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A GENERALIZATION OF LIFE EXPECTANCY WHICH INCORPORATES THE AGE DISTRIBUTION OF THE POPULATION AND ITS USE IN THE MEASUREMENT OF THE IMPACT OF MORTALITY REDUCTION

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INTRODUCTION

When analysing the contribution of a specific cause of death to the total mortality picture, it is natural to speculate on the consequences of eliminating this condition altogether. Within the limits of our current knowledge of disease processes and the present state of demographic methodology, this question is answered, at least in part, by the cause-deleted life table. If the life expectancy (at birth) is taken as an index summarizing the information in the life table, then a comparison of life expectancies before and after the elimination of the cause of death affords the investigator one means of measuring the impact of this condition on the population.

Although it is possible and even likely that the elimination of a cause of death will have beneficial effects on the population as a whole, for example, as a consequence of concomitant improvements in general medical care, it is reasonable to assume that the major advantage will accrue to those individuals who would otherwise have died of the disease in question. This unequal distribution of improved outcome is not accounted for by the cause-deleted life table which, in a sense, averages whatever gains are made over the entire life table cohort. Consequently, the elimination of a cause of death such as motor vehicle accidents, which affects relatively few individuals, results in only a modest increase in life expectancy according to the traditional method of accounting. The fact that each victim of a motor vehicle accident will tend to have his or her life dramatically shortened is not brought out in this approach. On the other hand, the elimination of cardiovascular disease produces a significant increase in life expectancy, not because this disease results in markedly premature mortality, but mainly because so many of the life table cohort are scheduled to die of this cause.

It seems that in order to give causes of death such as motor vehicle accidents a fair treatment, mortality analyses should be conducted not only from the point of view of the population as a whole (globally), as is usually done, but also focusing specifically on those individuals due to die of the cause under consideration (locally). In this article we describe a local counterpart to the well-established global method of measuring gains in life expectancy.

Life tables may be defined solely in terms of forces of mortality, and consequently are independent of the age distribution of the population. This is something of a limitation in practical applications since both magnitude of risk and the age distribution of those at risk are important. In this article we extend the notion of life expectancy to incorporate the age distribution of the population.

Finally, we observe that cause-deletion is a somewhat unrealistic notion, since it is unlikely that the diseases of importance are going to be eliminated in the near future. In what follows we will also consider less extreme approaches than complete elimination of causes of death.

LIFE TABLE CONSTRUCTION

We employ the method of life table construction developed by Chiang (1968), which rests on a number of fundamental assumptions: (1) death is due to a single cause; (2) causes of death act independently; and (3) forces of mortality are (calendar) time invariant.

We take the underlying cause of death as recorded on the medical death certificate to be "the" cause of death. It is clear that this represents a gross simplification of biological processes. Very often a number of interacting conditions will coexist in the same individual and play a role in the chain of events leading up to death. These intercurrent causes are not accounted for in the above method of life table construction.

Manton and coworkers, in a series of papers (1976, 1979, 1980b, 1982) have elaborated a concept that circumvents this problem. By defining patterns of failure in terms of the underlying cause as well as secondary causes of death, a broader family of causes is defined. Aside from this definitional modification, the formal aspects of the life table theory proceed as before; that is, Chiang's (1968) method and the above three assumptions are adopted. This means that the theoretical results of this article apply to the methods of Manton and coworkers.

There is a definite departure from the present approach in the model developed by Wong (1977). Once again the secondary causes on the death certificate are considered, but in this case they are used to introduce a notion of covariance among the causes of death. This method of life table construction does not assume that causes of death act independently, so our theoretical findings do not apply here.

A comparison of the methods of Chiang (1968), Manton, Tolley, and Poss (1976), and Wong (1977) has been reported by Manton and Poss (1979).

GLOBAL AND LOCAL GAINS IN LIFE EXPECTANCY

This section is devoted to the derivation of a pair of indices that measure the gain in life expectancy resulting from a reduction in the force of mortality for a given cause of death. The first index looks at the consequences of such a reduction from a global perspective, while the second takes the local point of view. Both indices have the flexibility to incorporate the age distribution of the population.

