter are available only until age 90.

contain a direct cohort component and includes only elements derived from period data.

Table 2 presents the estimates of parameter $\mu_0(t)$ and the average of parameter b for the analyzed populations from 1975 to 2004, as done by Bongaarts and Feeney (2002) for the US, Sweden, Japan, and France. The estimates for b for the whole series of single observation years are shown in Tables 3 and 4, respectively. Corresponding to the observed death rates, $\mu_0(t)$ declines over time for all four populations. Similar to what is known for several other countries, the estimated values of b are close to 0.09 among males and 0.10 among females for both western and eastern Germany. During the observation period, the annual estimates of b vary only little over time in each of these populations, as can be seen from the standard deviation of b in Table 2 or from the single values in Tables 3 and 4, respectively. As with the populations analyzed by Bongaarts and Feeney (2002), the Gompertz model fits the observed adult death rates very well, with the average variance explained (R^2) being around 99 percent. This shows that the proportionality (or " constant shape") assumption is approximately valid and the indirect method as introduced by Bongaarts and Feeney (2002) can be applied.

Table 2. Estimates of the parameters of the Gompertz mortality change model, males and females, western and eastern Germany, 1975-2004.

	Average 1975-2004				
	$\mu_0(1975)$	$\mu_0(2004)$	b	St. dev. b	R^2
Western Germany. males	$6.639(\cdot 10^{-5})$	$2.276(\cdot 10^{-5})$	0.092	0.0023	0.997
Eastern Germany. males	$4.116(\cdot 10^{-5})$	$2.211(\cdot 10^{-5})$	0.094	0.0029	0.994
Western Germany. females	$1.729(\cdot 10^{-5})$	$4.322(\cdot 10^{-6})$	0.105	0.0036	0.984
Eastern Germany. females	$1.080(\cdot 10^{-5})$	$4.254(\cdot 10^{-6})$	0.108	0.0043	0.991

Based on these data, I used a three-step procedure similar to the procedure proposed by Bongaarts and Feeney (2002). First, I calculated the annual estimates of $e_0(t)$ from 1950 to 2004 with life tables that have mortality under age 30 set to 0. Next, I smoothed the estimates by fitting a sixth degree polynomial, using the computer program Microsoft Excel. The resulting values for the smoothed time series for life expectancy $e_0(t)^S$ are provided in Tables 3 and 4, respectively. Figure 2 shows the corresponding functions together with the original estimates for $e_0(t)$ with no mortality under age 30. It can be seen that the trends in $e_{30} + 30$ (what corresponds to setting mortality below age 30 to 0) are very similar to the trends in e_0, shown in Figure 2. They differ only slightly in the years 1950 to 1970, owing to the fact that mortality below age 30 (and especially infant mortality) had a higher impact on overall life expectancy than it has had in years more recent. Note that the significant decrease in life expectancy at birth $e_0(t)$ for East German men in 1990 diminishes in the smoothed values $e_0(t)^S$.

To estimate tempo-adjusted life expectancy $e_0^*(t)$, the original values for $e_0(t)$ are substituted by values $e_0(t)^S$ derived from the polynomial functions. To finally solve Equation (2) for $e_0^*(t)$, I used the so-called Euler's method, with $S(1950) = 2$ as the initial condition for the differential equation. From Equation (1) then follows that $e_0^*(1950)$ can be directly estimated from $e_0(1950) - S(1950)$. For instance, for West German males it follows that $e_0^*(1950) = 71.28 - 2.00 = 69.28$. This value represents the assumed tempo distortion for mortality changes until the year 1950, which was equally set for all populations observed, and thus the female and male populations of West and East Germany. The results for the analyzed years after 1975 are not entirely insensitive to this assumed initial condition for the year 1950 but its effect on the estimates is relatively weak[11]. An application of Euler's method leads to an estimate for the tempo-adjusted life expectancy $e_0^*(1951)$ for the next year from the equation:

$$e_0^*(1951) = e_0^*(1950) + \left\{1 - \exp\left[-b(1950)\left(e_0(1950)^S - e_0^*(1950)\right)\right]\right\}$$

or generally written from

$$e_0^*(t+1) = e_0^*(t) + \left\{1 - \exp\left[-b(t)\left(e_0(t)^S - e_0^*(t)\right)\right]\right\}. \tag{3}$$

Equation (3) was used to estimate a complete time series of values for tempo-adjusted life expectancy at birth (with no mortality under age 30) until 2004 for western and eastern German females and males. For a more detailed derivation of Equation (3), see Luy (2005a).

[11] Setting the initial condition to $S = 1$ or $S = 3$ causes the estimated West-east German differences in tempo-adjusted life expectancy to deviate from the chosen initial condition $S = 2$ by less than 0.01 years for the analyzed years 1975 to 2004; when setting the initial condition to $S = 0$ or $S = 4$, the deviation does not exceed 0.02 years.

Table 3. Estimates of $e_0(t)$, $e_0(t)^S$, b, $e_0^*(t)$, and $S(t)$ for single calendar years, males, western and eastern Germany, 1975-2004 (no mortality under age 30).

Year	West Germany					East Germany				
t	$e_0(t)$	$e_0(t)^S$	b	$e_0^*(t)$	$S(t)$	$e_0(t)$	$e_0(t)^S$	b	$e_0^*(t)$	$S(t)$
1975	71.17	71.46	0.091	70.91	0.27	71.28	71.49	0.098	71.36	-0.08
1976	71.49	71.59	0.089	70.96	0.53	71.46	71.47	0.093	71.37	0.09
1977	71.96	71.73	0.090	71.01	0.95	71.59	71.44	0.094	71.38	0.21
1978	71.85	71.89	0.091	71.07	0.77	71.33	71.41	0.094	71.39	-0.06
1979	72.20	72.06	0.088	71.15	1.05	71.20	71.38	0.095	71.39	-0.19
1980	72.29	72.23	0.088	71.22	1.07	71.07	71.35	0.097	71.39	-0.31
1981	72.39	72.42	0.089	71.31	1.08	71.38	71.32	0.092	71.38	-0.01
1982	72.63	72.62	0.090	71.40	1.23	71.44	71.30	0.092	71.38	0.06
1983	72.78	72.82	0.089	71.51	1.27	71.66	71.28	0.093	71.37	0.29
1984	73.18	73.03	0.091	71.62	1.57	71.70	71.28	0.094	71.36	0.34
1985	73.26	73.24	0.092	71.74	1.52	71.57	71.29	0.096	71.35	0.21
1986	73.54	73.45	0.091	71.87	1.67	71.51	71.33	0.095	71.35	0.16
1987	73.84	73.67	0.094	72.00	1.84	71.70	71.38	0.094	71.35	0.35
1988	74.07	73.88	0.093	72.14	1.92	71.56	71.46	0.092	71.35	0.21
1989	74.13	74.09	0.094	72.29	1.84	71.88	71.57	0.093	71.36	0.52
1990	74.22	74.29	0.094	72.45	1.77	71.14	71.71	0.090	71.38	-0.24
1991	74.35	74.49	0.092	72.61	1.74	71.31	71.89	0.087	71.41	-0.10
1992	74.68	74.69	0.091	72.77	1.91	71.77	72.10	0.090	71.45	0.32
1993	74.70	74.89	0.094	72.93	1.77	72.01	72.34	0.092	71.51	0.51
1994	74.96	75.08	0.094	73.09	1.86	72.40	72.63	0.090	71.58	0.82
1995	75.05	75.27	0.092	73.26	1.78	72.81	72.95	0.094	71.67	1.14
1996	75.27	75.47	0.093	73.43	1.84	73.27	73.30	0.092	71.78	1.49
1997	75.68	75.67	0.094	73.61	2.07	73.89	73.68	0.095	71.91	1.98
1998	75.99	75.89	0.094	73.78	2.21	74.43	74.08	0.095	72.07	2.37
1999	76.28	76.12	0.095	73.96	2.31	74.82	74.49	0.099	72.24	2.58
2000	76.54	76.37	0.093	74.15	2.39	75.03	74.91	0.096	72.44	2.59
2001	76.93	76.66	0.095	74.33	2.60	75.52	75.32	0.096	72.65	2.87
2002	77.06	77.00	0.095	74.53	2.53	75.63	75.71	0.098	72.88	2.75
2003	77.16	77.38	0.096	74.74	2.42	75.76	76.06	0.100	73.12	2.64
2004	77.76	77.84	0.096	74.96	2.80	76.33	76.34	0.099	73.37	2.96

Table 4. Estimates of $e_0(t)$, $e_0(t)^S$, b, $e_0^*(t)$, and $S(t)$ for single calendar years, females, western and eastern Germany, 1975-2004 (no mortality under age 30).

Year	West Germany					East Germany				
t	$e_0(t)$	$e_0(t)^S$	b	$e_0^*(t)$	$S(t)$	$e_0(t)$	$e_0(t)^S$	b	$e_0^*(t)$	$S(t)$
1975	76.84	77.10	0.101	75.59	1.25	75.91	76.07	0.106	75.34	0.57
1976	77.17	77.28	0.100	75.73	1.43	76.15	76.10	0.105	75.41	0.73
1977	77.74	77.48	0.102	75.88	1.86	76.53	76.14	0.103	75.48	1.05
1978	77.74	77.68	0.101	76.03	1.71	76.34	76.18	0.106	75.55	0.79
1979	78.03	77.89	0.099	76.18	1.85	76.40	76.23	0.106	75.61	0.79
1980	78.27	78.11	0.102	76.34	1.93	76.17	76.28	0.105	75.68	0.50
1981	78.34	78.33	0.102	76.50	1.84	76.37	76.35	0.105	75.74	0.63
1982	78.57	78.56	0.102	76.67	1.90	76.57	76.43	0.105	75.80	0.77
1983	78.76	78.79	0.102	76.85	1.91	76.82	76.52	0.106	75.87	0.95
1984	79.23	79.01	0.102	77.03	2.20	76.85	76.64	0.107	75.93	0.92
1985	79.30	79.24	0.104	77.21	2.09	76.77	76.77	0.107	76.01	0.76
1986	79.47	79.45	0.106	77.40	2.07	76.73	76.93	0.112	76.08	0.64
1987	79.82	79.67	0.108	77.60	2.22	77.14	77.11	0.111	76.17	0.97
1988	80.02	79.87	0.105	77.80	2.22	77.17	77.31	0.108	76.27	0.90
1989	80.07	80.07	0.104	77.99	2.08	77.49	77.54	0.105	76.38	1.11
1990	80.05	80.25	0.105	78.19	1.87	77.44	77.80	0.103	76.49	0.95
1991	80.26	80.43	0.106	78.38	1.87	77.69	78.09	0.101	76.62	1.07
1992	80.59	80.59	0.104	78.58	2.02	78.38	78.40	0.108	76.76	1.62
1993	80.50	80.74	0.106	78.77	1.74	78.86	78.74	0.106	76.92	1.94
1994	80.78	80.89	0.107	78.96	1.82	79.18	79.10	0.118	77.10	2.08
1995	80.89	81.02	0.107	79.14	1.75	79.53	79.47	0.108	77.31	2.23
1996	80.98	81.15	0.106	79.32	1.66	79.86	79.86	0.105	77.52	2.35
1997	81.32	81.29	0.108	79.50	1.82	80.43	80.26	0.115	77.73	2.69
1998	81.55	81.42	0.108	79.68	1.87	80.76	80.65	0.111	77.99	2.77
1999	81.71	81.57	0.108	79.85	1.86	81.18	81.03	0.115	78.24	2.94
2000	81.93	81.74	0.108	80.02	1.91	81.47	81.39	0.113	78.52	2.95
2001	82.19	81.93	0.111	80.19	2.00	81.75	81.71	0.110	78.79	2.96
2002	82.20	82.17	0.114	80.36	1.84	81.72	81.98	0.117	79.07	2.65
2003	82.21	82.46	0.112	80.55	1.66	81.97	82.18	0.115	79.36	2.61
2004	82.76	82.81	0.111	80.74	2.02	82.48	82.30	0.111	79.63	2.84

III. Comparison of period and cohort measures of longevity

Five period measures of longevity*

John Bongaarts

Population Council, 1 Dag Hammarskjold Plaza, New York, NY 10017, USA.
E-mail: jbongaarts@popcouncil.org

Summary. This study provides a summary of recently proposed alternatives period measures of "longevity" and assesses whether empirical differences between these measures are consistent with predictions from analytic studies. Particular attention is given to the tempo effect. Three of the five period measures are virtually equal to one another in a simulated population in which mortality follows a Gompertz model with a constant rate of improvement. Similar results are observed among females in Denmark, England and Wales and Sweden in the last quarter century. However, these three measures differ substantially from the conventional period life expectancy when mortality changes over time. These findings are consistent with theoretical analysis by Bongaarts and Feeney (2002, in this volume p. 11 and p. 29) which demonstrated that this deviation is caused by a tempo effect whose size varies with the rate of change in mortality.

