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On the Decomposition of Changes in Expectation of Life and Differentials in Life Expectancy

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The projection of mortality rates requires inter alia close examination of the mortality experience of a population over a long period of time and will usually also involve the analysis of mortality trends by cause of death. In two of the more important recent contributions, techniques were devised for explaining change in life expectancy in terms of mortality changes in particular age groups and by different causes of death. The approaches adopted by the authors differ, and the purpose of this article is to reconcile the two and tie the results in with those obtained by earlier writers. A new method for explaining the change in a life expectancy differential in terms of the observed changes in the mortality differentials and the observed change in overall mortality level is also described.

The expectation of life $\mathring{e}_x$ and the temporary expectation of life ${}_n\mathring{e}_x$ are often used as convenient summary measures of the mortality of a population. Changes in life expectancies are also used for summarizing changes in the mortality of a population. There are good reasons for adopting such summary measures. Apart from the fact that a single index such as $\mathring{e}_x$ or a small number of indices such as ${}_{10}\mathring{e}_0$, ${}_{55}\mathring{e}_{10}$, and $\mathring{e}_{65}$ condense the information in a full mortality schedule considerably, life expectancies have the advantage of ready interpretation. Even an expert in the mortality field has difficulty interpreting the meaning of an improvement in the mortality rate at age 30 of, say, 0.0023. On the other hand, both expert and layman have some feeling for an improvement in expectation of life at birth of, say, 0.2 years.

The wider use of expectation of life and temporary life expectancies for summarizing mortality levels and trends has therefore been advocated in a number of papers in recent years (Arriaga, 1982, 1984; Pollard, 1982b). The connection between expectation of life and the mortality rate at a particular age, however, is not a particularly simple one. In two more recent papers (Arriaga, 1984; Pollard, 1982a), techniques were devised for explaining change in life expectancy in terms of mortality changes in particular age groups and by different causes of death. These are not the first attempts at the decomposition of change in life expectancy. An earlier analysis, for example, is United Nations (1982). The approaches adopted by the recent authors differ, and the main purpose of this article is to reconcile the two.

The continuous approach of Pollard (1982a), summarized in the next section, partitions the change in expectation of life as a result of mortality changes into various components: the main effects of the mortality changes and the interaction terms of various orders. These interaction terms play important roles in the reconciliation of this continuous model and the discrete analysis of Arriaga (1984) in the third section.

A generalization of the continuous analysis is summarized in the fourth section and is then used in the following section to derive a formula for analyzing a change in an

expectation of life differential in terms of the changed mortality differentials and the changed overall level of mortality. The results of Lopez and Ruzicka's (1977) and Retherford's (1972) analyses of trends in expectation of life differentials are explained in terms of this new continuous method.

The Continuous Approach

Pollard (1982a) showed that the change in expectation of life of a population between time 1 and time 2 can be written in the following form:

$$\mathring{e}_0^2 - \mathring{e}_0^1 = \int_0^\infty (\mu_t^1 - \mu_t^2) \exp\left\{\int_0^t (\mu_u^1 - \mu_u^2)\, du\right\} {}_tp_0^1 \mathring{e}_t^1\, dt, \tag{1}$$

where the suffixes 1 and 2 on the standard life table functions refer to times 1 and 2. The same formula can also be used to analyze mortality differences between cohorts and to explain differentials between populations (e.g., sex differentials), with the suffixes then representing the different cohorts or populations.

If the exponential term in equation (1) is expanded in terms of the powers of the integral,

$$\int_0^t (\mu_u^1 - \mu_u^2)\, du, \tag{2}$$

then the main effects of the mortality changes at the various ages on the change in expectation of life at birth and the interactions of various orders may be discerned. The main effects come from the first term in the expansion and are in fact those enumerated in the well-known approximate formula

$$\mathring{e}_0^2 - \mathring{e}_0^1 \doteqdot \int_0^\infty (\mu_t^1 - \mu_t^2) {}_tp_0^1 \mathring{e}_t^1\, dt. \tag{3}$$

The *jth*-order interactions are given by

$$\frac{1}{j!} \int_0^\infty \left[\int_0^t (\mu_u^1 - \mu_u^2)\, du\right]^j (\mu_t^1 - \mu_t^2) {}_tp_0^1 \mathring{e}_t^1\, dt, \tag{4}$$

a result quoted in Pollard (1982a) and proved in an appendix in Pollard (1986a).