We begin by establishing some notation that is based on the cohort interpretation of the life table. The ordinary life table describes the overall survival of the life table birth cohort. Denote by $l(x)$ the number of survivors to age x, and by $e(x)$ the associated life expectancy. The multiple decrement life table for a given cause of death, say cause k, focuses on the survival of that subcohort of the life table birth cohort scheduled to die of cause k. The number of survivors to age x out of this subcohort is denoted by $l_k(x)$, and the corresponding life expectancy by $e_k(x)$.

We are interested in measuring the gain in life expectancy that would result from reducing the force of mortality from cause k by an amount $r \times 100$ percent at each age greater than x. Here r is a constant between 0 and 1, and x is an arbitrary but fixed age. For brevity we will speak of "reducing cause k by r." If such a reduction were to take place, then the $l(x)$ members of the birth cohort surviving to age x would have an increased life expectancy, which we denote by $e^{\cdot rk}(x)$. Similarly the $l_k(x)$ members of the birth cohort originally scheduled to die of cause k would now face a lesser threat of extinction from this condition with a resulting increased life expectancy, which we denote by $e_k^{\cdot rk}(x)$. It should be observed that with a reduction in cause k a certain proportion of those $l_k(x)$ individuals previously due to die of cause k will now die of other causes. The notation $e_k^{\cdot rk}(x)$ refers to the "new" life expectancy of the entire subcohort of size $l_k(x)$, *not* just those who will persist in

dying of cause k after cause of death reduction. In practice it will usually be impossible to identify that subcohort destined to die of a specified cause. Consequently, the discussion that follows refers to a group of individuals known to exist but who cannot be identified until the death of the last member of the birth cohort.

The function $e^{\cdot rk}(x)$ is introduced more formally in appendix A. When $r = 1$ we adopt the conventional notation and write $e^{\cdot k}(x)$ and $e_k^{\cdot k}(x)$. In order to distinguish $e(x)$, $e_k(x)$, $e^{\cdot rk}(x)$ and $e_k^{\cdot rk}(x)$ from a more general notion of life expectancy to be described shortly, we will refer to these functions as stationary life expectancies.

Although we have specified that cause k is only to be reduced at ages greater than x, the value of $e^{\cdot rk}(x)$ would be the same even if, for example, cause k were to be reduced starting at birth. This is because both $e(x)$ and $e^{\cdot rk}$ do not depend on the past survival history of the life table cohort. The mathematical expressions for these functions given in appendix A justify this observation. Consequently, our use of the notation $e^{\cdot k}(x)$ does not conflict with conventional usage, and the value of this function may be computed by standard cause-deleted life table techniques (Chiang 1968). A straightforward extension of the cause-deleted life table method (Tsai, Lee, and Hardy 1978) results in a computational approach to $e^{\cdot rk}(x)$.

Since the multiple decrement life expectancy is not independent of the past history of the life table cohort, a different approach to computing $e_k^{\cdot rk}(x)$ must be found. As a result of reducing cause k, the $l(x)$ members of the life table cohort surviving to age x will each live an additional $e^{\cdot rk}(x) - e(x)$ years, resulting in a gain to the cohort of

$$l(x)\,[e^{\cdot rk}(x) - e(x)] \tag{1}$$

person-years. Similarly the $l_k(x)$ members of the subcohort due to die of cause k will experience an added

$$l_k(x)\,[e_k^{\cdot rk}(x) - e_k(x)] \tag{2}$$

person-years. By assumption, cause k is independent of the other causes of death. It follows that any additional person-years resulting from a reduction in cause k must in fact be due to contributions from the subcohort scheduled to die of that cause. That is, expressions (1) and (2) must be equal. So we have

$$e_k^{\cdot rk}(x) = e_k(x) + \frac{l(x)}{l_k(x)}\,[e^{\cdot rk}(x) - e(x)]. \tag{3}$$

Identity (3) was established by Greville (1948) for the case $r = 1$. His argument relied more on first principles than the somewhat heuristic demonstration provided above. The line of reasoning used by Mitra (1979) is related to that of Greville. Identity (3) has been rediscovered on at least one occasion (Manton, Patrick, and Stallard 1980).

