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Research Article

Tempo effects in mortality: An appraisal

Michel Guillot

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a research article

Tempo effects in mortality: An appraisal

Michel Guillot[1]

Abstract

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 paper 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

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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 paper, 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 TFRs 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 TFRs. Tempo effects thus pose a challenge for the interpretation of levels and trends in period TFRs.

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, 2003, 2005). 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 paper, 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, 2003). 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:

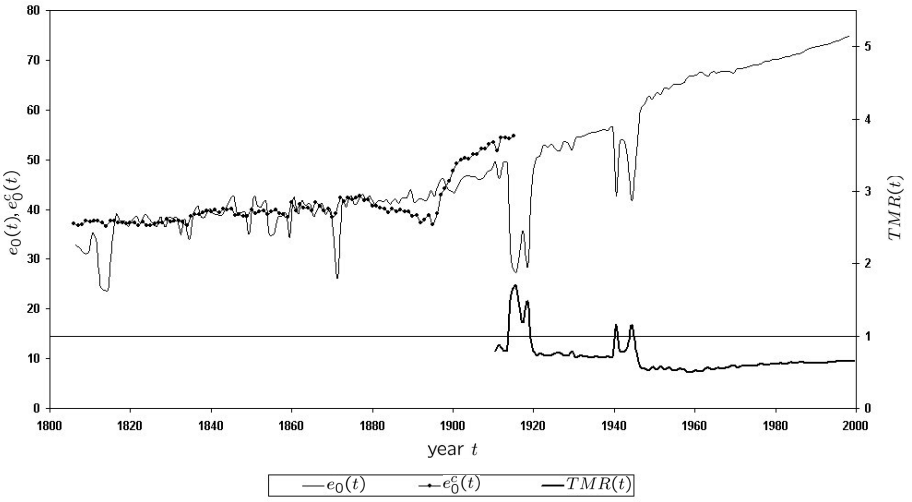
$$TMR(t) = \int_0^\infty d_c(x,t-x)dx \tag{2}$$

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. (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, TMRs 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

Figure 1: Period life expectancy, $\mathbf{e_0(t)}$; cohort life expectancy, $\mathbf{e_0^c(t)}$; and period total mortality rate, $\mathbf{TMR(t)}$. France, males, 1806-1998.

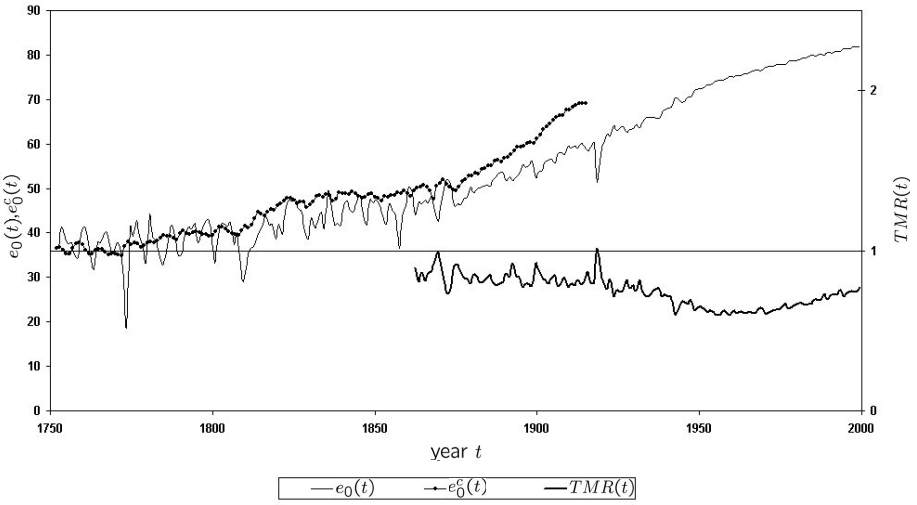


Note: Data source: Vallin-Meslé database. http://www.ined.fr/publications/cdrom_vallin_mesle/contenu.htm
Note: $e_0^c(t)$ is plotted at time when the cohort was born.

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,

Figure 2: Period life expectancy, $\mathbf{e_0(t)}$; cohort life expectancy, $\mathbf{e_0^c(t)}$; and period total mortality rate, $\mathbf{TMR(t)}$. Sweden, females, 1752-1998.



Data source: Human Mortality Database. www.mortality.org.

Note: $e_0^c(t)$ is plotted at the time (t) when the cohort was born.

there are a few years – the WWI years – during which period e_0 levels have no relevance for any particular 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.

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, 2003; Schoen and Canudas-Romo, 2005; Goldstein, 2006).