When changes in mortality are all improvements, the interaction term (4) also has another interesting interpretation. According to Vaupel and Yashin (1987), lives surviving under a new improved life table can be subdivided into those who have been saved 0, 1, 2, . . . , times as a result of the improved mortality rates. The expected lifetime in the "saved $j + 1$ times" category can be shown to be equal to the interaction term (4) (Pollard, 1986a).

Except in cases of populations that have extremely low (under 55 years, say) initial expectations of life and then experience extremely large improvements in mortality, the proportion of the total change in expectation of life due to interactions is relatively small (Pollard, 1982a). Interaction terms are also comparatively difficult to compute and not easy to interpret. It is easy to deduce from equation (1) that the change in expectation of life can also be written *exactly* as

$$\mathring{e}_0^2 - \mathring{e}_0^1 = \int_0^\infty (\mu_t^1 - \mu_t^2) {}_tp_0^2 \mathring{e}_t^1\, dt \tag{5}$$

and, by interchanging suffixes and changing signs, as

$$\mathring{e}_0^2 - \mathring{e}_0^1 = \int_0^\infty (\mu_t^1 - \mu_t^2)_t p_0^1 \mathring{e}_0^2 \, dt. \tag{6}$$

The following *exact* formula, which combines the relatively small interaction terms with the main effects and is simple to use, was therefore proposed:

$$\mathring{e}_0^2 - \mathring{e}_0^1 = \int_0^\infty (\mu_t^1 - \mu_t^2) w_t \, dt, \tag{7}$$

with weight

$$w_t = \tfrac{1}{2}({}_t p_0^1 \mathring{e}_t^2 + {}_t p_0^2 \mathring{e}_t^1), \tag{8}$$

which is almost linear over most of the age range (Pollard, 1982a).

According to the usual model, the force of mortality at a particular age is the sum of the forces of mortality at that age for the various causes of death. Distinguishing n distinct causes of death and denoting the force of mortality by cause i at age t at time j by $\mu_t^j(i)$, it is deduced from equation (7) that

$$\mathring{e}_0^2 - \mathring{e}_0^1 = \sum_{i=1}^{n} \int_0^\infty [\mu_t^1(i) - \mu_t^2(i)] w_t \, dt. \tag{9}$$

This formula allows the contributions of the various causes of death to the change in expectation of life (or life expectancy differential) to be distinguished by age in a two-way table. For purposes of evaluation, observed central mortality rates are substituted for forces of mortality and the weights are calculated at the midpoint of each age range. Examples of the use of this technique are shown in Table 1. Age intervals of 5 years were used in the calculations for all ages except the under-1-year group, the 1–4 age group, and the open-ended over-85 group. The technique allows the effects of mortality changes by age and cause of death to be readily discerned.

A limitation of formula (9) is that the weighting factor $\{w_t\}$ for a particular cause is itself a function of the level of mortality from the various causes of death in the two populations, including the cause itself. For most descriptive and analytic purposes, this poses no problems, but difficulties can arise when the formula is employed as a tool within a much more complicated and detailed analysis.

The Discrete Approach

The approach adopted by Arriaga (1984) involves discrete age groups. Let us assume that these are $(0, x)$, (x, y), (y, z), and (z, ∞), with $0 < x < y < z < \infty$. Changes in the mortality rates in the age range $(0, x)$ will result in changes in the number of years of life lived between ages 0 and x and hence affect $\mathring{e}_0$. Arriaga referred to this effect on $\mathring{e}_0$ as the *direct effect* of the mortality change in the $(0, x)$ age range. There is also an *indirect effect* on $\mathring{e}_0$ as a result of the mortality change in the $(0, x)$ age range: even if there are no mortality changes beyond age x, the number of years of life lived beyond age x will be altered because the number of lives reaching the start of the (x, ∞) range will have changed.

In a similar manner, a direct effect and an indirect effect on $\mathring{e}_0$ can be calculated with respect to mortality changes in the age range (x, y) in the absence of mortality changes below age x or beyond y. Formulas for these effects are given in Arriaga (1984).