The quantities $e^{\cdot rk}(x) - e(x)$ and $e_k^{\cdot rk}(x) - e_k(x)$ form the basis of the global and local approaches to mortality analysis, respectively. As yet there is no allowance made for the age distribution of the population, a situation we now consider.

Suppose that we observe a population at calendar time t. For simplicity we will assume that there is only a finite set of ages $X = \{x_i : i = 1, \ldots, I\}$ represented in the population; it will be clear that the following arguments may be formulated in terms of a continuous age variable.

Let $n(x_i)$ be the number in the population at age x_i. Then there are a total of

$$n(X) = \sum_i n(x_i) \tag{4}$$

individuals under observation. The quantities $n(x_i)$ are available from census estimates. Similarly we denote by $n_k(x_i)$ the number in the population at age x_i due to die of cause k, with the corresponding total

$$n_k(X) = \sum_i n_k(x_i). \tag{5}$$

We now assume that certain life tables have been computed from cross-sectional data arising from a time period centered at time t, and that they describe the survival of the observed population after time t. So, for example, those members of the population at age x_i are now assumed to have a life expectancy $e(x_i)$. It follows that these individuals will contribute $n(x_i)\ e(x_i)$ person-years to the experience of the population after time t. Consequently, the average number of years of life left to an arbitrary member of the population is

$$e(X) = \frac{\sum_i n(x_i)\ e(x_i)}{n(X)}. \tag{6}$$

By a similar argument the average number of years left to someone due to die of cause k is

$$e_k(X) = \frac{\sum_i n_k(x_i)\ e_k(x_i)}{n_k(X)}. \tag{7}$$

If we now assume that cause k is reduced by r across all age groups after time t then (6) and (7) are replaced by

$$e^{\cdot rk}(X) = \frac{\sum_i n(x_i)\ e^{\cdot rk}(x_i)}{n(X)} \tag{8}$$

and

$$e_k^{\cdot rk}(X) = \frac{\sum_i n_k(x_i)\ e_k^{\cdot rk}(x_i)}{n_k(X)}, \tag{9}$$

respectively. The component functions of formulas (6) through (9) are all available except for $n_k(x_i)$. However, in the life table the probability of dying of cause k given survival to age x is $l_k(x)/l(x)$. Consequently,

$$n_k(x_i) = \frac{l_k(x_i)}{l(x)}\ n(x_i). \tag{10}$$

We refer to $e(X)$, $e_k(X)$, $e^{\cdot rk}(X)$ and $e_k^{\cdot rk}(X)$ as the population life expectancies. Obviously when X consists of a single element the stationary and population notions of life expectancy coincide.

Finally, we are able to define the global and local gains in life expectancy by

$$G^{rk}(X) = e^{\cdot rk}(X) - e(X) \tag{11}$$

and

$$L^{\cdot rk}(X) = e_k^{rk}(X) - e_k(X), \tag{12}$$

respectively. As a result of reducing cause k across all age groups after time t the population will gain

$$n(X)\, G^{rk}(X) = n_k(X)\, L^{\cdot rk}(X) \tag{13}$$

person-years, by an argument similar to that leading up to expression (3). It follows that

$$\frac{G^{rk}(X)}{G^{k}(X)} = \frac{L^{\cdot rk}(X)}{L^{\cdot k}(X)} = R^{\cdot rk}(X). \tag{14}$$

We will refer to $R^{\cdot rk}(X)$ as the relative gain in life expectancy. It is a measure of the extent to which a reduction in cause k by r achieves the gain in population life expectancy which would result from the elimination of cause k altogether. In view of (14) this relative gain is the same whether looked at globally or locally.