Bongaarts and Feeney (referred to as BF in the remainder of the paper) 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 proportion of cohort survivors aged x at time t.

$$MAD(t) = \frac{\int_0^\infty x \cdot d_c(x, t-x)dx}{\int_0^\infty d_c(x, t-x)dx} \tag{4}$$

$$M_4(t) = \int_0^\infty \exp\left\{-\int_0^x \frac{\mu(a,t)}{TMR(t)}da\right\}dx \tag{5}$$

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, 2003, 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 paper, Bongaarts and Feeney (2003) 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:

$$\mu(x,t) = p(t)\nu^*(x,t) \tag{7}$$

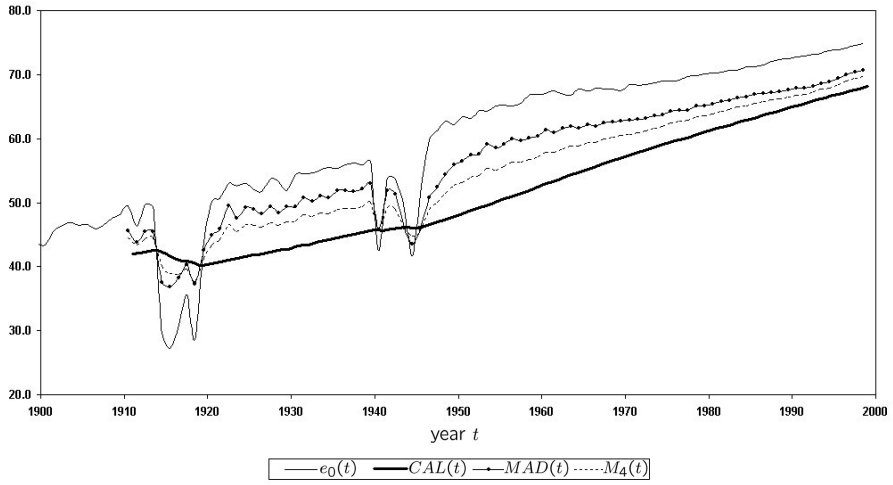
They refer to this assumption as the "proportionality" assumption, and claim that this assumption is a good approximation of the current situation in Sweden, France and the US. As Wachter demonstrates in this volume, one situation which approximately produces proportionality is when all cohorts experience a Gompertz force of mortality and a constant, age-invariant rate of decline in age-specific death rates (Wachter, 2005). 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.

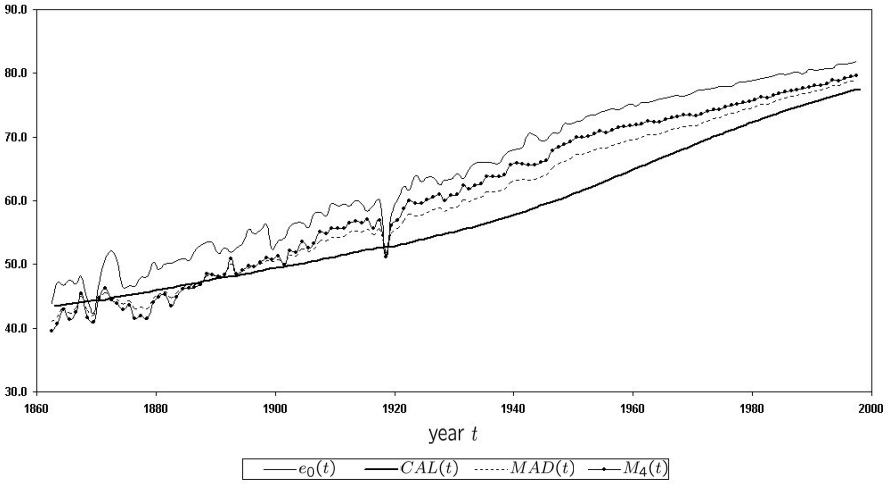
Figures 3 and 4 show that among French males and Swedish females, there are important differences between the three indicators. Typically, CAL has the lowest value, MAD has the highest value, and M_4 is somewhere in between. The difference between CAL and MAD is as large as 9.46 years in 1953 in France. 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.

Figure 3: Period life expectancy, $\mathbf{e_0(t)}$; cross-sectional average length of life, $\mathbf{CAL(t)}$; mean age at death in the constant-birth population, $\mathbf{MAD(t)}$; and Bongaarts and Feeney's $\mathbf{M_4(t)}$. France, males, 1900-1998.