1 Introduction

The most widely used measure of period mortality is life expectancy at birth calculated with a conventional life table. Alternative period measures of "longevity" exist, but have found very limited application. The purpose of this note is to provide a brief summary of recently proposed alternatives and to assess whether empirical differences between these measures are consistent with predictions from analytic studies. Particular attention will be given to the tempo effect as a cause of differences between measures. Comparisons rely on simulations in which the force of mortality follows a Gompertz model with a constant rate of improvement over time. Empirical estimates are also provided for three countries with long historical data series. A brief concluding comment summarizes the main reasons why certain measures differ and why others are nearly the same.

2 Definitions of period longevity measures

2.1 Life expectancy

$$e_0(t) = \int_0^\infty \exp\left\{-\int_0^x \mu(a,t)\,da\right\}\,dx \tag{1}$$

where $\mu(a,t)$ is the force of mortality at age a and time t. Standard demographic text books (e.g. Preston et al. 2001) discuss the calculation of this conventional measure. Estimates of e_0 are available for most countries of the world (United Nations, 2003)

2.2 Cross-sectional average length of life (Brouard 1986, Guillot, 2003)

$$CAL(t) = \int_0^\infty p_c(a, t-a)\,da \tag{2}$$

where $p_c(a, t-a)$ equals the proportion of survivors at age a and time t for the cohort born at time $t-a$. $CAL(t)$ sums proportions of cohort survivors at time t and it therefore equals the size of the population at time t in a closed population in which births have occurred at a constant rate of 1 per year in the past.

2.3 Tempo adjusted life expectancy (Bongaarts and Feeney in this volume p. 11; Vaupel in this volume p. 93)

$$e_0^*(t) = \int_0^\infty \exp\left\{-\int_0^x \frac{\mu(a,t)}{1-r(t)}\,da\right\}\,dx \tag{3}$$

This is a variant of the conventional period life expectancy, but the tempo effect in the force of mortality is removed by dividing this rate by $1-r(t)$. The variable $r(t)$, which is assumed to be the same for all ages, denotes the increments to life (or the delay in deaths) due to mortality improvements at time t. Vaupel (in this volume p. 93) refers to $e_0^*(t)$ as the "true life expectancy." Bongaarts and Feeney (in this volume p. 11) estimate $r(t)$ as

$$r(t) = \frac{dCAL(t)}{dt} \tag{4}$$

This estimate holds in populations in which the function $pc(a, t-a)$ shifts to higher and lower ages over time while maintaining its shape as longevity rises or falls. Bongaarts and Feeney (in this volume p. 11) examine this so-called "shifting assumption" and demonstrate that it provides a good approximation of observed patterns of adult mortality in recent decades in three high income countries.

2.4 Lagged cohort life expectancy (Bongaarts and Feeney in this volume p. 29; Goldstein in this volume; Rodriguez in this volume)

$$LCLE(t) = e_0^c(c) = e_0^c(t - e_0^c(c)) \tag{5}$$

$LCLE$ at time t is estimated as the life expectancy of the cohort born at time c with the lag between t and c equal to the life expectancy of the cohort: $c = t - e_0^c(c)$. $LCLE$ equals the life expectancy of the cohort that reaches its mean age at death at time t.

This measure is similar to one proposed and used by Ryder (1980) to study fertility trends. In Ryder's analysis of tempo effects in fertility he compared the quantum and tempo observed at time t with the quantum and tempo of the cohort born M years ago where M equals the mean age at birth for the cohort. In the mortality process there are no cohort quantum effects, but period and cohort tempo may be compared with (5).

2.5 Average weighted cohort life expectancy (Schoen and Canudas-Romo, 2005)

$$ACLE(t) = \int_0^\infty w(a,t) e_0^c(t-a)\, da \tag{6}$$

where $w(a,t)$ are the weights for the life expectancy of cohorts born at time $t - a$.

Schoen and Canudas-Romo (2004) estimate these weights as

$$w(a,t) = \frac{p_c(a, t-a)}{CAL(t)} \tag{7}$$

Thus, $ACLE(t)$ equals the weighted average of the life expectancies of the cohorts present at time t, with each cohort weighted by its probability of survival to time t.

The following empirical analysis will be limited to these five measures although others have been proposed (see for example Bongaarts and Feeney in this volume p. 11) and variants of these five might be constructed (e.g., alternative weights for $ACLE(t)$).

3 Results

To compare these five period indicators simulations are used in which mortality follows a Gompertz model with a constant rate of improvement over time. Following the basic model of Vaupel (1986) as extended by Schoen et al. (2004), the force of mortality at time t and age a is given by

$$\mu(x) = \alpha e^{\beta x} e^{-\rho t} \tag{8}$$

where α and β are the Gompertz level and slope parameters, and ρ equals the rate of mortality improvement. These parameters are held constant throughout a simulation.

Each simulation calculates the five longevity measures for a 50-year period using $\alpha = 0.00001887$ and $\beta = 0.1$. With these parameter values $e_0(t)$ equals 80.0 years at time $t = 0$. The trend in $e_0(t)$ during the 50 year simulation depends on the rate of mortality improvement. With $\rho = 0$, $e_0(t)$ remains constant, with $\rho = 0.01$ it rises linearly to 85 years and with $\rho = 0.02$ it rises linearly to 90 years. The simulation results are presented in Table 1. Figure 1 plots values for $\rho = 0.02$.

In the absence of mortality change ($\rho = 0$) all five period measures are constant and equal to one another (first panel of Table 1). With declining mortality ($\rho > 0$) differences arise, but three of the measures are nearly equal to one another throughout the simulation period:

$$CAL(t) \approx e_0^*(t) \approx LCLE(t) \tag{9}$$

However, $e_0(t)$ is higher than these three measures and $ACLE(t)$ is much higher still.

The simulations in Table 1 were repeated for different values for α, β with similar results. Lower values of α raised all estimates for all measures by the same amount, but kept the difference between them unchanged. Variations in β also made a difference but results will not be presented here because empirical estimates differ little from 0.1. The effect of changes in values of β as well as ρ can be estimated with an equation obtained by Bongaarts and Feeney (2002). They prove that the difference $e_0(t) - e_0^*(t)$ (called the tempo effect) can be estimated as $-\ln(1 - de_0^*(t)/dt)/\beta = -\ln(1 - \rho/\beta)/\beta)$ if mortality follows a Gompertz pattern with a constant rate of change in the force of mortality. According to this equation the difference between $e_0(t)$ and $e_0^*(t)$ equals 1.05 years when $\rho = 0.01$ and 2.23 years when $\rho = 0.02$ (assuming $\beta = 0.1$). These analytic estimates agree closely with the simulation results in Table 1.

Figures 2a, b, c plot estimates of four of the five period measures of longevity for females in Denmark, England and Wales, and Sweden from 1925-2000. Each measure is calculated with $\mu(a, t) = 0$ for $a < 30$ to insure that the shifting assumption holds approximately. $ACLE(t)$ is not included because its estimation requires projections of future mortality for more than a century. The results are consistent with the simulations: conventional life expectancy is higher than the other measures and $CAL(t) \approx e_0^*(t) \approx LCLE(t)$ in recent decades.

Table 1. Values of five period longevity measures in Gompertz model with declining mortality.

Time. t	$e_0(t)$	$CAL(t)$	$e_0^*(t)$	$LCLE(t)$	$ACLE(t)$
			$\rho = 0$		
0	80	80	80	80	80
25	80	80	80	80	80
50	80	80	80	80	80
			$\rho = 0.01$		
0	80	78.96	78.96	78.97	83.23
25	82.5	81.46	81.45	81.46	85.87
50	85	83.95	83.95	83.96	88.5
			$\rho = 0.02$		
0	80	77.8	77.79	77.84	87.25
25	85	82.79	82.78	82.81	92.88
50	89.99	87.78	87.77	87.79	98.5

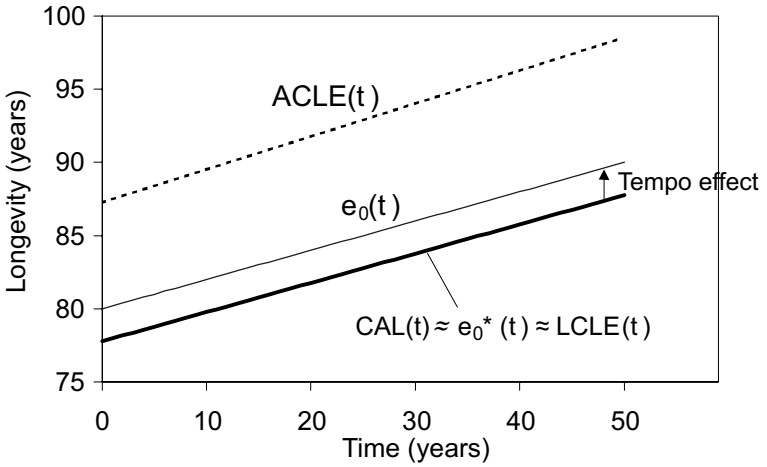


Fig. 1. Trends in five period longevity measures in a Gompertz model with declining mortality ($\alpha = 0.000018866, \beta = 0.1, \rho = 0.02$).

4 Discussion

A detailed discussion of the alternative longevity measures and their strength and weaknesses is beyond the scope of this descriptive note, and only brief comments on the most noteworthy findings will be provided.

First, in the simulations, three of the period longevity indicators $CAL(t)$, $e_0^*(t)$ and $LCLE(t)$ are virtually identical to one another. This finding is consistent with the analytic results by Bongaarts and Feeney (in this volume p. 11) who prove that $CAL(t) = e_0^*(t)$ in populations in which the shifting

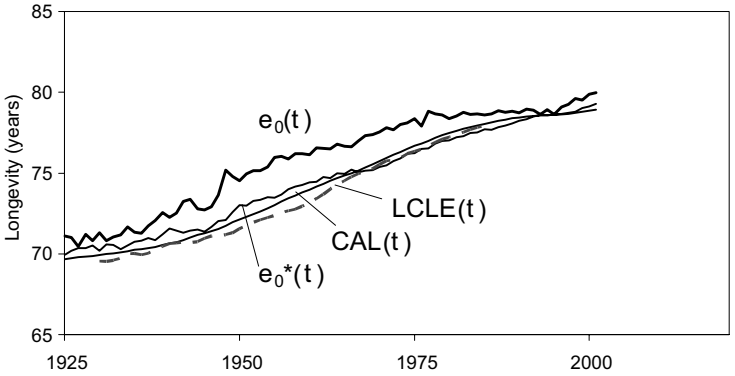


Fig. 2a. Trends in alternative period measures of longevity for females in Denmark, 1925-2000. No mortality under age 30.