Table 1. Australia, Expectation of Life at Birth: Contributions of the Various Causes of Death to the Change in Expectation of Life Between 1971 and 1981 and to the 1981 Sex Differential (in hundredths of a year of life)

Code, cause	Males, 1971/1981								Females, 1971/1981								Males/Females, 1981							
	Age group								Age group								Age group							
	0	1–4	5–14	15–29	30–49	50–69	70+	All ages	0	1–4	5–14	15–29	30–49	50–69	70+	All ages	0	1–4	5–14	15–29	30–49	50–69	70+	All ages
1, infective, parasitic	7	3	0	0	1	0	0	12	5	2	1	0	0	0	0	9	−1	0	0	0	0	0	0	1
2, neoplasm, excluding 3, 4, 5	0	0	1	1	0	−2	−4	−3	0	1	1	2	2	4	−3	8	0	1	1	3	4	18	30	55
3, neoplasms, digestive	0	0	0	0	0	0	2	1	0	0	0	0	0	4	4	9	0	0	0	0	2	20	15	37
4, neoplasm, respiratory	0	0	0	0	0	0	−4	−5	0	0	0	0	0	−4	−2	−6	0	0	0	0	5	43	30	77
5, neoplasms of breast	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	3	0	0	0	0	−11	−21	−9	−42
6, endocrine disease	1	0	0	0	1	2	1	7	1	1	0	0	1	4	5	12	0	0	0	0	0	3	2	4
7, blood, mental, sense	0	1	0	−2	2	3	0	3	0	−1	0	0	4	2	−1	5	1	−1	0	4	1	4	4	13
8, circulatory, excluding 9, 10	0	0	0	0	4	6	13	22	0	0	0	1	5	10	31	46	0	0	0	0	4	19	16	40
9, ischaemic heart disease	0	0	0	0	19	70	33	122	0	0	0	0	6	41	55	101	0	0	0	0	25	133	86	244
10, cerebrovascular disease	0	0	0	0	3	17	21	40	0	0	0	0	8	24	47	80	0	0	0	0	1	12	6	18
11, respiratory disease	16	2	1	0	3	5	5	32	13	2	0	1	2	4	7	30	0	0	0	0	0	4	8	14
12, bronchitis, emphysema, asthma	0	0	0	0	0	7	3	11	0	0	0	0	1	−4	−3	−5	0	0	0	0	1	17	38	54
13, digestive, excluding 14	1	0	0	0	0	1	−1	2	1	0	0	0	2	1	−2	2	0	0	0	0	2	4	3	10
14, cirrhosis of liver	0	0	0	0	0	−3	0	−5	0	0	0	0	0	−1	0	−1	0	0	0	0	5	10	1	16
15, genito-urinary system	0	0	0	0	1	3	2	8	0	0	0	0	5	6	0	13	0	0	0	0	−1	−1	3	2
16, pregnancy, childbirth	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	2	0	0	0	−1	0	0	0	−1
17, skin, tissue disease	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	3	0	0	0	0	0	0	0	−1
18, congenital anomalies	48	2	0	0	0	0	0	51	46	1	1	0	0	0	0	50	12	0	0	0	0	0	0	13
19, ill-defined conditions	−13	−1	0	0	0	0	0	−10	−9	0	0	0	0	0	2	−5	5	0	0	0	0	0	0	8
20, external, excluding 21, 22	3	2	2	3	5	3	1	22	3	2	0	2	0	2	5	15	1	3	2	14	14	6	2	42
21, motor vehicle accidents	0	1	2	14	6	4	1	29	0	1	1	3	1	3	0	10	0	0	3	36	13	4	3	60
22, suicide, self-inflicted	0	0	0	0	2	1	0	3	0	0	0	2	5	2	0	11	0	0	0	12	10	5	1	29
Total, all causes	63	10	7	22	47	121	74	342	60	10	6	17	50	102	149	394	20	3	8	70	76	276	240	693

Source: Pollard (1986a).