When X consists of a single age x we simplify (11) and (12), and write

$$G^{rk}(x) = e^{\cdot rk}(x) - e(x) \tag{15}$$

and

$$L^{\cdot rk}(x) = e_k^{rk}(x) - e_k(x). \tag{16}$$

As yet we have placed no restrictions on X. By taking X equal to $\{0\}$ we have the indices $G^{rk}(0)$ and $L^{\cdot rk}(0)$. At the other extreme we may take X equal to $\{0, 1, 5, 10, \ldots, 80, 85\}$, and in this case denote the indices by $G^{rk}(*)$ and $L^{\cdot rk}(*)$. These four special cases of (11) and (12) provide a solution to the problem posed in the introduction to this article; that is, they provide ways of measuring gain in life expectancy which (1) have a global and local perspective, (2) incorporate the age distribution of the population, and (3) allow reductions other than complete elimination of forces of mortality.

INEQUALITIES

We now show that cause-reduction and cause-elimination stand in a definite relationship to each other. In appendix A it is demonstrated that

$$[e^{\cdot rk}(x) - e(x)] \leqq r[e^{\cdot k}(x) - e(x)]\ . \tag{17}$$

It follows immediately from (6) and (8) that

$$G^{rk}(X) \leqq r\, G^{k}(X) \tag{18}$$

and from (14) that

$$L^{\cdot rk}(X) \leqq r\, L^{\cdot k}(X) \tag{19}$$

Inequalities (18) and (19) show that by reducing cause k by $r \times 100$ percent we are able to achieve at most $r \times 100$ percent of the gain in life expectancy resulting from

complete elimination of cause k. In view of (14) these remarks may summarized in the inequality

$$R^{\cdot rk}(X) \leqq r \,. \tag{20}$$

The better $R^{\cdot rk}(X)$ approximates r the closer $G^{\cdot rk}(X)$ and $L^{\cdot rk}(X)$ are to being linear functions of r. An empirical investigation of this phenomenon is the subject of a subsequent section.

Another question which arises is the behavior of $G^{\cdot rk}(X)$ and $L^{\cdot rk}(X)$ when two causes of death, say j and k, are reduced simultaneously. Let $j + k$ denote the cause consisting of deaths due to either j or k. In appendix B we show that

$$[e^{\cdot r(j+k)}(x) - e(x)] \geqq$$
$$[e^{\cdot rj}(x) - e(x)] + [e^{\cdot rk}(x) - e(x)] \,. \tag{21}$$

It follows from (6) and (8) that $G^{\cdot r(j+k)}(X) \geqq [G^{\cdot rj}(X) + G^{\cdot rk}(X)]$ (22)

and from (13) that

$$L^{\cdot r(j+k)}(X) \geqq \left[\frac{n_j(X)}{n_{j+k}(X)} L^{\cdot rj}(X) + \frac{n_k(X)}{n_{j+k}(X)} L^{\cdot rk}(X) \right] \tag{23}$$

where $n_{j+k}(X) = n_j(X) + n_k(X)$.

WEIGHTED GAINS IN LIFE EXPECTANCY

In the analysis of the preceding sections it is implicit that years of life gained through cause-reduction are all to be given the same weight regardless of where they fall in the life cycle. In certain situations this point of view may require modification. For example, it is clear that the earning power of a child is far less than that of a middle-aged adult, and so from a purely economic point of view an alternate weighting scheme would have to be devised.

The notion of weighted life expectancy has been developed by Hickman and Estell (1969) and more recently by Arriaga (1984). Following their example we define

$$ {}_{70}e(x) = \frac{T(x) - T(70)}{l(x)} \tag{24}$$
$$= \int_x^{70} l(v)dv \Big/ l(x)$$

for all x less than or equal to 70. In effect, years of life before age 70 are given a weight of 1 and those after age 70 a weight of 0.

Similarly we may define ${}_{70}e_k(x)$ and ${}_{70}e^{\cdot rk}(x)$; the definition of ${}_{70}e_k^{\cdot rk}$ follows from equation (3) since the arguments leading to that identity are equally valid when a weighted analysis is used. The definitions of ${}_{70}G^{\cdot rk}(X)$ and ${}_{70}L^{\cdot rk}(X)$ are analogous to (11) and (12) except that now it is assumed that X only includes ages up to 70.