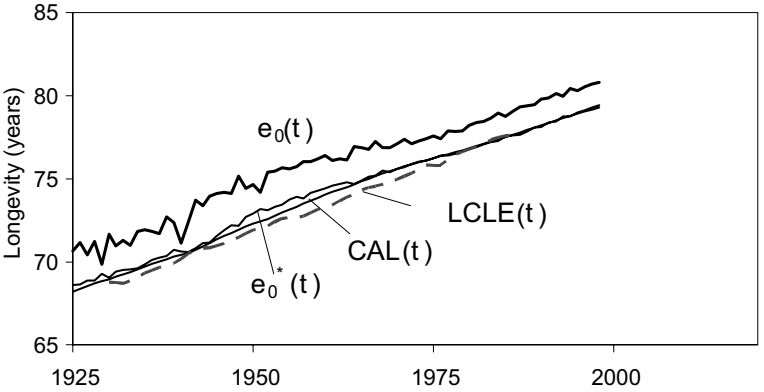


Fig. 2b. Trends in alternative period measures of longevity for females in England and Wales, 1925-2000. No mortality under age 30.

assumption holds. As noted, $p_c(a, t-a)$ is assumed to shift to higher and lower ages over time as longevity rises or falls. In the simulations based on the Gompertz model presented in Table 1, this shifting assumption is very closely approximated except at ages near zero. The error is small because the force of mortality around age zero is very small for a Gompertz with the parameter values used here. The finding that lagged cohort life expectancy $LCLE(t)$ is virtually identical to $CAL(t)$ is expected from Goldstein (in this volume) and Rodriguez (in this volume) who prove that $LCLE(t) = CAL(t)$ if the shifting assumption holds and if the shift is linear (i.e. annual changes in $CAL(t)$ are constant). This result is also consistent with the analysis of cohort and period tempo of fertility by Ryder (1980).

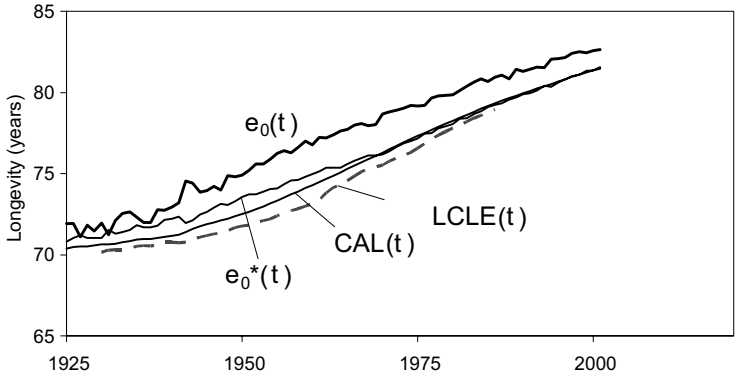


Fig. 2c. Trends in alternative period measures of longevity for females in Sweden, 1925-2000. No mortality under age 30.

Second, the conventional life expectancy at birth $e_0(t)$ is substantially higher than three other period measures $CAL(t)$, $e_0^*(t)$ and $LCLE(t)$. The difference between $e_0(t)$ and $e_0^*(t)$ is constant throughout the simulation but varies with the rate of improvement in mortality: it equals 2.2 years with $\rho = 0.02$, 1.05 years with $\rho = 0.01$ and 0 when $\rho = 0$. These findings are consistent with the mortality tempo effect described by Bongaarts and Feeney (2002, in this volume p. 11 and p. 29).

Third, the weighted average cohort life expectancy $ACLE(t)$ is much higher than the other four indicators. This difference is not surprising since the weights applied to the life expectancies of cohorts alive at time t are highest for the youngest (i.e. most recent) cohorts. As a consequence, this measure is heavily influenced by the mortality that young cohorts will experience in the future. This is confirmed by Schoen and Romo (2004) who conclude that $ACLE$is roughly the arithmetic mean of the period life expectancy at time t and the cohort life expectancy of the cohort born in year t.

The empirical results for females in Denmark, Sweden, and England in Figure 2 are similar to the simulation findings for recent decades i.e., since approximately the 1970s. However, in earlier decades, differences between $CAL(t)$, $e_0^*(t)$ and $LCLE(t)$, while still small, are no longer negligible. The probable reason for the modest divergence between $CAL(t)$ and $e_0^*(t)$ before ca. 1970 is that the shifting assumption is then less accurate. The reason for the appearance of a small but significant divergence between $CAL(t)$ and $LCLE(t)$ in the earlier period is presumably that the assumption of linear change in $CAL(t)$ is more accurate later than earlier in the last century.

5 Conclusion

Three of the five period measures of longevity are virtually equal to one another in a population in which mortality follows a Gompertz model with a constant rate of improvement. Similar results are observed among females in Denmark, England and Wales and Sweden in last quarter century. This equality is as expected from earlier analytic work by Bongaarts and Feeney (2002, in this volume p. 11 and p. 29), Goldstein (in this volume) and Rodriguez (in this volume). The finding that these three measures differ substantially from the conventional period life expectancy when mortality changes over time is consistent with theoretical analysis by Bongaarts and Feeney (2002,in this volume p. 11 and p. 29). They demonstrate that the deviation of $e_0(t)$ from the other period longevity measures is caused by a tempo effect whose size varies with the rate of change in mortality.

Acknowledgements

This research was supported by grant no R03 AG024083-01A1 from the National Institute of Aging, USA.

References

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. The quantum and tempo of life cycle events. *In this volume, also published in Vienna Yearbook of Population Research*, pages 115–151. 2006.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Brouard, N. (1986). Structure, et dynamique des populations. la pyramide des annes a vivre, aspects nationaux et exemples rgionaux. *Espaces, Populations, Socits*, 2(14-15):157–168.

Goldstein, J. Found in translation? a cohort perspective on tempo-adjusted life expectancy. *In this volume, also published in Demographic Research*, 14(5):71–84. 2006.

Guillot, M. (2003). The cross-sectional average length of life (CAL): A cross-sectional mortality measure that reflects the experience of cohorts. *Population Studies*, 57(1):41–54.

Nations, U. (2003). *World Population Prospects, The 2002 Revision*. New York: United Nations.

Preston, S. H., Heuveline, P., and Guillot, M. (2001). *Demography: Measuring and Modeling Population Processes*. Malden, MA: Blackwell.

Rodriguez, G. Demographic Translation and Tempo Effects: An Accelerated Failure Time Perspective. *In this volume, also published in Demographic Research*, 14(6):85–110. 2006.

Ryder, N. B. (1980). Components of temporal variations in American fertility. In Hiorns, R., editor, *Demographic Patterns in Developed Societies*, pages 15–54. London: Taylor & Francis.

Schoen, R. and Canudas-Romo, V. (2005). Changing mortality and average cohort life expectancy. *Demographic Research*, 13(5):117 – 142.

Schoen, R., Jonsson, S. H., and Tufis, P. (2004). A population with continually declining mortality. paper presented at the PAA 2004, Boston.

Vaupel, J. W. Lifesavings, lifetimes and lifetables. *In this volume, also published in Demographic Research*, 13(24):597 – 614. 2005.

Vaupel, J. W. (1986). How change in age-specific mortality rates affect life expectancy. *Population Studies*, 40:147–157.

Found in translation? A cohort perspective on tempo-adjusted life expectancy ⋆

Joshua R. Goldstein

Max Planck Institute for Demographic Research, Konrad-Zuse-Strasse 1, 18057 Rostock, Germany. E-mail: goldstein@demogr.mpg.de

Summary. What does tempo-adjusted period life expectancy measure? Taking a cohort perspective, I show that under conditions of constant linear mortality shifts the tempo-adjusted period indicator translates exactly to the cohort born $e_0^*(t)$ years earlier. I discuss the implications of cohort translation for the interpretation and application of tempo-adjusted period life expectancy.

1 Introduction

Life expectancy at birth is at root a cohort concept. It tells us how long, on average, the members of a cohort survive. Actual life expectancy can only be known fully for cohorts born long ago. To summarize recent mortality conditions and period-to-period variation, the hypothetical concept of period life expectancy is conventionally used. But even period life expectancy refers conceptually to a cohort – the hypothetical one that lives according to the rates observed in a single period.

When mortality conditions are improving, period life expectancy is *less* than that of the cohort born in the period. This is because the hypothetical cohort following the period life table is deprived of future mortality improvement.

I recite this basic property of period life expectancy because the "tempo adjusted" method of measuring period life expectancy – as developed by Bongaarts & Feeney (2002) – arrives at exactly the opposite conclusion. According to Bongaarts and Feeney, period life expectancy *overstates* longevity when mortality conditions are improving. They conclude: "Our main finding is that the conventional calculation of period life expectancy at birth gives a misleading indication of how long we live. We are not living as long as we thought we were." (p. 25).

To be fair, Bongaarts and Feeney, except at a few points, are not talking about cohorts. Instead, they intend e_0^* as a period measure that tries to improve upon period life expectancy. What such an improved period indicator actually measures is the subject of much debate as is clear from many of the chapters in this volume. The approach taken here is to recast tempo-adjustment in cohort terms. Doing this enables us to resolve the counter-intuitive direction of tempo-adjustment by showing which cohort B&F are referring to when they say "we."

The approach is similar to that of Goldstein & Wachter (2004), which showed – using a different model of temporal mortality change – the correspondence between period life expectancy e_0 and the life expectancy of particular cohort. Here, I look at which cohort has the life expectancy equal to current *tempo-adjusted* life expectancy e_0^*. I find that under linearly shifting mortality, defined below, tempo-adjusted life expectancy for year t translates to the cohort *dying* in year t: this is the cohort *born* e_0^* years earlier.[2]

An additional assumption is needed for this simple cohort translation of e_0^* to hold exactly. B&F's tempo-adjustment assumes that deaths are postponed uniformly across all ages, with the size of the shift possibly varying from year to year. To this I add the assumption that the size of the shift is constant from year to year, a pattern I call "linear shifts." As will be seen, the linear shift pattern is consistent with quite recent mortality trends above age 30 in low mortality populations. The linear shift assumption is, however, not a general feature of human populations. Prior to World War II, change was distinctly non-linear in many countries. It remains to be seen whether the recent linear shift pattern will continue.

Under the linear shift model, the current tempo-adjusted period life expectancy has the same value as the life expectancy of a past cohort. This correspondance with cohorts from the past explains why Bongaarts and Feeney's measure is less, not more, than current period life expectancy.

Furthermore, under linear shifts, it is possible to obtain directly the life expectancy of the cohort born in every period, including the current one, a quantity that is arguably of more interest than e_0^*.

Neither B&F's tempo-adjustment nor the discussion presented here applies to life expectancy at birth. Instead, both ignore all mortality before about age 30. For notational simplicity, the current discussion follows B&F, using the shorthand of e_0, e_0^*, and e_0^c to refer to the period, tempo-adjusted period, and cohort life expectancies at birth, assuming no mortality below age 30. In traditional demographic notation, these quantities would be written $e_{30} + 30$, $e_{30}^* + 30$, and $e_{30}^c + 30$.[3]

[2] This result was suggested in simulation by Bongaarts (2004), who also found that it held approximately in modern real-world populations.

[3] Although we use B&F's shorthand here, it is worth keeping in mind that although mortality below age 30 is low in modern industrialized populations, $e_{30} + 30$ does not equal e_0. In the 2002 Swedish female period life table, ignoring under-30

2 Proof of exact cohort translation

Let $l^c(a,t)$ be the surviving proportion of a cohort born at time $t-a$ and aged a at time t. For all $a \leq 0$, define $l^c(a,t) = 1$ for all t. This formulation amounts to the same thing as B&F's requirement of no mortality below age 30.

A proportionally shifting surface $l^c(a,t)$ consistent with B&F's proportionality assumption is obtained by shifting the baseline $l^c(a,0)$ up or down the age axis by an amount $F(t)$ such that

$$l^c(a,t) = l^c(a - F(t), 0), \tag{1}$$

again letting $l^c(a,t) = 1$ for $a - F(t) \leq 0$. The fact that $F(t)$ is not a function of age is the B&F's proportionality assumption. The additional assumption of linearity over time in the shifts can be introduced by letting $F(t) = rt$.