In the notation of the preceding section, the sum of the direct and indirect effects on $\mathring{e}_0$ of the mortality changes in the $(0, x)$ age range can be shown to total

$$A(0, x) = \int_0^x (\mu_t^1 - \mu_t^2) \exp\left[\int_0^t (\mu_u^1 - \mu_u^2)\, du\right] {}_tp_0^1 \mathring{e}_t^1\, dt, \tag{10}$$

which is equivalent to

$$\int_0^x (\mu_t^1 - \mu_t^2) {}_tp_0^2 \mathring{e}_t^1\, dt. \tag{11}$$

Formula (10) [which differs from equation (1) only in the upper limit of the first integral] is made up of a main effects term

$$\int_0^x (\mu_t^1 - \mu_t^2) {}_tp_0^1 \mathring{e}_t^1\, dt \tag{12}$$

and terms representing the interactions between the mortality improvements at the various ages within the $(0, x)$ range, of which the jth-order interaction term

$$\frac{1}{j!} \int_0^x \left[\int_0^t (\mu_u^1 - \mu_u^2)\, du\right]^j (\mu_t^1 - \mu_t^2) {}_tp_0^1 \mathring{e}_t^1\, dt \tag{13}$$

is typical.

Arriaga's direct effect and (zero) indirect effect terms resulting from mortality changes over the age range (x, ∞) total

$$A(x, \infty) = \int_x^\infty (\mu_t^1 - \mu_t^2) \exp\left[\int_x^t (\mu_u^1 - \mu_u^2)\, du\right] {}_tp_0^1 \mathring{e}_t^1\, dt. \tag{14}$$

Once again, this formula may be partitioned into a main effects term and terms corresponding to the interaction between the various mortality changes beyond age x.

It should be noted that the direct and indirect effects term $A(0, x)$, corresponding to mortality changes below age x, and the direct and indirect effects term $A(x, \infty)$, corresponding to mortality changes beyond age x, do not sum to $A(0, \infty)$ [formula (1), or formula (10) with $x = \infty$, or formula (14) with $x = 0$]. The difference represents the interactions between the mortality changes below age x and those above age x. This is Arriaga's interaction term for the age group $(0, x)$. If

$$B(x) = A(0, \infty) - [A(0, x) + A(x, \infty)], \tag{15}$$

Arriaga's interaction term between mortality changes in the age range $(0, x)$ and mortality changes at all ages beyond x is

$$I(0, x) = B(x). \tag{16}$$

For mortality changes in the age range (x, y), Arriaga's direct and indirect effects on $\mathring{e}_0$ may be shown to total

$$A(x, y) = \int_x^y (\mu_t^1 - \mu_t^2) \exp\left[\int_x^t (\mu_u^1 - \mu_u^2)\, du\right] {}_tp_0^1 \mathring{e}_t^1\, dt; \tag{17}$$

and his interaction term between mortality changes in the range (x, y) and mortality changes at all ages beyond y is given by

$$I(x, y) = B(y) - B(x). \tag{18}$$

In each of these formulas, the interaction terms of the various orders can be separated by expanding the exponential terms in the manner outlined earlier. Numerical examples of the discrete approach are given in Arriaga (1984), using United States data. It is clear from equation (17) that the direct and indirect effects term for the age range (x, y) includes interaction terms *within* the (x, y) age range. Using finer and finer age spans in the discrete analysis separates more and more of the interactions from the direct and indirect effects. In the limit, the direct and indirect effects for the various age groups would sum to the right side of equation (3) and the interactions would total equation (5) minus the right side of equation (3).

A Generalization of the Continuous Analysis

The expectation of life at birth $\mathring{e}_0$ is an example of a more general integral,

$$\mathring{e}_0 = \int_0^\infty \lambda(t)_t p_0 \, dt, \tag{19}$$

where $\lambda(t)$ is any given function of the age t. If, for example, $\lambda(t) = 1$ for $18 < t < 65$ and 0 elsewhere, $\mathring{e}_0$ might represent the expected productive lifetime of a newborn individual. If $\lambda(t) = (1 + i)^{-t}$ for some interest rate i, $\mathring{e}_0$ would represent the present value of an annuity of 1 per annum payable throughout life.