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

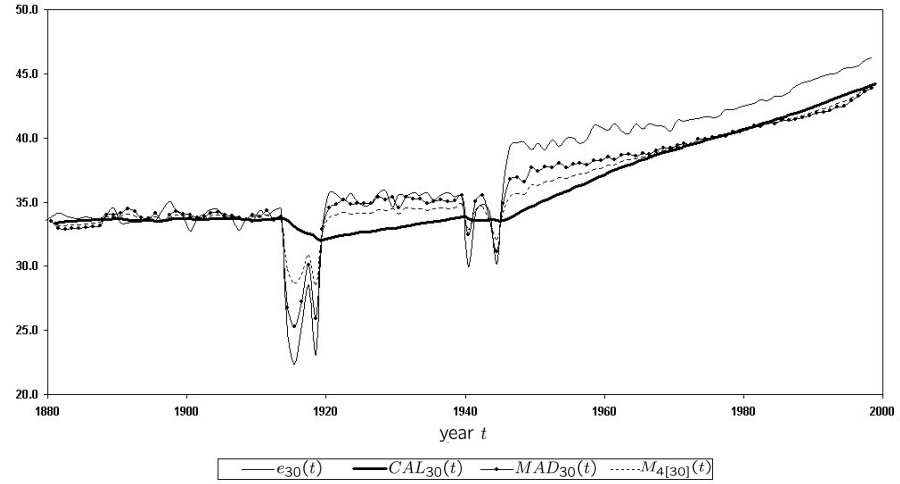
Figure 4: Period life expectancy, $\mathrm{e_0(t)}$; cross-sectional average length of life, $\mathrm{CAL(t)}$; mean age at death in the constant-birth population, $\mathrm{MAD(t)}$; and Bongaarts and Feeney's $\mathrm{M_4(t)}$. Sweden, females, 1862-1998.



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). 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.)

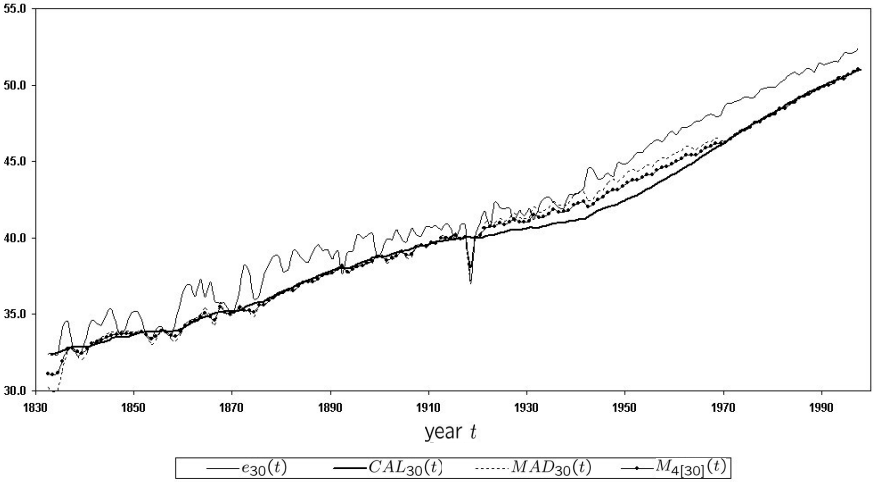
Figure 5: Period life expectancy, $\mathbf{e_{30}(t)}$; cross-sectional average length of life, $\mathbf{CAL_{30}(t)}$; mean age at death in the constant-birth population, $\mathbf{MAD_{30}(t)}$; and Bongaarts and Feeney's $\mathbf{M_{4[30]}(t)}$. France, males, 1880-1998.



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.

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. 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.)

Figure 6: Period life expectancy, $\mathbf{e_{30}(t)}$; cross-sectional average length of life, $\mathbf{CAL_{30}(t)}$; mean age at death in the constant-birth population, $\mathbf{MAD_{30}(t)}$; and Bongaarts and Feeney's $\mathbf{M_{4[30]}(t)}$. Sweden, females, 1832-1998.



Data source: Human Mortality Database. www.mortality.org.

Note: $e_0^c(t)$ is plotted at the time (t) when the cohort was born.

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 paper) 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.