Since the results of appendices A and B do not depend on the upper limit of integration in the integrals, it follows that there are weighted versions of inequalities (17) to (23).

AN ILLUSTRATION

We now illustrate the preceding theoretical considerations with an analysis of mortality in the Canadian male population of 1981. Three causes of death have been selected for study: circulatory disease (CIR, 390 - 459), neoplasms (NEO, 140 - 239), and external causes (EXT, 800 - 999). Our abbreviations and the codes according to the International Classification of Diseases, 9th Revision (ICD-9) are shown in parentheses. We will denote by CIR + NEO deaths due to either circulatory disease or neoplasms.

As remarked earlier, the life table functions appearing in this paper were computed using the methods developed by Chiang (1968). Table 1 gives values of $l(x)$ and $l_k(x)/l(x)$ for selected ages and for k = CIR, NEO, EXT, CIR + NEO. It will be noted that circulatory disease accounts for 48.9 percent of deaths in the life table birth cohort, compared to 23.1 percent for neoplasms, and 7.1 percent for external causes. The entries under CIR + NEO are simply the sum of those under CIR and NEO.

Table 2 gives values of $e(x)$ and $e_k(x)$. Those members of the life table birth cohort due to die of circulatory disease have a life expectancy at birth of 75.8 years, compared to 71.6 years in the case of neoplasms and 48.0 years in the case of external causes. Temporarily setting C = CIR and N = NEO, we observe that the value of $e_{C+N}(x)$ is intermediate between $e_C(x)$ and $e_N(x)$. This is a consequence of the following relationship:

$$e_{C+N}(x) = \frac{l_C(x)e_C(x) + l_N(x)e_N(x)}{l_C(x) + l_N(x)} \tag{25}$$

Tables 1 and 2 point out the need for methods of analyzing the impact of mortality reduction from both the global and local points of view. On the one hand, there are causes of death, such as circulatory disease, which affect a large proportion of the life table cohort, but which tend to kill at a relatively advanced age. At the same time, there are other causes of death, such as external causes, which pose a risk to a comparatively small proportion of the life table cohort, but which result in markedly premature death.

By specializing $G^{rk}(X)$ and $L^{rk}(X)$ to the case where X consists of the single age x and $r = 1$, we obtain, in the notation of (15) and (16), $G^k(x) = e^k(x) - e(x)$ and $L^k(x)$

Table 1.—Ordinary and multiple decrement survival curves, Canada, males, 1981

		$l_k(x) / l(x) \times 100\%$			
Age	$l(x)$	CIR	NEO	EXT	CIR + NEO
0	100,000	48.94	23.09	7.14	72.03
10	98,516	49.65	23.37	7.00	73.02
20	97,646	50.08	23.52	6.34	73.60
30	96,197	50.78	23.80	5.21	74.58
40	94,672	51.33	23.97	4.40	75.30
50	91,158	51.93	24.06	3.62	75.99
60	82,452	52.93	23.41	2.93	76.34
70	64,107	54.39	21.38	2.51	75.77
80	35,455	56.64	17.40	2.49	74.04

Note: See text for definitions of column headings.

Table 2.—Ordinary and multiple decrement life expectancies, Canada, males, 1981

Age	e(x)	$e_k(x)$ CIR	NEO	EXT	CIR + NEO
0	71.87	75.80	71.63	47.96	74.46
10	62.93	65.83	61.81	39.52	64.55
20	53.43	55.85	51.96	33.29	54.61
30	44.17	45.91	42.12	30.03	44.70
40	34.79	36.12	32.44	25.10	34.95
50	25.92	26.92	23.36	20.38	25.79
60	18.07	18.73	15.77	16.03	17.82
70	11.70	12.07	10.02	11.60	11.49
80	6.95	7.08	6.17	7.16	6.87

$= e_k^{\cdot k}(x) - e_k(x)$. Values of these functions are given in tables 3 and 4. The elimination of circulatory disease results in the largest gains in life expectancy from the global point of view, while the gains resulting from the elimination of external causes is comparatively modest (table 3). When the impact of the elimination of circulatory disease and external causes is viewed from the local perspective, however, the relative importance of these two causes of death is reversed (table 4), at least at the youngest ages.