The cohort born at time τ has life expectancy

$$e_0^c(\tau) = \int_0^\infty l^c(a, \tau + a)\, da.$$

Following Bongaarts & Feeney (in this volume p. 11), the adjusted period life expectancy $e_0^*(t)$ is equal to

$$CAL(t) = \int_0^\infty l^c(a,t)\, da.$$

I use the CAL notation to emphasize its correspondence with the "cross sectional average length of life" introduced by Brouard (1986) and developed by Guillot (2003) .[4]

I want to show that

$$e_0^c(\tau) = CAL(\tau + e_0^c(\tau)). \tag{2}$$

Showing this demonstrates that the approximation given by Bongaarts (2004),

$$e_0^c(t - e_0^*(t)) \approx e_0^*(t), \tag{3}$$

actually holds exactly.[5]

This equality is shown as follows by expressing $e_0^c(\tau)$ and $CAL(\tau + e_0^c(\tau))$ in terms of $CAL(0) = \int_0^\infty l^c(a,0)\, da$.

---

mortality increases life expectancy by 0.6 years, more than a third of the 1.6 years tempo-effect that B&F find for Sweden 1980-1995.

[4] The quantity CAL used here differs from that used by Brouard and Guillot in that it assumes no child or young adult mortality under a given age such as 30.

[5] To see the correspondance, substitute $t = \tau + e_0^c(\tau)$ and note that from (2) $e_0^c(\tau) = CAL(t) = e_0^*(t)$.

Linear proportional shifts means that the cohort born at time τ has a survival curve that resembles the initial profile $l^c(a,0)$, except that each age "a" is shifted to age $a - r(\tau + a)$. In effect, a member of the cohort "feels younger" than they are by a factor of $r(\tau + a)$, where the $r\tau$ term accounts for the improvements up to the date at which the cohort is born, and the ra term accounts for the additional improvements obtained by the time the cohort reaches age a. Cohort period life expectancy under linear shifts can be written in terms of the baseline survival at time 0 as

$$e_0^c(\tau) = \int_0^\infty l^c(a - r(\tau + a), 0)\, da.$$

To evaluate, substitute $u = a(1 - r) - r\tau$ and $da = du/(1 - r)$. This gives

$$e_0^c(\tau) = \frac{1}{1 - r} \int_{0-r\tau}^\infty l^c(u, 0)\, du. \tag{4}$$

Recalling that for $u \leq 0$, $l^c(u) = 1$, the integral evaluates to

$$e_0^c(\tau) = \frac{CAL(0) + r\tau}{1 - r}. \tag{5}$$

We can evaluate $CAL(\tau + e_0^c(\tau))$ in a similar manner. The linearly shifting age distribution means that $CAL(t)$ is simply growing linearly with time. For any t,

$$CAL(t) = \int_0^\infty l^c(a, t)\, da = \int_0^\infty l^c(a - rt, 0)\, da.$$

Substituting $u = a - rt$ and $du = da$,

$$CAL(t) = \int_{0-rt}^{\infty-rt} l^c(u)\, du = CAL(0) + rt. \tag{6}$$

To prove (2), we are interested in $t = \tau + e_0^c(\tau)$. From (6),

$$CAL(\tau + e_0^c(\tau)) = CAL(0) + r(\tau + e_0^c(\tau)).$$

Substituting from (5) for $e_0^c(\tau)$,

$$CAL(\tau + e_0^c(\tau)) = CAL(0) + r\tau + r\frac{CAL(0) + r\tau}{1 - r},$$

which simplifies to

$$CAL(\tau + e_0^c(\tau)) = \frac{CAL(0) + r\tau}{1 - r}. \tag{7}$$

The right-hand side of this last expression is identical to the right-hand side of equation (5) for $e_0^c(\tau)$, which is what we wanted to show to prove (2).

Note that this change of variable approach is perfectly general for any survival curve $l^c(a, 0)$ and any $r \neq 1$. It does not require Gompertzian survival or any other particular form of the hazards.

3 Discussion

We have shown that tempo-adjusted period life expectancy $e_0^*(t)$ under linear shifts is equal to the life expectancy of the cohort dying in that year t.

The equality of tempo-adjusted life expectancy with lagged cohort life expectancy provides us with an alternative way to think about e_0^*. Whereas B&F use e_0^* as a counterfactual estimate of period mortality corrected for tempo distortion, we have shown here that in the context of steadily shifting survival curves e_0^* is also a measure of cohort life expectancy.

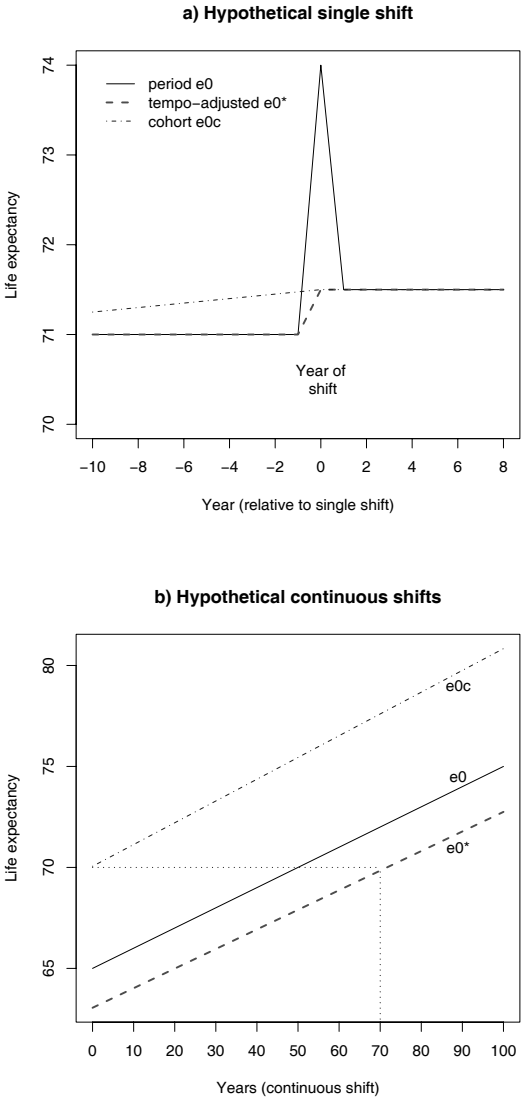
Both interpretations are interesting and potentially useful. I would argue that the B&F interpretation is most valuable in conditions of sudden mortality change, whereas the cohort interpretation is more valuable in conditions of steady mortality change.

Below I lay out two extreme scenarios that help us to understand the difference. Bongaarts and Feeney introduced the first in their 2002 paper; the second is explored by their paper in this volume as well as the chapter by Rodriguez (in this volume).

3.1 A single magic pill

The story of the "life extension" pill discussed by Bongaarts and Feeney (in this volume p. 11) illustrates the potential advantages of tempo-adjustment in the case of a sudden shift in survival. (See Figure 1a.) On January 1, everyone in a previously stationary population takes a pill postponing their previously programmed date of death by 3 months. Everyone born afterwards also takes the pill. The effect of such a pill in the year it is taken is to reduce the number of deaths by one-fourth, since no one will die in the first 3 months of the year. In the year the pill is introduced, death rates also fall by about one-fourth, raising life expectancy dramatically, not by the three months indicated by the pill but by several years because of the enormous drop in death rates.

In the case of a single such pill, period life expectancy spikes in the year the pill is taken and then falls thereafter to a constant value equal to the pre-pill life expectancy plus the extension granted by the pill. This makes the measurement taken in the year the pill appeared suspect, a candidate for tempo-adjustment. As panel (a) shows, e_0 shows a spike in the year the pill is introduced, but e_0^* shows no spike, instead attributing the appropriate 3 month increase in life expectancy.



Source: Panel (a) from B&F (2003) Figure 5, with illustrative $e_0^c(t)$ added. Panel (b) calculated for e_0 growing linearly from 65 to 75 at a rate of 0.1 years per year, with $e_0^*(t) = e_0(t)[1 - r * H]$ and $e_0^c(t) = e_0^*/(1 - r)$, where $r = 0.1$ and $H = .3$.

Fig. 1. Time paths of period and cohort life expectancy and of tempo-adjusted period life expectancy in (a) single shift scenario and (b) linear shift scenario.

The figure also shows cohort life expectancy for those born in each year. From a cohort view, adjusted-life expectancy performs well in the year that the pill is taken. In that year, unadjusted e_0 overestimates the life expectancy of the cohort being born, but the adjusted period measure e_0^* accurately predicts cohort life expectancy. In the years following the pill introduction, both e_0^* and e_0 are equal to e_0^c. In the years before the pill is taken, however, neither period life expectancy nor adjusted period life expectancy matches cohort life expectancy because neither period measure can foresee the subsequent sudden increase in longevity.

The lesson to be drawn from this scenario is that under a sudden mortality shock, akin to the one-time pill[6], e_0^* provides a better indication of the implications of the shock for cohort mortality than does e_0.

3.2 A series of magic pills

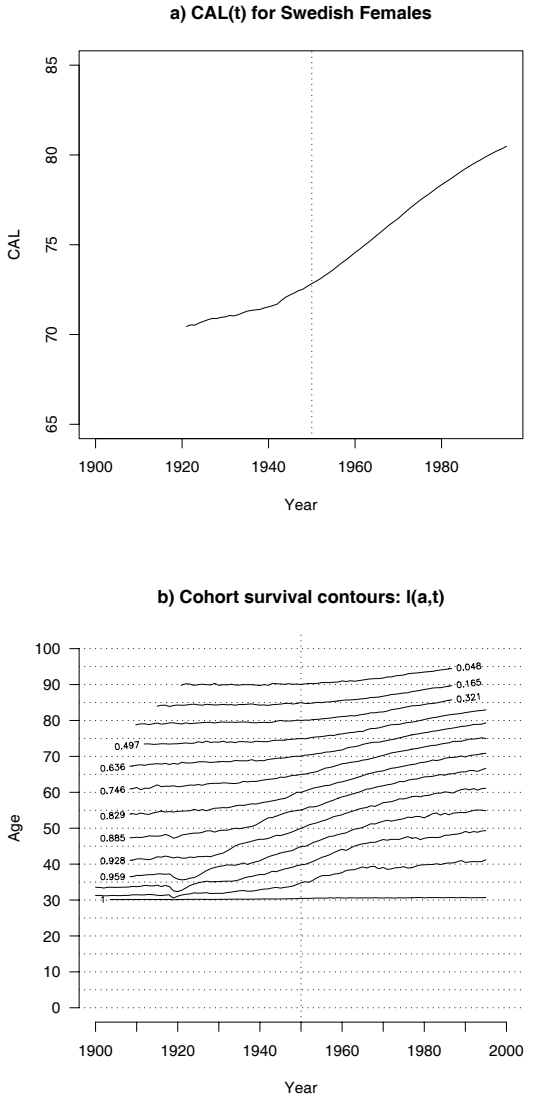
Now let us consider the case where such pills are given year after year, continually re-extending life by some constant amount each year. This scenario is the one investigated mathematically above and is illustrated in Figure 1b. In this case, we still have $e(t)$ larger than $e_0^*(t)$, but rather than this difference occurring in a single year as in the single-pill example, it persists over time. Now, the equality of $e_0^*(t)$ is not with the cohort born in year t, as in the single year example, but rather with the cohort born $e_0^*(t)$ years earlier that is *dying* in year t. This result from the formal analysis is illustrated by the dotted lines in the figure showing that $e_0^c(0) = 70 = e_0^*(70)$.