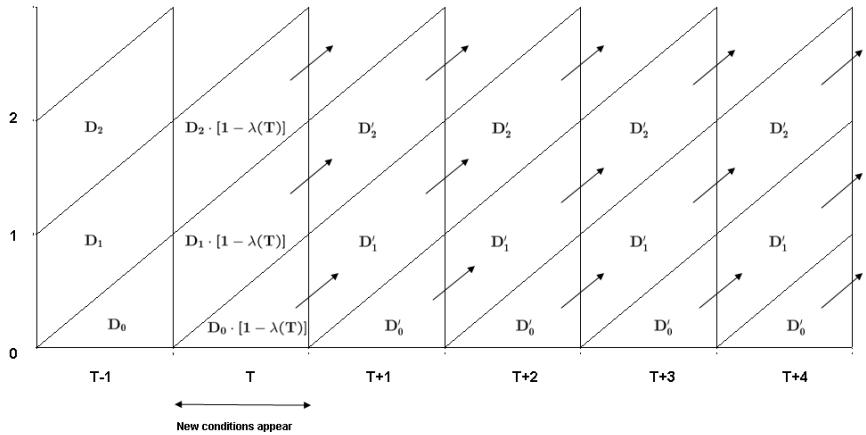
As in the classic approach, BF assume that populations are homogeneous with respect to the risk of death, but they address mortality change differently. They define period mortality changes in terms of changes overtime in the $p_c(x, t-x)$ curve. According to them, a change in mortality conditions during 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 a Lexis diagram in Figure 7. 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 (2005) in this volume 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, gen-

erating 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.

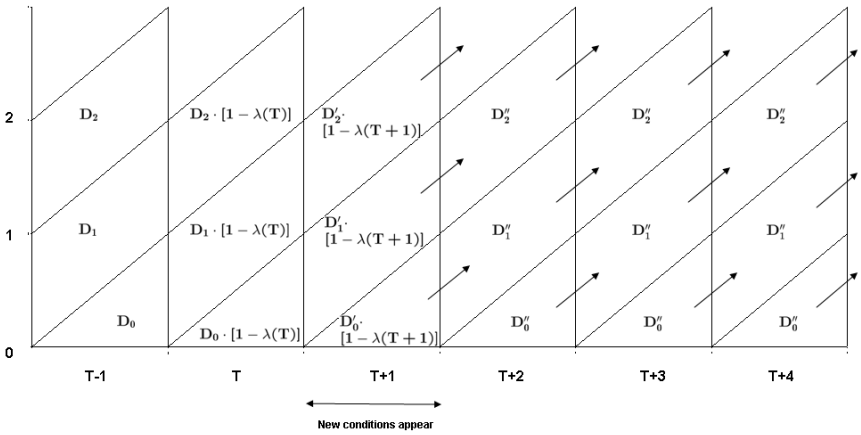
Figure 7: Lexis diagram illustrating Bongaarts and Fenney's scenario of mortality change.
Diagram A: New conditions appear at time T:



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 $\lambda(T)$ 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 $T-1$. $D'_x = D_x \cdot [1-\lambda(T)] + D_{x-1} \cdot \lambda(T)$

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.

Figure 7 (continued): Diagram B: New conditions appear at time T + 1



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). $D''_x = D'_x \cdot [1-\lambda(T+1)] + D'_{x-1} \cdot \lambda(T+1)$

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 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, 2003, p.53):

$$TMR(t) = 1 - \frac{dCAL(t)}{dt} \tag{8}$$

(Note that unlike BF's similar equation (Bongaarts and Feeney, 2003, p.13129, 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 (Feeney, 2006)). 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, 2003, 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 $p(x,t) = p_c(x,t-x) = 0$.

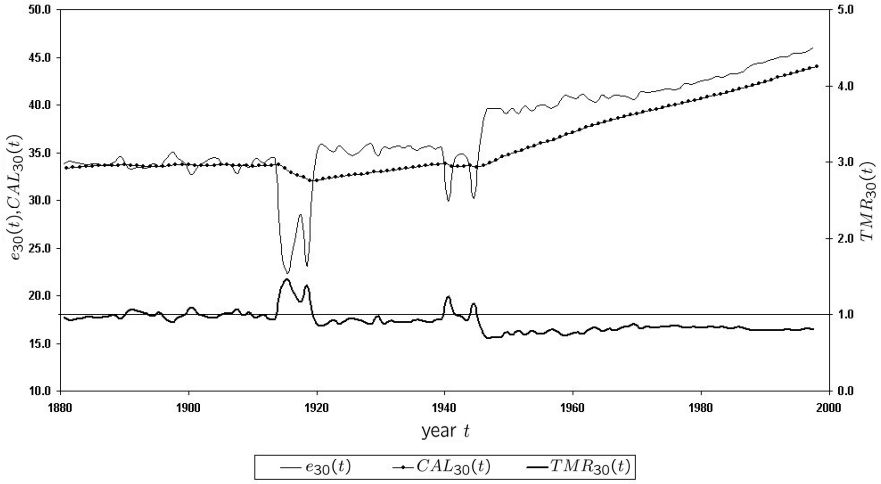
Under steady mortality decline, $p(x,t)$ tends to be greater than $p_c(x,t-x)$, and CAL tends to increase. In fact, if $p(x,t) \neq p_c(x,t-x)$ for any x in the interval $(0,\omega)$ (which happens for most years in France and Sweden), the direction of the change in CAL will be determined by the sign 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)).