In table 5 the global and local gains in life expectancy due to cause-deletion for the birth cohort are contrasted with those for the whole population, that is, $G^{\cdot k}(0)$ and $L^{\cdot k}(0)$ are compared to $G^{\cdot k}(*)$ and $L^{\cdot k}(*)$. The entries under birth cohort are taken directly from tables 3 and 4. It is apparent that for this data set there is little gained by incorporating age distribution into the analysis. We also note that inequalities (22) and (23) are satisifed by our data, and that the inequalities are quite far from being equalities. This shows that $G^{\cdot rk}(X)$ and $L^{\cdot rk}(X)$ are in general not additive functions of the variable k.

In table 6 we consider the relative gain in life expectancy for the birth cohort, $R^{\cdot rk}(0)$, for selected values of r. By inequality (20) we always have $R^{\cdot rk}(0) \leqq r$; the closer this inequality is to being an equality the closer $G^{\cdot rk}(0)$ and $L^{\cdot rk}(0)$ are to being linear functions of r. We see that for external causes, and to a somewhat lesser

Table 3.—Age-specific global gains in life expectancy due to cause-deletion, Canada, males, 1981

Age	$G^{\cdot k}(x)$ CIR	NEO	EXT	CIR + NEO
0	8.86	3.25	2.23	19.41
10	8.98	3.25	2.08	19.63
20	9.05	3.25	1.68	19.75
30	9.15	3.25	1.08	19.95
40	9.17	3.22	0.74	19.99
50	8.99	3.07	0.48	19.67
60	8.59	2.62	0.29	18.63
70	7.93	1.90	0.18	16.75
80	7.38	1.16	0.14	14.68

Table 4.—Age-specific local gains in life expectancy due to cause-deletion, Canada, males, 1981.

Age	$L^{\cdot k}(x)$ CIR	NEO	EXT	CIR + NEO
0	18.10	14.08	31.23	26.95
10	18.09	13.91	29.71	26.88
20	18.07	13.82	26.50	26.83
30	18.02	13.66	20.73	26.75
40	17.86	13.43	16.82	26.55
50	17.31	12.76	13.26	25.88
60	16.23	11.19	9.90	24.40
70	14.58	8.89	7.17	22.11
80	13.03	6.67	5.62	19.83

Table 5.—Global and local gains in life expectancy due to cause-deletion, for the birth cohort and the whole population, Canada, males, 1981

Gain	CIR	NEO	EXT	CIR + NEO
		Birth cohort		
$G \cdot^{k}(0)$	8.86	3.25	2.23	19.41 (12.11)[a]
$L \cdot^{k}(0)$	18.10	14.08	31.23	26.95 (16.81)[b]
		Whole population		
$G \cdot^{k}(*)$	8.93	3.08	1.28	19.44 (12.01)[a]
$L \cdot^{k}(*)$	17.54	13.18	23.93	26.16 (16.17)[b]

[a] Computed from the right side of inequality (22)
[b] Computed from the right side of inequality (23)

extent for neoplasms, there is near equality. However, for circulatory disease the discrepancy is significantly large. Consequently, an $r \times 100$ percent reduction in the force of mortality for external causes and neoplasms results in a gain in life expectancy at birth that is nearly $r \times 100$ percent of the gain due to total elimination. In the case of circulatory disease the gain is much less. Table 7 gives the corresponding analysis of $R^{\cdot rk}(*)$, and the findings are similar to those of table 6.

Tables 8, 9, and 10 repeat the analyses of tables 5, 6, and 7, except that now weighted life expectancies are considered.