Under linear shifts that result from a series of pills, it would clearly be wrong to interpret tempo-adjusted life expectancy as an estimate of the cohort born in year t. The adjustment moves period life expectancy farther from, not closer to, that of the cohort.

3.3 Which scenario is more realistic?

We can now ask which of these two cases bears more resemblance to observed patterns of mortality change. Here there is no debate. Bongaarts and Feeney (2002 and in this volume p. 11) answer this question quite clearly in their empirical analysis of 1980-1995 France, Sweden and the United States (See for example figure 6 of B&F in this volume p. 11). In every case, the improvement of mortality by all measures has been steady. There is no historical example that bears any resemblance to the one-time pill example. Tuljapurkar, Li & Boe (2000) show, using methods different from the shift model, that since World War II, steady mortality decline is the rule throughout the industrialized world.

[6] See Le Bras (in this volume), who argues that even those one-time shocks that are observed do not occur in a manner that delay or advance deaths uniformly by age.



Note: Both panels assumes no mortality under age 30. $CAL(t) = \int_{30}^{\infty} l^c(a,t)\,da + 30$. Since $r = \frac{d}{dt} CAL(t)$, constant shifts imply linear $CAL(t)$. Source: Calculated from $m(x,t)$ data from www.mortality.org.

Fig. 2. Observed time paths of mortality change for Swedish females. Panel (a): Mortality change over all ages as measured by $\mathbf{CAL(t)}$ Panel (b): Contour plot of cohort survival $\mathbf{l^c(a,t)}$ using isoclines intersecting $\mathbf{l^c(30)}, \mathbf{l^c(35)}, \ldots, \mathbf{l^c(95)}$ in 1950.

It is useful to look at a longer course of time. Panel (a) shows CAL – what Bongaarts and Feeney call e_0^* – for Sweden females from 1920 to 1995. Bongaarts & Feeney (in this volume p. 11) figure 6 shows the last 15 years of this series. Linearity in CAL implies linear shifts. We see that the near linearity they find for 1980-1995 is a continuation of the post-World War II pattern. Before this, however, the pace of improvement was considerably slower. There is no evidence from looking at CAL of sporadic large mortality shifts of the kind in the single-pill scenario. Rather, the last half-century has been consistent with the linear shift scenario.

We can see in detail at how close both the proportionality and linear shift assumptions hold by looking at the full $l^c(a,t)$ surface (Panel (b)). The contour plot shows the isoclines of $l^c(x,t)$ at the levels seen in 1950 for $x = 30, 35, \ldots 95$. For example, the contour labeled "0.048" shows the age at which $l^c(90, 1950)$ is reached over the course of the century, and we can see that by 1995 this level of survival was reached at age 95 rather than 90.

Proportionality can be checked by looking at whether the slopes at different ages change simultaneously. The linearity of the shifts requires further that the contours be straight lines. The figure shows there were few shifts at all in the first two decades of the century in Sweden. Starting after World War I, and the influenza epidemic, survival to younger ages started to shift, followed by shifts in survival to older ages after World War II. Since about 1950, the contours are nearly linear and nearly parallel, particularly above age 60, when most deaths are occurring. Overall, neither proportionality or linearity seems a good description for the whole century. However, the linear shift model does not seem at odds with recent decades. The only evidence of mortality change that resembles the single-pill example is perhaps the 1918 influenza epidemic, but even this does not appear across all ages.

3.4 Telling the future

If we expect linear shifts well into the future, then we can go one step further. We have seen that under linear shifts, e_0^* understates even more dramatically than period life expectancy the survival of those born in a period. However, the same derivation we used to show the cohort that has life expectancy $e_0^*(t)$ can also be used to show the life expectancy of the cohort born in year t. Replacing τ with t and substituting from (6), we find

$$e_0^c(t) = e_0^{**} = \frac{e_0^*(t)}{1-r}, \tag{8}$$

where we use e_0^{**} to denote the rescaled e_0^*. Although $e_0^*(t)$ is itself a rather out-of-date measure referring to a cohort born long before t, the simplicity of the linear shift model allows us to go from $e_0^*(t)$ to the cohort born in year t by rescaling.

These exact relationships for steady mortality change should hold approximately when there are small variations in the pace mortality improvement.

If there is no temporal autocorrelation, the variations will cancel each other out. Such random ups and downs seem to encompasses the modern experience of mortality decline in advanced industrial countries, forming the basis of the Lee-Carter stochastic forecasting method (Lee and Carter 1992). Systematic slowdowns or accelerations that last many years can make the relationship between e_0^* and e_0^c quite different from the results found here. [7]

3.5 The order of mortality measures

With an exact expression for cohort life expectancy, we can now provide a full description of the ordering of different measures of life expectancy and their cohort translations under linear shifts. Table 1 shows tempo-adjusted period life expectancy, unadjusted period life expectancy, and rescaled tempo-adjusted life expectancy for Sweden using the same data as B&F and the cohort translation of these quantities. The table reiterates the point we began with that cohort life expectancy is larger, not smaller, than period life expectancy if we are considering the cohort born in the period. It shows that e_0^* actually refers to the cohorts born around 1900-1915, not cohorts born in 1980-1995.

In this example, period life expectancy and tempo-adjusted period life expectancy are close to each other relative to cohort life expectancy. The ordering $e_0^* < e_0 << e_0^{**}$ applies quite generally in conditions of improving mortality. Letting H denote Keyfitz's measure of life table entropy, e_0^* increases the observed mortality rates by a factor of about $1+r$, which reduces life expectancy by about a factor of $1-Hr$ (Keyfitz 1985). Life table entropy is small, on the order of 0.2, and so if $r = 0.1$, $e_0^* \approx 0.98 e_0$. To see that e_0^{**} is larger than either of these, note that dividing e_0^* by $1-r$ gives a quantity substantially greater than e_0.[8]

[7] A more general expression for cohort mortality can be given as follows. Let r_t be the shift in year t and R_t be the cumulative shift $\int_0^t r_t dt$. In this case, cohort life expectancy is given in terms of the baseline survival profile as

$$e_0^c(\tau) = \int_0^\infty l^c(a - R_{\tau+a}, 0)\, da.$$

Substituting $u = a - F(\tau + a)$ and $da = du/[1 - r(\tau + a)]$,

$$e_0^c(\tau) = \int_{-F_\tau}^\infty l^c(u)[1 - r_{\tau+a}]^{-1}\, du.$$

This reduces to (4) for when $r_{\tau+a}$ is a constant. When $r_{\tau+a}$ varies only slightly and in a manner that is uncorrelated with $l^c(u)$, then fluctuations should not influence $e_0^c(\tau)$ much, since shifts larger-than-average shifts will be cancelled out by smaller-than-average shifts.

[8] Formally, if entropy is sufficiently large then the inequality need not hold. But high H implies a high variance of age at death, typically in the form of high mortality among children, an age-group that is excluded from the Bongaarts-Feeney shift model.

Table 1. Ordering and cohort translation of period and tempo-adjusted period measures under linear shifts.

Period or tempo-adjusted period measure	Estimate for Sweden, 1980-95	Cohort translation	
$e^*_{30} + 30$	79.4	$e^c_0(t - e^*_0)$	$\approx e^c_0(1900 - 1915)^a$
$e_{30} + 30$	81.1	$e^c_0(t - \lambda)$	$\approx e^c_0(1905 - 1920)^b$
$e^{**}_{30} + 30 = \frac{e^*_0 + 30}{(1-r)}$	94.5	$e^c_0(t)$	$\approx e^c_0(1980 - 1995)^c$

[a] This cohort life table value was reached by the cohort of 1909, according to www.mortality.org.
[b] $\lambda < e^*_0$ but exact value unknown; 1905-1920 is a rough estimate.
[c] Assuming continued linear shifts.

Values for $e^*_{30} + 30$ and $e_{30} + 30$ from B&F(2004) Table 1. $e^{**}_{30} + 30$ calculated as $\frac{e^*_0}{(1-r)}$ using $r = 0.16$ as estimated by author from www.mortality.org. In "Cohort translation" column e^c_0 is used as shorthand for $e^c_{30} + 30$.

Without a crystal ball, we don't know for sure how long the cohorts born from 1980 to 1995 will live. But what we do know, assuming continued mortality decline, is that e^*_0 is clearly the worst measure, giving an even lower figure than the already too low period life expectancy. If we are going to adjust period life expectancy, we should readjust it again to produce not the cohort born long ago, but rather our best guess at the cohort born today, e^{**}.[9]

4 Conclusion

Some critics of Bongaarts and Feeney's theory of mortality tempo effects argue that its assumption of uniform postponement of death across all ages is unrealistic. Others argue that e^*_0 is not really a period measure, but rather depends on the history of the population. In this chapter, my approach has not been to try to debunk tempo-adjustment but rather to take it even further by assuming that the Bongaarts and Feeney's uniform shift repeats itself over many decades – so long that cohort mortality becomes a simple function of the baseline mortality schedule and the pace of the shift.

Under these conditions, two results were found. First, $e^*_0(t)$ translates to cohort life expectancy for those born $e^*_0(t)$ years earlier, long before the period under consideration. Second, the cohort life expectancy of those born today, or in any year t, can be found by a simple inflation of $e^*_0(t)$. Viewed this way, $e^*_0(t)$ itself is not a measure of great interest. It does not tell us what is

[9] Incidentally, this figure of 94.5 years is not out-of-line with optimistic forecasts. Oeppen & Vaupel (2002) predict that record period life expectancy will be 95 by 2040, which would apply to cohorts born about 1970 or 1980 (Goldstein and Wachter 2004).

happening in year t – this is given by the unadjusted period life table. It does not tell us the future – this is given by the life table of the new-born cohort. Rather it tells us about the cohort born in the past that is, on average, dying in year t.[10]

If mortality change were to be sudden, and to occur in such a way as to advance or to postpone deaths uniformly across all ages, tempo-adjustment could produce measures giving a valuable sense of the implications of the mortality rates seen during shocks. The difficulty, so far, is that mortality change has not occurred in this way. Recent history in the industrialized world has been has been one of steady, not sudden, mortality change. In this context, the linear shift model provides a framework for understanding what tempo-adjusted life expectancy is actually measuring and for developing even more informative indicators.

Acknowledgements

I would like to thank John Bongaarts, Tom Cassidy, German Rodriguez, Kenneth Wachter, and all of the participants in the Population Council/Max Planck workshop for many useful discussions. The views, and any errors, expressed here are my own.

References

Bongaarts, J. (2004). Six Period measures of longevity. *Unpublished manuscript.*

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Brouard, N. (1986). Structure et dynamique des populations. La pyramide des années à vivre, aspects nationaux et exemples régionaux. *Espaces, Populations, Sociétés*, 2(14-5):157–68.

Goldstein, J. R. and Wachter, K. W. (2004). Gaps and lags: Relationships between cohort and period life expectancy. Unpublished manuscript presented at the annual meeting of the Population Association of America, Boston.

[10] The backward-looking tendency of e_0^* is not due to the way it is measured, which after all is from current data, but rather from fact that the post-adjustment longevity estimates are equal to those of cohorts born long ago. Wachter (in this volume) shows that the differential equations that define e_0^* effectively define a moving average of recent period life expectancies.

Guillot, M. (2003). The cross-sectional average length of life (CAL): A cross-sectional mortality measure that reflects the experience of cohorts. *Population Studies*, 57(1):41–54.

Keyfitz, N. (1985). *Applied Mathematical Demography*. Springer.

Le Bras, H. Mortality tempo versus removal of causes of mortality: opposite views leading to different estimatinos of life expectancy. *In this volume, also published in Demographic Research*, 13(25):615–640. 2005.

Lee, R. D. and Carter, L. (1992). Modeling and forecasting U.S. mortality. *JASA*, 87(419):659–671.

Oeppen, J. and Vaupel, J. W. (2002). Broken Limits to Life Expectancy. *Science*, 296(5570):1029–1031. http://www.sciencemag.org.