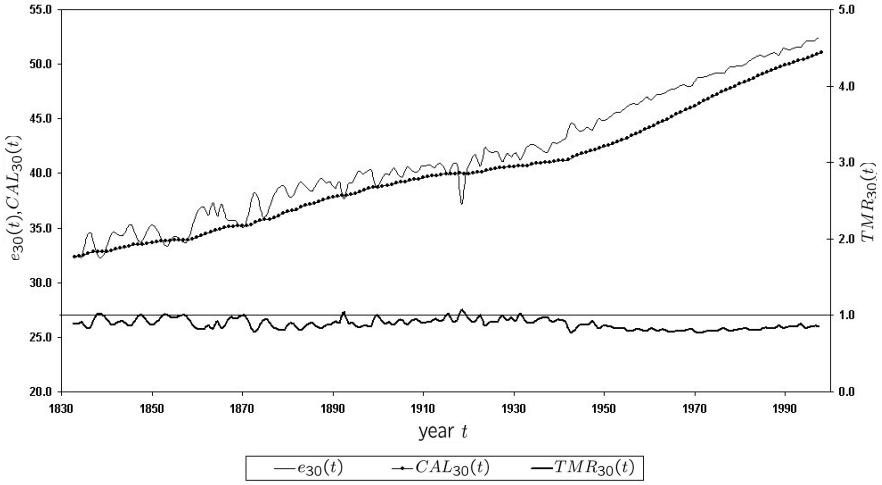
Figure 8: Period life expectancy, $\mathbf{e_{30}(t)}$; cross-sectional average length of life, $\mathbf{CAL_{30}(t)}$; and period total mortality rate, $\mathbf{TMR_{30}(t)}$. France, males, 1880-1998.



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 what-

Figure 9: Period life expectancy, $\mathbf{e_{30}(t)}$; cross-sectional average length of life, $\mathbf{CAL_{30}(t)}$; and period total mortality rate, $\mathbf{TMR_{30}(t)}$. Sweden, females, 1832-1998.



ever 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 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 oc-

currence 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 paper 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 paper 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 compo-

sition 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 Russia 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.

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References

Arthur, W., & Vaupel, J. (1984). Some general relationships in population dynamics. *Population Index, 50*(2), 214-226.

Avdeev, A., Blum, A., Zakharov, S., & 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., & Feeney, G. (1998). On the quantum and tempo of fertility. *Population and Development Review*, *124*(2), 271-291.

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

Bongaarts, J., & Feeney, G. (2003). Estimating mean lifetime. *Proceedings of the National Academy of Sciences*, *100*(23), 13127-13133.

Bongaarts, J., & Feeney, G. (2005). The quantum and tempo of life-cycle events. *Policy Research Division Working Paper,* New York City: Population Council, *207*. (http://www.popcouncil.org/)

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., & Preston, S. (1992). Effects of early-life conditions adult mortality: a review. *Population Index, 58*(2), 186-212.

Feeney, G. (2006). Increments to life and mortality tempo. Forthcoming Volume 14, *Demographic Research*. (http://www.demographic-research.org)

Goldstein, J. (2003). Late but not never: The tempo and quantum of first marriage in france. Submitted for publication.

Goldstein, J. (2006). Found in translation. Forthcoming Volume 14, *Demographic Research*. (http://www.demographic-research.org)

Guillot, M. (1999). The period average life (PAL). Paper presented at the 1999 PAA meeting, New York City.

Guillot, M. (2003a). 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. (2003b). 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. (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., & J.A.Ortega. (2002). Tempo-adjusted period parity progression measures, fertility postponement and completed cohort fertility. *Demographic Research*, *6*(6), 92-144. (http://www.demographic-research.org/volumes/vol6/6/)

Lièvre, A., Brouard, N., & 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., & 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, 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., & 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. (2005). Lifesaving, lifetimes and lifetables. *Demographic Research*, *13*(24), 597-614. (http://www.demographic-research.org/volumes/vol13/24/)

Vaupel, J., Manton, K., & Stallard, E. (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography*, *16*(3), 439-454.

Wachter, K. (2005). Tempo and its tribulation. *Demographic Research*, *13*(9), 201-222. (http://www.demographic-research.org/volumes/vol13/9/)

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., & 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., & 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.