The findings in table 8 present a different picture from those of table 5. Generally, the elimination of circulatory disease produces a much smaller gain in life expectancy compared to the gain from the elimination of external causes. This is a consequence of only counting additional years of life up to age 70, an age reached by most individuals who will die of circulatory disease, but not one ordinarily reached by those dying of external causes. We observe that the birth cohort and whole population findings differ somewhat more in table 8 than they did in table 5. In particular, the global gain from the elimination of external causes exceeds that for circulatory disease in the case of the birth cohort but not for the whole population

Table 6.—Relative gain in life expectancy, for the birth cohort, Canada, males, 1981

x 100%	$R \cdot^{rk}(0)$ x 100% CIR	NEO	EXT	CIR + NEO
1	.57	.88	.97	.41
5	2.90	4.40	4.88	2.08
10	5.92	8.85	9.78	4.27
25	15.69	22.54	24.53	11.57
50	35.24	46.61	49.35	27.29
75	61.27	72.35	74.52	51.54

Table 7.—Relative gain in life expectancy, for the whole population, Canada, males, 1981

r x 100%	$R \cdot^{rk}(*)$ x 100% CIR	NEO	EXT	CIR + NEO
1	.56	.87	.98	.40
5	2.85	4.38	4.89	2.02
10	5.80	8.81	9.80	4.15
25	15.44	22.47	24.57	11.28
50	34.80	46.51	49.43	26.78
75	60.86	72.27	74.57	50.98

Table 8.—Global and local gains in weighted life expectancy due to cause-deletion, for the birth cohort and the whole population (up to age 70), Canada, males, 1981

Gain	CIR	NEO	EXT	CIR + NEO
		Birth cohort		
$_{70}G^{\cdot k}(0)$	1.29	.93	1.63	2.30 (2.22)[a]
$_{70}L^{\cdot k}(0)$	2.63	4.01	22.86	3.19 (3.07)[b]
		Whole population (up to age 70)		
$_{70}G^{\cdot k}(*)$	1.16	.79	.87	2.02 (1.95)[a]
$_{70}L^{\cdot k}(*)$	2.29	3.34	15.91	2.73 (2.62)[b]

[a] Computed from the right side of weighted version of inequality (22)
[b] Computed from the right side of weighted version of inequality (23)

(table 8). We note that the weighted versions of inequalities (22) and (23) are very nearly equalities.

Tables 9 and 10 demonstrate that for these data, performing a weighted analysis results in a linearity with respect to r for circulatory disease that was absent from tables 6 and 7.

A PROPOSAL FOR INDICES OF MORTALITY

Inequalities (22) and (23) were derived principally from inequalities (17) and (21). It follows from results in the appendices that near equality can be expected to hold provided the causes of death under investigation are relatively rare. This was certainly observed to be the case for external causes, and to lesser extent for neoplasms, but not for circulatory diseases. However, by considering mortality risk only up to age 70, the weighted approach makes even circulatory disease a rare cause of death. As a result the weighted versions of inequalities (20), (22) and (23) were found to closely approximate equalities (tables 8, 9 and 10).

If it is granted that linearity in r and additivity in k are important attributes of a measure of the impact of mortality reduction, then, on empirical grounds, we

Table 9.—Relative gain in weighted life expectancy, for the birth cohort, Canada, males, 1981

	$_{70}R^{\cdot rk}(0)$ x 100%			
r x 100%	CIR	NEO	EXT	CIR + NEO
1	.96	.96	.98	.93
5	4.78	4.85	4.91	4.64
10	9.58	9.73	9.83	9.31
25	24.12	24.44	24.64	23.55
50	48.81	49.25	49.53	48.02
75	74.09	74.44	74.63	73.47

Table 10.—Relative gain in weighted life expectancy, for the whole population (up to age 70), Canada, males, 1981

	$_{70}R^{\cdot rk}(*)$ x 100%			
r x 100%	CIR	NEO	EXT	CIR + NEO
1	.95	.98	.98	.92
5	4.78	4.86	4.93	4.63
10	9.58	9.72	9.85	9.31
25	24.11	24.43	24.68	23.54
50	48.79	49.23	49.58	48.01
75	74.08	74.42	74.68	73.48

propose the indices ${}_{70}G^{\cdot rk}(X)$ and ${}_{70}L^{\cdot rk}(X)$. For populations with a mortality picture similar to that of Canada we can expect approximate linearity in r for both of these indices and approximate additivity in k for the first.