For a given population, the function $\lambda(t)$ and the survivorship function ${}_t p_0$ may both change from one point of time to another. [Alternatively, in comparing two different cohorts or distinct populations, the function $\lambda(t)$ and the survivorship term may both differ.] The following formula was derived (Pollard, 1986b) to explain the difference $\mathring{e}_0^2 - \mathring{e}_0^1$ in terms of the changed values of the age-specific function $\lambda(t)$ and the changed mortality level:

$$\mathring{e}_0^2 - \mathring{e}_0^1 = \int_0^\infty [\lambda^2(t) - \lambda^1(t)]_t p_0^1 \, dt + \int_0^\infty (\mu_t^1 - \mu_t^2)_t p_0^1 \mathring{e}_t^2 \, dt. \tag{20}$$

Once again, an alternative exact partition can be obtained by interchanging the suffixes 1 and 2 in equation (20) and reversing the differences on both sides:

$$\mathring{e}_0^2 - \mathring{e}_0^1 = \int_0^\infty [\lambda^2(t) - \lambda^1(t)]_t p_0^2 \, dt + \int_0^\infty (\mu_t^1 - \mu_t^2)_t p_0^2 \mathring{e}_t^1 \, dt. \tag{21}$$

Both equations (20) and (21) are exact formulas, but the weights differ slightly. The following symmetric partition was therefore suggested:

$$\mathring{e}_0^2 - \mathring{e}_0^1 = \int_0^\infty [\lambda^2(t) - \lambda^1(t)] w_t^\lambda \, dt + \int_0^\infty (\mu_t^1 - \mu_t^2) w_t^\mu \, dt, \tag{22}$$

with

$$w_t^{\lambda} = \tfrac{1}{2}({}_tp_0^1 + {}_tp_0^2); \tag{23}$$

$$w_t^{\mu} = \tfrac{1}{2}({}_tp_0^2\mathring{e}_t^1 + {}_tp_0^1\mathring{e}_t^2); \tag{24}$$

$$\mathring{e}_t^j = \int_0^{\infty} {}_up_t^j\lambda^j(t+u)\,du, \qquad j = 1, 2. \tag{25}$$

Integration of the first integrand in equation (22) over selected age ranges explains the contributions of the changed $\lambda(t)$ values within those age ranges to the change in $\mathring{e}_0$. Similarly, integration of the second integrand over selected age ranges explains the contributions of mortality changes within those age ranges to the change in $\mathring{e}_0$. The forces of mortality can also be partitioned by cause of death in the manner of equation (9).

If $\lambda(t)$ equals the birth rate of daughters at age t, $\mathring{e}_0$ represents the net reproduction rate of the female population in question. One can therefore explain the difference in net reproduction rates between two populations or cohorts (or the change over time for a given population) in terms of the different mortality rates by cause of death and the different age-specific birth rates. This is demonstrated in Table 2 with respect to cross-sectional net reproduction rates of Australian females in 1921 and 1971, published by the Australian Bureau of Statistics.

Trends in Life Expectancy Differentials

Various authors have sought to explain changes in life expectancy differentials in terms of the changed age-specific mortality differentials of the populations in question. Lopez and Ruzicka (1977), for example, partitioned the change in the $\mathring{e}_0$ sex differential for Australia, 1910–1912 to 1970–1972, by using 11 causes of death and 7 age groups. Their method computed the change in life expectancy differential separately for each age/cause cell on the assumption that the mortality rates changed to their new levels within the cell and that the

Table 2. Australian Females: Contribution of Changes in Age-Specific Mortality Rates by Selected Causes and Changes in Age-Specific Fertility Rates to the Increase in Net Reproduction Rate (NRR) Between 1921 and 1971

	Contribution to increase in NRR of change in age-specific mortality due to				Contribution to change in NRR of		
Age group	Infectious diseases	Complications of pregnancy	External causes	All other causes	Age-specific mortality rate (all causes)	Age-specific fertility rate	Total
0	0.0036	0.0000	−0.0001	0.0536	0.0571	0.0000	0.0571
1–4	0.0076	0.0000	0.0016	0.0197	0.0289	0.0000	0.0289
5–14	0.0048	0.0000	0.0010	0.0109	0.0167	−0.0093	0.0074
15–19	0.0042	0.0004	−0.0014	0.0049	0.0081	0.0625	0.0706
20–24	0.0056	0.0025	−0.0011	0.0049	0.0119	0.0857	0.0976
25–29	0.0037	0.0021	−0.0003	0.0041	0.0096	0.0648	0.0744
30–39	0.0024	0.0016	−0.0003	0.0038	0.0075	−0.1942	−0.1867
40–49	0.0001	0.0000	0.0000	0.0002	0.0003	−0.0708	−0.0705
Total	0.0320	0.0066	−0.0006	0.1021	0.1401	−0.0613	0.0788