Rodriguez, G. Demographic translation and tempo effects: An accelerated failure time perspective. *In this volume, also published in Demographic Research*, 14(6):85–110. 2006.

Tuljapurkar, S., Li, N., and Boe, C. (2000). A universal pattern of mortality decline in the G7 countries. *Nature*, 405:789–792.

Wachter, K. Tempo and its tribulations. *In this volume, also published in Demographic Research*, 13(9):201–222. 2005.

IV. Conclusions

Afterthoughts on the mortality tempo effect

John Bongaarts[1] and Griffith Feeney[2]

[1] Population Council, 1 Dag Hammarskjold Plaza, New York, NY 10017, USA. E-Mail: jbongaarts@popcouncil.org
[2] 9 Fairview Road, Scarsdale, NY 10583, USA. E-Mail: feeney@gfeeney.com

Summary. The preceding chapters in this volume provide a broad ranging and stimulating analysis of our claim that conventional estimates of period life expectancy may be distorted by a mortality tempo effect. Much new insight into the process of mortality change and its measurement has been gained, but there is no clear consensus on the existence, nature and size of the tempo effect. Views from different contributors range widely from strongly supportive to dismissive.

The purpose of this note is to comment briefly on the main question raised about our analysis of the mortality tempo effect: Is our tempo adjusted life expectancy a current measure of mortality conditions as we (and Vaupel in this volume p. 93 and Guillot in this volume) believe or a measure of the past as suggested by Rodriguez (in this volume) and Wachter (in this volume)?

1 Do tempo adjusted period longevity measures reflect current mortality conditions?

Conventional analyses of levels and trends in period mortality indicators such as life expectancy at birth are based on the assumption that current mortality *rates* measure current mortality *conditions*. Vaupel (2002, in this volume p. 93) concludes that mortality rates do not necessarily represent mortality conditions because of heterogeneity in mortality risks and/or because of delays in deaths, where "death delays" refer to the empirical tendency of survival curves to shift uniformly to the right at ages beyond young adulthood. We focus here on the role of death delays.

To clarify what is meant by "conditions" we turn to a published comment of Hajnal (1948). Discussing the relation between the marriage rate and the population age and sex structure he observes that

> ...we must know how, given the present marriage habits of the population, marriage rates would change as the age and sex structure of the population changes.

The remark is notable as an early articulation of the idea that demographic phenomena may or may not be adequately described by *rates* in the conventional sense of the word.

By "conditions" we mean what Hajnal meant by "habits:" an idea about how the phenomena in question "works" that may be represented by a mathematical model. The model will express the phenomena in question in terms of one or more parameters that describe the state of the system. If the values of these parameters cannot be directly observed, we will search for observable quantities that provide information on (and preferably determine) the parameter values.

The distinction between rates and conditions implies two potentially different period longevity measures defined as follows:

$e_0(t)$ = the mean age at death implied by current mortality *rates*
$M(t)$ = the mean age at death implied by current mortality *conditions*

We will refer to $e_0(t)$ as the conventional or unadjusted life expectancy and to $M(t)$ as the adjusted life expectancy. The interpretation of these period measures and any distortions in them depends on how the underlying process is modeled, as we discuss below.

1.1 The conventional "rates" perspective

The conventional "rates" perspective on the measurement of longevity holds that observed mortality rates (hazards or rates of the first kind) are the appropriate measures of period mortality and $e_0(t)$, calculated from these rates, is the most appropriate measure of period longevity. All other mortality variables, including $M(t)$, are derived from these rates. Expectation of life at birth, for example, is calculated as

$$e_0(t) = \int_0^\infty \exp\left(-\int_0^a \mu(x,t)\,dx\right)\,da. \tag{1}$$

where $\mu(x,t)$ denotes the force of mortality at age x and time t.

As Wachter (in this volume) points out, our estimate of $M(t)$, obtained with equation (3) below, is related to the conventional period life expectancy as

$$M(t) \approx \int_{-\infty}^{t} w_t(x) e_0(x)\,dx \tag{2}$$

thus making $M(t)$ a weighted average of $e_0(t)$. (We follow Wachter's simplifying assumptions and use his notation)

The conception of "how mortality works" in this context, how the underlying process is modeled, is rarely made explicit, but a plausible underlying model is that of organisms exposed to shocks and stresses imposed by their environment that may result in immediate death. This being the case, the

numbers of deaths will be linear function of the number of persons exposed to risk, which function is fully specified by the ratio of deaths to persons at risk. Mortality *rates* are in this case a faithful representation mortality *conditions*.

1.2 The "conditions" perspective

In what we call the "conditions" perspective, adult mortality in high life expectancy populations "works" differently. Individuals die when their allotment of life has been exhausted. An allotment can increase or decrease over time as mortality conditions in successive periods vary during the individual's life. In this perspective the most appropriate measure of period longevity is $M(t)$, with all other mortality variables, including $e_0(t)$, derived from these fundamental conditions.

$M(t)$ is a period indicator of current mortality conditions and is defined as the life expectancy of the cohort born in year t if no further changes in conditions occur after time t (see Vaupel in this volume p. 93 and Guillot in this volume for similar definitions).

As shown by Vaupel (in this volume p. 93) current conditions by age can be estimated as $\frac{\mu(x,t)}{1-\delta(t)}$ and the adjusted period life expectancy of life is given by

$$M(t) = \int_0^\infty \exp\left(-\int_0^a \frac{\mu(x,t)}{1-\delta(t)}\,dx\right)\,da \tag{3}$$

where $\delta(t)$ equals the increment to life at time t, i.e., the addition made to the life lines of everyone alive at time t as mortality declines.

Equation (3) is the same as the one provided by Bongaarts and Feeney (in this volume p. 11 and p. 29), who estimate $\delta(t)$ as the rate of change in the adjusted life expectancy, $\delta(t) = \frac{dM(t)}{dt}$, and present methods for the estimation of $\delta(t)$. Note that nothing on the right side of equation (3) depends on the past: $\mu(x,t)$ is the current force of mortality and $\delta(t)$ equals the delay in the timing of future deaths caused by changes in conditions at time t.Vaupel (in this volume p. 93) calls $M(t)$ the "true" life expectancy at birth. Guillot (in this volume) concludes that $M(t)$ can be interpreted as an indicator reflecting current mortality conditions under specific assumptions.

In the mortality conditions perspective, the conventional period life expectancy is considered distorted and it is determined by $M(t)$ as follows

$$e_0(t) = M(t) + g(t)\frac{dM(t)}{dt} \tag{4}$$

This equation is obtained by rearranging equation (7) in Wachter (in this volume); Bongaarts and Feeney (2002) and Guillot (2003, in this volume) provide similar equations. (For Gompertz mortality with a fixed slope, $g(t) \approx \beta^{-1}$.)

Equation (4) shows that conventional period life expectancy differs from the adjusted life expectancy $M(t)$ by an amount that depends on the rate at

which $M(t)$ is changing. This means that $e_0(t)$ will be a distorted measure of period longevity implied by current mortality conditions. The difference between $e_0(t)$ and $M(t)$ is the mortality tempo effect.

It is important to note that (2) and (4) are both expressions relating $e_0(t)$ and $M(t)$. In fact, (2) is the solution to differential equation (4), which means that substitution of (2) in (4) yields an identity. The difference between these equations is that $M(t)$ is the independent variable in (4), whereas $e_0(t)$ is the independent variable in (2).

The chapters by Rodriguez, and Wachter focus on the conventional rates perspective. In this perspective, current rates and the $e_0(t)$ calculated from them are not distorted and $M(t)$ depends on past rates. Vaupel (2002) and Bongaarts and Feeney (2002, in this volume p. 11 and p. 29) focus on the perspective in which individuals die when their allotment of life has been exhausted. In this perspective, $M(t)$ is independent of the past force of mortality, as shown by (3), and $e_0(t)$ is distorted. Guillot (in this volume) provides descriptions and insightful comments on these two perspectives.

The preceding discussion contrasts two quite different perspectives, based on two different models for the process of mortality, but it leaves open the question as to which model is the better representation of the reality of human mortality. A full discussion of this issue is beyond the scope of this note but we believe that the rates perspective is largely correct for causes of deaths that occur more or less at random (e.g. deaths from infection, accidents and violence, which predominate in childhood and among young adults). In contrast the conditions perspective is correct for mortality at older ages when deaths do not occur randomly but are instead the result of senescence. Senescence refers to the slow deterioration of cellular and physiological processes which precedes deaths from degenerative diseases, mostly above about age 30. This is why Bongaarts and Feeney (2002, in this volume p. 11 and p. 29) restrict their analysis of the mortality tempo effect to ages above 30.

1.3 An illustration

To clarify the distinction between the rates and conditions perspectives we will now present a brief analysis of these contrasting approaches in a model population. In this population every newborn receives a ticket with a predetermined age at death (a random variable). Let $T(t)$ denote the average age on the tickets issued in year t, but the age on any person's ticket can be changed at any time during the person's life, for example if the person lives through a year in which medical or public health discoveries occur. Innovations in year t (e.g. new drugs, surgical techniques) in medicine or public health raise everyone's life expectancy (the ticket value) provided the innovations remain effective over time.

Let $r(t)$ denote the increment to the age on the ticket made in year t. Suppose that the increment $r(t)$ may vary from year to year, but it is the

same for all individuals alive at time t. This implies that the increment to the value of the ticket does not depend on the age of the person holding it.

To illustrate, suppose that the average value of these tickets has been constant and equal to $T(0)$ until year 0 (i.e. $r(t) = 0$ for $t < 0$), and that mortality improvements occur after $t = 0$. The average value of the ticket given to a newborn in year t then equals $T(0)$ plus the sum of all improvements between years 0 and t so that in continuous time

$$T(t) = T(0) + \int_0^t r(x)\, dx \tag{5}$$

and

$$r(t) = \frac{dT(t)}{dt} \tag{6}$$

In this model population the above equations apply and it can be shown that

a. the adjusted life expectancy, which measures current conditions, equals $T(t)$, because $T(t)$ equals the mean age at death of the cohort born at time t if no further improvements in mortality conditions occur in the future:

$$M(t) = T(t) \tag{7}$$

b. the conventional unadjusted life expectancy differs from the ticket value and hence from $M(t)$, and the difference depends on the rate of improvement in mortality conditions. This follows from equation (4) :

$$e_0(t) > M(t) \quad \text{for } r(t) > 0 \tag{8}$$

and

$$e_0(t) < M(t) \quad \text{for } r(t) < 0$$

That is, when mortality conditions are improving the conventional life expectancy derived from rates exceeds the ticket value $M(t)$ and the reverse is true when the mortality conditions are deteriorating. The difference between these measures is the mortality tempo effect which varies with the value of $r(t)$ but is independent of the past. (If mortality follows a Gompertz the tempo effect equals approximately $\frac{r(t)}{\beta}$). The reason for the existence of the tempo effect is the thinning out of events in any year in which $r(t)$ is positive. As conditions improve in year t deaths that would have occurred in the year t without the improvement are postponed to some future year thus reducing the density of deaths in year t. This postponement and hence the thinning out of

events continues as long as conditions keep improving (e.g. with constant non zero $r(t)$, the values of $e_0(t)$ and $M(t)$ will differ but they will change over time at the same pace).

As shown by Bongaarts and Feeney (2002, in this volume p. 11 and p. 29) the distortion caused by this thinning can be removed by making an adjustment which divides the observed but distorted force of mortality by $(1 - r(t))$. Using this adjusted force of mortality in a conventional life table yields $M(t)$ which equals $T(t)$. Vaupel (in this volume p. 93) has a very similar view of this process (in his chapter he uses $\delta(t)$ for $r(t)$)

In sum, as noted by Vaupel (2003), life expectancy under current conditions does not equal life expectancy under current rates. The conventional period life expectancy is of course a summary measure of current rates ($e_0(t)$ in fact equals the inverse of the weighted average of age specific mortality rates), but when mortality conditions are changing $e_0(t)$ does not measure these conditions accurately. Under the specified simplifying assumptions, our adjusted life expectancy measures the life expectancy implied by current mortality conditions and it is therefore not a measure of past mortality conditions.

2 Conclusion

The calculation of period life expectancy from hazard rates with conventional mortality life tables originated more than two centuries ago, when infectious diseases were the primary causes of death and life expectancy at birth in European countries was less than half of current levels. The contemporary "model" for human mortality seems never to have been made explicit, but it evidently embodied the idea of people being "struck down" by events in the environment. This model is far less relevant today than it was two centuries ago, but we are so accustomed to the rates perspective on which the period life table is based that we tend to accept it without question.

Our research into tempo effects has lead us to a thorough reconsideration of the fundamentals of mortality measurement. We take it for granted that measurement is based on some understanding of the process that generates the observed phenomena, deaths in this case. If the nature of the phenomena changes, as it has with respect to mortality, it is appropriate to reconsider whether existing measurements are still appropriate. We agree with Vaupel that, with respect to measurement of mortality, this is often not the case.

This brief note documents the mathematical relationships between longevity measures derived from the rates and conditions perspectives. We believe that the assumptions underlying the latter are applicable to senescent mortality which dominates in contemporary low mortality countries. In the conditions perspective the conventional period life expectancy gives a distorted estimate of the life expectancy implied by current mortality conditions. This tempo distortion is positive when mortality conditions are improving and negative when they are deteriorating. Most countries are currently experiencing

improvements, and their conventionally calculated period life expectancies therefore have an upward distortion.

References

Bongaarts, J. and Feeney, G. Estimating mean lifetime. *In this volume, also published in Proceedings of the National Academy of Sciences*, 100(23):13127–13133. 2003.

Bongaarts, J. and Feeney, G. The quantum and tempo of life cycle events. *In this volume, also published in Vienna Yearbook of Population Research*, pages 115–151. 2006.

Bongaarts, J. and Feeney, G. (2002). How long do we live? *Population and Development Review*, 28(1):13–29.

Guillot, M. Tempo effects in mortality: An appraisal. *In this volume, also published in Demographic Research*, 14(1):1–26. 2006.

Guillot, M. (2003). The cross-sectional average length of life (CAL): A cross-sectional mortality measure that reflects the experience of cohorts. *Population Studies*, 57(1):41–54.

Hajnal, J. (1948). Some comments on Mr Karmel's paper 'The relations between male and female reproduction rates.'. *Population Studies*, 2(3):354–360.

Rodriguez, G. Demographic translation and tempo effects: An accelerated failure time perspective. *In this volume, also published in Demographic Research*, 14(6):85–110. 2006.

Vaupel, J. Lifesaving, lifetimes and life tables. *In this volume, also published in Demographic Research*, 13(24):597–614. 2005.

Vaupel, J. (2002). Life expectancy at current rates vs. current conditions: A reflexion stimulated by Bongaarts and Feeney's "How Long Do We Live?". *Demographic Research*, 7(8):366–376.

Wachter, K. Tempo and its tribulations. *In this volume, also published in Demographic Research*, 13(9):201–222. 2005.

Turbulence in lifetables: Demonstration by four simple examples

James W. Vaupel

Max Planck Institute for Demographic Research, Konrad-Zuse-Strasse 1, 18057 Rostock, Germany. Email: jwv@demogr.mpg.de

Summary. To understand why mortality change can distort calculations of death rates and life expectancy, it is informative to consider some examples that are as simple as possible. This short chapter presents four such illustrations. They show how lifesaving can roil lifetable statistics.

1 Introduction

Demographic change can distort demographic rates. In particular, mortality change can distort period death rates, life expectancy and other lifetable statistics. This fact is considered from various perspectives in earlier chapters of this monograph, including my chapter on "Lifesaving, lifetimes and lifetables". Some demographers, however, still do not accept the surprising and uncomfortable reality. The main reason seems to be that published explanations, including those in this book, are difficult to comprehend. My chapter and my earlier article on "Life expectancy at current rates vs. current conditions" are hard to read, in large part because they were hard to write. John Bongaarts and Griff Feeney started to correspond with me about tempo effects on mortality in early 2001. It took me hundreds of hours of thinking, spread out over several years, before I finally understood the issues sufficiently well to feel confident about my comprehension and it is only recently that I have been able to explain to students why demographic change roils period rates.

As with other puzzling concepts that seem counterintuitive given our education (but eventually become intuitive when we re-educate ourselves), the best entre to comprehending mortality turbulence is to consider some examples that are as simple as possible. This short chapter presents four such illustrations.

2 Saving infant lives for one year

Consider the special case of a closed population with a constant mortality regime that has prevailed for many years. Suppose there are only four ages, ages 0, 1, 2 and 3. Each year exactly 100 individuals are born, all on January 1st. All deaths occur on July 1st, halfway through the year. The last survivors die at exact age 3.5. The first three columns of Table 1 provide statistics about this prevailing regime. Exactly 40 individuals die at age 0, 20 at age 1, 20 at age 2 and 20 at age 3. Life expectancy at birth is 1.7 years. The statistics for each period are identical to the corresponding statistics for each birth cohort. To highlight the fact that individuals age along diagonals, the population sizes and death counts for the cohorts born in years 1 and 4 are shaded light grey.

Table 1. Population statistics at different ages and times when 30 lives are saved at age 0 and extended by one year.

		Year						
Age	Statistic	1	2	3	4	5	6	7
0	e	*1.7*	*1.7*	*1.7*	**2.3**	2	2	2
	P	*100*	*100*	*100*	100	100	100	100
	D	*40*	*40*	*40*	10	10	10	10
	q	*0.4*	*0.4*	*0.4*	0.1	0.1	0.1	0.1
1	e	*1.5*	*1.5*	*1.5*	**1.5**	1.17	1.17	1.17
	P	*60*	*60*	*60*	**60**	90	90	90
	D	*20*	*20*	*20*	**20**	50	50	50
	q	*0.33*	*0.33*	*0.33*	**0.33**	0.556	0.556	0.556
2	e	*1*	*1*	*1*	1	1	1	1
	P	*40*	*40*	*40*	40	40	40	40
	D	*20*	*20*	*20*	20	20	20	20
	q	*0.5*	*0.5*	*0.5*	0.5	0.5	0.5	0.5
3	e	*0.5*	*0.5*	*0.5*	0.5	0.5	0.5	0.5
	P	*20*	*20*	*20*	20	20	20	20
	D	*20*	*20*	*20*	20	20	20	20
	q	*1*	*1*	*1*	1	1	1	1
Total deaths		100	100	100	**70**	100	100	100

Note: The new mortality regime starts in year 4. The letter e denotes remaining life expectancy, P population size, D number of deaths and q probability of death. The values in italics pertain to times prior to the mortality shift. The values in bold are discordant with subsequent values prevailing after the shift. Population sizes and death counts for the cohorts born in years 1 and 4 are shaded grey.

At the start of year 4, a new mortality regime replaces the old one. The number of deaths at age zero is reduced from 40 to 10. The 30 individuals whose lives are saved die at age one: their lives are extended by exactly one year. This is the only change in the mortality regime-but it results in changes in various statistics.

Note that the number of deaths in year 4 is 70 rather than 100. This reflects the fact that 30 lives were saved. These individuals, however, die in year 5, so in year 5 the number of deaths returns to the stationary level of 100 per year. As in year 4, the lives of 30 infants are saved in year 5 and in subsequent years, but starting in year 5 the resuscitated die, adding 30 deaths that balance the 30 lives saved.

Examine the number of age-specific deaths each year. Note that at age 0 this number falls from 40 to 10 in year 4, but that it rises at age 1 from 20 to 50 in *year* 5.

Consider the age-specific probabilities of death. Note that $q(0)$ drops from .4 to .1 in year 4 and afterwards and that $q(1)$ *rises* and does so starting in *year* 5. This is a very simple example of the "delayed gerontological failure of pediatric success": saving lives at younger ages can result in higher death rates at older ages in later years.

In the 4th year, 60 individuals are alive at the start of age 1. They are the survivors of the 100 individuals born in the 3rd year. The new mortality regime does not affect them: as in earlier years 20 of them die and the probability of death remains 0.33. Because remaining life expectancy at age 2 stays at 1.0, remaining life expectancy at age 1 stays at a level of 1.5. This is readily verified by using the formula:

$$e(1) = 0.5q(1) + (1 + e(2))(1 - q(1)) .$$

The formula is true because those who die in the year die halfway through the year, adding half a year to life expectancy, whereas those who survive get a year of life that year plus remaining life expectancy at age 2.

A similar formula can be used to calculate life expectancy at birth:

$$e(0) = 0.5q(0) + (1 + e(1))(1 - q(0)) .$$

The formula yields a life expectancy at birth of 2.3, some 0.6 years higher than the previous level of 1.7.

In year 5 and thereafter life expectancy at birth is calculated not as 2.3 but as 2.0. The value of 2.0 is the correct value for the new regime: 30 lives–30% of the number of births–were saved for one year, adding $0.3x1 = 0.3$ to the previous life expectancy of 1.7. The underlying mortality regime in year 4 is the same as in later years, but the calculated $q(1)$'s are different. The reason is that at age 1 in year 4 there were 20 deaths out of a population of 60 but in subsequent years there are 50 deaths out of a population of 90. The population size at age 1 in year 5 is 90 and the number of deaths is 50

because 30 lives were saved for one year at age 0 the year before. These saved individuals do not contribute to the death count in year 4.

This simple example is proof that mortality change can roil lifetable statistics. And the example points to the explanation. A second example helps clarify the nature of the turbulence.

3 Saving infant lives for three years

In Table 2, the mortality regime before the shift is the same as in Table 1. Now, however, the 30 lives saved at age zero are extended not by one but by three years. Note that the column of statistics for year 4 is identical for Tables 1 and 2. The same number of lives are saved–and it is this that determines the column of statistics. How long the lives are extended has no impact on year 4. Hence, as in Table 1, life expectancy at birth in year 4 in Table 2 is calculated as 2.3 years.

Lifetable statistics are also distorted in year 5. Note that the values of e, P, D and q at age 2 in year 5 are the same as they were in earlier years-even though the mortality regime changed in year 4. The underlying reason for this is that those saved in year 4 have not died yet–they first die in year 7.

As shown for years 6 and 7, life expectancy under the new regime is not 2.3 or 2.45 years but 2.6 years. Under the old regime, life expectancy was 1.7. Under the new regime, 30% of the population at age zero gains 3 years of life. Hence the new life expectancy is 1.7 plus .3 times 3, or 2.6. The erroneous values of 2.3 and 2.45 calculated in years 4 and 5 are simply a result of the fact that 30 deaths were averted in year 4 and it is not known, in years 4 and 5, when these individuals will die. In year 6 and subsequently, it is known-they will die at age 3. Hence the true life expectancy can be calculated.

4 Saving infant lives for two years on average

In Table 3, some 30 lives are also saved starting in year 4, but now 10 of them are extended by one year, 10 by two years and 10 by three years. As in Tables 1 and 2, life expectancy at birth in year 4 is computed as 2.3, but now this turns out to be the correct value. The age-specific population sizes and death counts do not reach a new equilibrium until year 5 at age 1, year 6 at age 2 and year 7 at age 3. The probabilities of death, however, that are implied by these numbers are the correct values and so are the resulting calculations of age-specific life expectancies.

The reason is as follows. In computing lifetables using standard methods, it is assumed that all persons have the same life chances. If lives are saved, then it is assumed that those saved get the same life chances as those who would have survived anyway. In Tables 1, 2, and 3, the individuals who survived to age 1 under both the old and new regimes faced probabilities of death such

Table 2. Population statistics at different ages and times when 30 lives are saved at age 0 and extended by three years.