Note: The sum of columns 2–5 is equal to column 6, and the sum of columns 6 and 7 is equal to column 8.
Source: Pollard (1986b).

male and female mortality rates remained unchanged in all of the other cells. The total for the 77 separate cells fell well short of the observed change in sex differential. The residual term in fact comprised almost 40 percent of the change in differential. Retherford (1972) reached similar conclusions for United States data, 1910–1965.

The explanation for the large residual term can be found in Pollard (1982b): If two populations experience equal absolute reductions in their forces of mortality (reductions that may vary from age to age but are equal in absolute size for the two populations at any given age), the life expectancy differential will widen. This result will be elaborated in the following analysis.

Let us assume that the average force of mortality of the two sexes at age t is μ_t^a and that the differential is δ_t. The female force of mortality is therefore $\mu_t^a - \frac{1}{2}\delta_t$ and the male force $\mu_t^a + \frac{1}{2}\delta_t$. If we define

$$\Delta_t = \int_0^t \delta_u \, du \tag{26}$$

and write ${}_tp_0^a$ for the survivorship function based on the average forces of mortality $\{\mu_u^a\}$, it is easy to deduce that the sex differential (SD) is given by

$$SD = \mathring{e}_0^f - \mathring{e}_0^m = \int_0^\infty [\exp(\tfrac{1}{2}\Delta_t) - \exp(-\tfrac{1}{2}\Delta)]_t p_0^a \, dt \tag{27}$$

or

$$SD = \int_0^\infty 2 \sinh(\tfrac{1}{2}\Delta_t)_t p_0^a \, dt. \tag{28}$$

This integral is of the generalized form (19) with $2 \sinh(\frac{1}{2}\Delta_t)$ as the $\lambda(t)$ function. Equation (20) can therefore be used to partition the change in sex differential over the period from time 1 to time 2 into its various components. The first integral in equation (20) will show the contribution of the changed mortality differentials (in the absence of any common change in mortality for the two sexes), and the second term will show the effect of the mortality changes common to both sexes.

For the present purpose, the first integral in equation (20) is not in the most convenient form. Noting that ${}_tp_0^a$ is the derivative with respect to t of $-{}_tp_0^a\mathring{e}_t^a$, and integrating the first integrand by parts, we find that the contributions of the changes in the mortality differentials are given by

$$\int_0^\infty [\delta_t^2 \cosh(\tfrac{1}{2}\Delta_t^2) - \delta_t^1 \cosh(\tfrac{1}{2}\Delta_t^1)]_t p_0^{a2} \mathring{e}_t^{a2} \, dt, \tag{29}$$

where the suffixes 1 and 2 indicate functions evaluated at times 1 and 2, respectively.

For all except the very old ages (beyond age 80 for most developed populations), the hyperbolic cosine functions are effectively one, so equation (29) represents a weighted average of the changes $\{\delta_t^2 - \delta_t^1\}$ in the age-specific mortality differentials.

The second integral in equation (20), giving (in the present context) the effect of mortality changes common to both sexes, takes the form

$$\int_0^\infty (\mu_t^{a1} - \mu_t^{a2})({}_tp_0^{f2}\mathring{e}_t^{f2} - {}_tp_0^{m2}\mathring{e}_t^{m2})({}_tp_0^{a1}/{}_tp_0^{a2}) \, dt, \tag{30}$$

which is a weighted average of the changes in age-specific mortality common to both sexes. The suffix f indicates a female function and the suffix m a male function.

As before, an alternative exact partition can be obtained by interchanging the suffixes 1 and 2 in equations (29) and (30) and reversing the differences on both sides. Both partitions provide similar numerical results. Rather than use either, the following exact symmetrical partition is proposed, based on the mean weights of the nonsymmetrical partitions:

$$SD^2 - SD^1 = \int_0^\infty [\delta_t^2 \cosh(\tfrac{1}{2}\Delta_t^2) - \delta_t^1 \cosh(\tfrac{1}{2}\Delta_t^1)] w_t^\delta \, dt + \int_0^\infty \mu_t^{a1} - \mu_t^{a2}) w_t^\mu \, dt, \tag{31}$$

with

$$w_t^\delta = \tfrac{1}{2}({}_tp_0^{a1} \mathring{e}_t^{a1} + {}_tp_0^{a2} \mathring{e}_t^{a2}), \tag{32}$$

$$w_t^\mu = \tfrac{1}{2}[({}_tp_0^{f2} \mathring{e}_t^{f2} - {}_tp_0^{m2} \mathring{e}_t^{m2})({}_tp_0^{a1}/{}_tp_0^{a2}) + ({}_tp_0^{f1} \mathring{e}_t^{f1} - {}_tp_0^{m1} \mathring{e}_t^{m1})({}_tp_0^{a2}/{}_tp_0^{a1})]. \tag{33}$$

In all except the very old ages, the hyperbolic cosine terms can be omitted from equation (31), making the computation easier but making the partition an accurate approximation rather than an exact subdivision. Cause of death is readily incorporated into equation (31) by attaching the subscript i for cause i to each δ and μ (but *not* Δ) and summing for all causes.

In 1971 the Australian sex differential in expectation of life at birth was 6.57 years. The differential increased over the ensuing decade by 0.46 years, reaching 7.03 in 1981. The increase took place despite the substantial reductions in the mortality differential at many ages for some of the major causes of death. Both sexes in fact experienced substantial mortality reductions for most causes, and the common improvements resulted in a net increase in the mortality differential. The analysis (by cause), using the partition formula (31) *without* the hyperbolic cosine terms, is set out in Table 3. For computation purposes, 5-year age groups were used at all ages other than those under 5 or over 85. The accuracy of the partition is evident.

The first block of Table 3 shows the effects on the sex differential in expectation of life at birth of the observed changes in mortality common to both sexes (by cause and age) in the absence of any changes in the mortality differentials. The second block shows the effects of the observed changes in the mortality differentials (by cause and age) on the sex differential in life expectancy in the absence of any changes in the overall mortality level. The entries in the latter block are essentially the nonresidual values calculated in the manner of Lopez and Ruzicka (1977).

The results in Table 3 would have remained almost unchanged had some further simplifying approximations been made. Indeed, if the life table radix $l_0 = 1$ and T_t has its usual life table meaning, then

$$T_t = \int_t^\infty l_u \, du; \tag{34}$$

and if the weights (32) and (33) had been replaced by the rather simpler approximations

$$w_t^{\delta *} = \tfrac{1}{4}(T_t^{m1} + T_t^{f1} + T_t^{m2} + T_t^{f2}) \tag{35}$$

Table 3. Australia, Sex Differential in Expectation of Life at Birth: Contributions of the Mortality Changes Common to Both Sexes, and the Differential Changes in Mortality, to the Changed Sex Differential in Life Expectancy Between 1971 and 1981