		Year						
Age	Statistic	1	2	3	4	5	6	7
0	e	*1.7*	*1.7*	*1.7*	**2.3**	**2.45**	2.6	2.6
	P	*100*	*100*	*100*	100	100	100	100
	D	*40*	*40*	*40*	10	10	10	10
	q	*0.4*	*0.4*	*0.4*	0.1	0.1	0.1	0.1
1	e	*1.5*	*1.5*	*1.5*	**1.5**	**1.67**	1.17	1.17
	P	*60*	*60*	*60*	**60**	90	90	90
	D	*20*	*20*	*20*	20	20	20	20
	q	*0.33*	*0.33*	*0.33*	**0.33**	0.222	0.222	0.222
2	e	*1*	*1*	*1*	**1**	**1**	1.214	1.214
	P	*40*	*40*	*40*	**40**	**40**	70	70
	D	*20*	*20*	*20*	**20**	**20**	20	20
	q	*0.5*	*0.5*	*0.5*	**0.5**	**0.5**	0.286	0.286
3	e	*0.5*	*0.5*	*0.5*	0.5	0.5	0.5	0.5
	P	*20*	*20*	*20*	**20**	**20**	**20**	50
	D	*20*	*20*	*20*	**20**	**20**	**20**	50
	q	*1*	*1*	*1*	1	1	1	1
Total deaths		100	100	100	**70**	**70**	**70**	100

Note: The new mortality regime starts in year 4. The letter e denotes remaining life expectancy, P population size, D number of deaths and q probability of death. The values in italics pertain to times prior to the mortality shift. The values in bold are discordant with subsequent values prevailing after the shift. Population sizes and death counts for the cohorts born in years 1 and 4 are shaded grey.

that a third of them died at age 1, a third at age 2 and a third at age 3. In Table 3, the individuals whose lives were saved at age 0 got exactly the same life chances–a third of them die at age 1, a third at age 2 and a third at age 3. But in Tables 1 and 2 the resuscitated face different mortality schedules than those not saved. In Table 1 they have a 100% chance of death at age 1; in Table 3 they have a 100% chance of death at age 3.

5 Saving everyone's life for one year

How is "turbulence in lifetables" illustrated above related to "tempo effects on mortality"? If tempo effects are broadly defined as distortions arising from

Table 3. Population statistics at different ages and times when 30 lives are saved at age 0, with 10 extended by one year, 10 by two years and 10 by three years.

		Year						
Age	Statistic	1	2	3	4	5	6	7
0	e	*1.7*	*1.7*	*1.7*	2.3	2.3	2.3	2.3
	P	*100*	*100*	*100*	100	100	100	100
	D	*40*	*40*	*40*	10	10	10	10
	q	*0.4*	*0.4*	*0.4*	0.1	0.1	0.1	0.1
1	e	*1.5*	*1.5*	*1.5*	1.5	1.5	1.5	1.5
	P	*60*	*60*	*60*	**60**	90	90	90
	D	*20*	*20*	*20*	**20**	30	30	30
	q	*0.33*	*0.33*	*0.33*	0.33	0.33	0.33	0.33
2	e	*1*	*1*	*1*	1	1	1	1
	P	*40*	*40*	*40*	**40**	**40**	60	60
	D	*20*	*20*	*20*	**20**	**20**	30	30
	q	*0.5*	*0.5*	*0.5*	0.5	0.5	0.5	0.5
3	e	*0.5*	*0.5*	*0.5*	0.5	0.5	0.5	0.5
	P	*20*	*20*	*20*	**20**	**20**	**20**	30
	D	*20*	*20*	*20*	**20**	**20**	**20**	30
	q	*1*	*1*	*1*	1	1	1	1
Total deaths		100	100	100	**70**	**80**	**90**	100

Note: The new mortality regime starts in year 4. The letter e denotes remaining life expectancy, P population size, D number of deaths and q probability of death. The values in italics pertain to times prior to the mortality shift. The values in bold are discordant with subsequent values prevailing after the shift. Population sizes and death counts for the cohorts born in years 1 and 4 are shaded grey.

changes over time in death rates, then the two phrases are synonymous. Bongaarts and Feeney introduced the concept of tempo effects on mortality with a more specific meaning: tempo effects result from changes in mortality that uniformly extend (or shorten) everyone's remaining lifespan by the same amount. This is analogous to their concept of tempo effects on fertility and is in keeping with the meaning of tempo in music. Bongaarts and Feeney's idea is so interesting and stimulating that it seems appropriate to use "tempo effects on mortality" to describe it. Table 4 provides a simple illustration, albeit a crude one because Bongaarts and Feeney hypothesize that tempo effects occur only at adult ages.

In year 4 everyone's life is extended by one year. Those who would have died at age 0 now die age 1 (one year later). Similarly those who would have died at age 1, 2 or 3 now die at age 2, 3 or 4–again, one year later. Because no one dies in year 4, life expectancy is infinite. Actually, however, life expectancy has been extended exactly one year–from 1.4 years to 2.4 years.

Table 4. Population statistics at different ages and times when all lives at all ages are saved and extended by one year.

		Year						
Age	Statistic	1	2	3	4	5	6	7
0	e	*1.7*	*1.7*	*1.7*	∞	2.7	2.7	2.7
	P	*100*	*100*	*100*	100	100	100	100
	D	*40*	*40*	*40*	0	0	0	0
	q	*0.4*	*0.4*	*0.4*	0	0	0	0
1	e	*1.5*	*1.5*	*1.5*	∞	1.7	1.7	1.7
	P	*60*	*60*	*60*	**60**	100	100	100
	D	*20*	*20*	*20*	**0**	40	40	40
	q	*0.33*	*0.33*	*0.33*	**0**	0.4	0.4	0.4
2	e	*1*	*1*	*1*	∞	1.5	1.5	1.5
	P	*40*	*40*	*40*	**40**	60	60	60
	D	*20*	*20*	*20*	**0**	20	20	20
	q	*0.5*	*0.5*	*0.5*	**0**	0.33	0.33	0.33
3	e	*0.5*	*0.5*	*0.5*	∞	1	1	1
	P	*20*	*20*	*20*	**20**	40	40	40
	D	*20*	*20*	*20*	**0**	20	20	20
	q	*1*	*1*	*1*	**0**	0.5	0.5	0.5
4	e					0.5	0.5	0.5
	P					20	20	20
	D					20	20	20
	Q					1	1	1
Total deaths		100	100	100	**0**	100	100	100

Note: The new mortality regime starts in year 4. The letter e denotes remaining life expectancy, P population size, D number of deaths and q probability of death. The values in italics pertain to times prior to the mortality shift. The values in bold are discordant with subsequent values prevailing after the shift. Population sizes and death counts for the cohorts born in years 1 and 4 are shaded grey.

6 Discussion

These four simple examples demonstrate how and why mortality change can distort lifetable statistics. Table 1 shows that if those whose lives are saved gain less additional life expectancy than those whose lives are not saved, then conventional calculations can overestimate true life expectancy. Table 2 shows that if the resuscitated gain more than the remaining life expectancy of those not saved, then conventional calculations can underestimate true life expectancy. Table 3 shows that if the remaining life chances (i.e., future age-specific chances of death) of the resuscitated and the non-resuscitated are the same, then conventional computations result in correct estimates of life expectancy and of age-specific death rates. Finally Table 4 shows that if mortality improvements extend everyone's life by the same amount, a tempo effect, then conventional calculations can result in overestimates of life expectancy. This is explained in detail in my earlier chapter in this volume, but the general point is more clearly illustrated by the simple examples of this chapter.

Note that turbulence in lifetables does not depend on a mental model in which the lifespans of individuals are predetermined. The lifetables in this chapter could have been described in terms of random hazards of death and I occasionally used this perspective in the explanations above. The crucial question is: what are the age-specific chances of death for those whose lives have been saved as a result of improvements in death rates? If their chances are the same as the chances for those not saved, then conventional calculations are correct. If, however, they have different chances, then conventional lifetables are distorted until it is learned, with the passage of time, when the resuscitated die.

Conventional calculations rely on the assumption of homogeneity in life chances for everyone, regardless of whether their lives were saved or not. Bongaarts and Feeney make a very different kind of homogeneity assumption: they postulate that mortality improvements extend everyone's life by the same amount. All populations are heterogeneous and the resuscitated undoubtedly differ from the non-resuscitated and from each other in their remaining life chances. Hence, both conventional calculations and the tempo assumption are wrong. The truth probably lies somewhere in-between.

Some readers may object to my using the phrase "true life expectancy". I define it as the average length of life for a synthetic cohort of newborns who live all their lives under the current mortality regime. An alternative definition would be the average length of life calculated from current age-specific death rates. The second definition, by definition, implies that conventional calculations are correct. This, as I hope the four Tables illustrate, is not an intelligent mode of thinking. The mortality regime in year 4 is the same as thereafter, but life expectancy at birth, in Tables 1, 2 and 4, differs in year 4 from subsequent levels. The distortion is an artifact of mortality change: it does not reflect any underlying differences in mortality regime.

Whether or not tempo effects exist in some age range, at some time periods and in some countries is an open question that merits further research. Whether or not mortality change produces turbulence in lifetables is no longer a question. Research is needed, however, on how much life is extended when a death is averted. The answer surely varies over age, time and place. Only when better knowledge about this is available, will demographers be able to correctly estimate life expectancy and other lifetable statistics in a world of changing mortality.

Acknowledgements

I am grateful to Elisabetta Barbi, Annette Baudisch, John Bongaarts and Joshua Goldstein for constructive suggestions.

Appendix

Two proofs of a recent formula by Griffith Feeney*

Jutta Gampe[1] and Anatoli Yashin[2]

[1] Max Planck Institute for Demographic Research, Konrad-Zuse-Strasse 1, 18057 Rostock, Germany. E-mail: gampe@demogr.mpg.de
[2] Center for Population Health and Aging, Duke University, Box 90408 002 Trent Hall, Durham, NC 27708, USA. E-mail: aiy@duke.edu

1 Introduction

In his chapter on increments to life and mortality tempo Feeney gives the following decomposition of the difference between the expectations of life at birth for two cohorts (see also equation (1) on page 154), but without mathematical proof:

$$e_0^c(t_2) - e_0^c(t_1) = -\int_0^\infty \lambda_c^{t_1,t_2}(x)\, dl_c(x,t_1), \tag{1}$$

Since the correctness of this formula was contested during the reviewing process, the editors decided to include the following brief proofs. They are equivalent, but they look different and each may be useful for a different group of readers.

2 Proof by Jutta Gampe

The function $\lambda(x)$ described by Feeney, is given formally as

$$\lambda(x) = l_2^{-1}\left[l_1(x)\right] - x \tag{2}$$

when we drop some obvious subscripts and implicitly assume that everything is invertible etc. Equation (1) can be written either as

$$e_0^2 - e_0^1 = \int_0^\infty \left\{l_2(x) - l_1(x)\right\}\, dx$$

or as

$$= \int_0^1 \left\{ l_2^{-1}(p) - l_1^{-1}(p) \right\} \, dp.$$

A change of variables $p \to l_1(x)$ leads to

$$\int_0^\infty \left\{ l_2^{-1}\left[l_1(x) \right] - x \right\} f_1(x) \, dx = \int_0^\infty \lambda(x) f_1(x) \, dx,$$

with $dl_1(x)/\, dx = -f_1(x)$. The latter integral equals

$$- \int_0^\infty \lambda(x) \, dl_1(x)$$

3 Proof by Anatoli Yashin

The condition $l_2(x + \lambda(x)) = l_1(x)$ is equivalent to the condition that the random variables T_2 and $T_1 + \lambda(T_1)$ are identically distributed, hence $ET_2 = ET_1 + E\lambda(T_1)$, which is equation (1).

References

Feeney, G. Increments to life and mortality tempo. *In this volume, also published in Demographic Research*, 14(2):27–46. 2006.