Code, cause	Effects of mortality improvements common to both sexes								Effects of changes in mortality differentials								
	Age group							All ages	Age group							All ages	Total
	0	1–4	5–14	15–29	30–49	50–69	70+		0	1–4	5–14	15–29	30–49	50–69	70+		
1, infective, parasitic	1	0	0	0	0	0	0	2	−3	−1	1	0	−1	0	0	−5	−3
2, neoplasm, excluding 3, 4, 5	0	0	0	0	0	0	−3	−2	0	0	0	0	2	6	4	13	11
3, neoplasms, digestive	0	0	0	0	0	3	2	4	0	0	0	0	1	−4	2	−1	3
4, neoplasm, respiratory	0	0	0	0	0	0	−3	−4	0	0	0	0	1	−3	5	2	−2
5, neoplasms of breast	0	0	0	0	0	0	0	1	0	0	0	0	0	1	0	2	3
6, endocrine disease	0	0	0	0	0	0	2	4	−1	0	0	0	0	2	1	2	6
7, blood, mental, sense	0	0	0	0	0	0	−1	0	0	−1	0	1	2	−2	0	2	2
8, circulatory, excluding 9, 10	0	0	0	0	0	4	16	20	0	0	0	−1	1	1	1	3	23
9, ischaemic heart disease	0	0	0	0	2	23	32	57	0	0	0	0	−16	−51	−11	−77	−20
10, cerebrovascular disease	0	0	0	0	0	8	25	33	0	0	0	0	5	−1	1	5	38
11, respiratory disease	1	0	0	0	0	1	5	9	−6	1	−1	0	−1	−1	−2	−11	−2
12, bronchitis, emphysema, asthma	0	0	0	0	0	1	0	2	0	0	0	−1	0	−12	−4	−17	−15
13, digestive, excluding 14	0	0	0	0	0	0	−1	0	0	0	0	0	0	−1	0	1	1
14, cirrhosis of liver	0	0	0	0	0	0	0	−1	0	0	0	0	2	4	0	5	4
15, genito-urinary system	0	0	0	0	0	0	1	4	0	0	0	0	2	0	−3	1	5
16, pregnancy, childbirth	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	2	2
17, skin, tissue disease	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1	2
18, congenital anomalies	4	0	0	0	0	0	0	5	−6	−1	1	−1	0	0	0	−6	−1
19, ill-defined conditions	−1	0	0	0	0	0	1	1	4	0	0	0	0	0	0	4	5
20, external, excluding 19, 20	0	0	0	0	0	0	3	5	0	0	−3	−3	−5	−2	1	−12	−7
21, motor vehicle accidents	0	0	0	1	0	0	0	4	0	0	−1	−13	−5	−4	0	−22	−18
22, suicide, self-inflicted	0	0	0	0	0	0	0	2	0	0	0	4	3	0	0	7	9
Total, all causes	6	1	0	2	10	46	80	145	−9	−1	−2	−8	−7	−67	−7	−100	45

and

$$w_t^{\mu*} = \tfrac{1}{2}[(T_t^{f1} - T_t^{m1}) + (T_t^{f2} - T_t^{m2})], \tag{36}$$

then the calculated effects of the mortality improvements common to both sexes would be indistinguishable from those in Table 3 and the total of the effects of the changes of the mortality differentials would differ from that shown in Table 3 by less than 5 percent. The partition formula, excluding the hyperbolic cosine terms, would then take the form

$$SD^2 - SD^1 \doteq \int_0^\infty (\delta_t^2 - \delta_t^1) w_t^{\delta*}\, dt + \int_0^\infty (\mu_t^{a1} - \mu_t^{a2}) w_t^{\mu*}\, dt, \tag{37}$$

which is very easy to evaluate.

Concluding Remarks

I have shown that the formulas of Arriaga (1984)—partitioning the change in expectation of life in terms of the direct and indirect effects of mortality changes in given discrete age groups and interactions between the mortality changes in a given age group and those in all later age groups—can be expressed in terms of the earlier continuous formulas of Pollard (1982a). The sum of the interaction terms in the discrete analysis depends very much on the age intervals used. If the age intervals are made smaller and smaller, Arriaga's direct and indirect effects approach in total the main effects term of the continuous analysis, and his interaction terms approach in total the sum of the interaction terms of various orders of the continuous analysis.

The interaction terms are relatively small, and the complications they cause can be avoided in the continuous analysis by confounding them with the main effects in the partition formula (7) [or formula (9) if cause of death is also of interest]. The resulting analysis is straightforward and numerical results are readily interpretable from a two-way table such as Table 1.

Generalizations of the continuous analysis of change in life expectancy allow the analysis of changes in life expectancy differentials. The partition formula (31) derived herein is exact, but rather more complicated than the continuous partition formula for change in life expectancy. The two-way table based on it, however, allows a ready interpretation of the contributions of common mortality changes (by age and cause) and changes in mortality differentials (by age and cause) to the change in the life expectancy differential. An approximate partition, close to the exact partition, is also available, which should be accurate enough for most purposes for most advanced populations.

The analyses described have all been in terms of life expectancy at birth. The formulas, however, can be readily adapted to analyze expectations of life at other ages and temporary life expectancies.

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