DISCUSSION

We have observed that the familiar cause-deleted gain in life expectancy $e^{\cdot k}(x) - e(x)$ suffers from a number of shortcomings: (1) it considers only the global, as opposed to the local, perspective; (2) it does not incorporate the age distribution of the population; and (3) it is restricted to the situation where an extreme form of mortality reduction is being considered, that is, total elimination of a cause of death. No one index can address all of these issues simultaneously. This article has been devoted to the derivation of two families of indices, denoted by $G^{\cdot rk}(X)$ and $L^{\cdot rk}(X)$, which meet the above requirements upon specialization.

A number of investigators have previously contributed to the discussion of mortality reduction as measured by gain in life expectancy. Greville (1948) appears to have been the first to recognize the need for local methods. His paper contains the equivalent of our equation (3).

Keyfitz (1977), as part of a search for an index of mortality which behaves additively for causes of death, was led to consider the first derivative of $e^{\cdot rk}(x) - e(x)$ with respect to r. An explicit formula for this derivative is available from equation (A7) in appendix A by setting $n = 1$. Mitra (1978) has discovered a convenient approximation to the integral. A claim is made by Keyfitz (1977:411) that is equivalent to asserting that $e^{\cdot rk}(0) - e(0)$ is approximately a linear function of r "within a considerable range." We have observed in table 6, however, that for circulatory disease there are departures from linearity for quite small values of r.

Tsai, Lee, and Hardy (1978) have conducted an analysis of mortality in the United States which, in our terminology, looks at global gains in life expectancy for the cohort of 15–year–olds using a weighted approach. In their formulation, years of life between 15 and 65 are given a weight of 1; outside of this range the weight is 0. For their data, the global index is linear in r; however, there is no comment upon its additivity over causes of death, a feature likely to be present in view of the findings in our table 8. In the appendix to Tsai, Lee, and Hardy (1978) an approximation is derived corresponding to our inequality (17), but only for the case $x = 0$. It is asserted in that paper that inequality (17) also holds, but no proof is given.

Elements of the work of Greville (1948), Keyfitz (1977) and Tsai, Lee, and Hardy (1978) are to be found in the present paper and have been discussed above. We depart from these approaches in that we give consideration to the age distribution of the population. We have observed, however, from tables 5 and 8, that the incorporation of age distribution may only be of importance in the weighted analysis.

It has been noted that the indices $G^{\cdot rk}(X)$ and $L^{\cdot rk}(X)$ do not have the desirable properties of linearity in r and additivity in k. By giving weight only to years of life up to age 70, however, the corresponding versions of these indices of mortality, ${}_{70}G^{\cdot rk}(X)$ and ${}_{70}L^{\cdot rk}(X)$, were found to come close to meeting these requirements. From (12) it can be seen that the numerator of ${}_{70}L^{\cdot rk}(X)$ is in fact the number of person-years up to age 70 lost to the population after a particular time because of cause k. This is reminiscent of another index well known in demography, namely, the potential years of life lost due to cause k (PYLL_k). What distinguishes ${}_{70}L^{\cdot rk}(X)$ from PYLL_k, apart from the denominator in the former, is the length of time during which the population is "followed up." In the case of PYLL_k the deaths due to cause k occurring during a short time period, usually one year, are considered. On the other hand, for ${}_{70}L^{\cdot rk}(X)$, or $L^{\cdot rk}(X)$, the time period extends to the death of the last

member of the population. Taking the latter view seems to have certain advantages, since for most causes of death the likelihood is that progress in mortality reduction will be a lengthy process.

One of the merits of the index $PYLL_k$ is that it places emphasis on deaths occurring at the youngest ages. As a result causes such as motor vehicle accidents gain a prominence that might otherwise be lost. This feature is also shared by ${}_{70}L^{\cdot rk}(X)$ and $L^{\cdot rk}(X)$. An unfortunate aspect of $PYLL_k$ is the rather arbitrary choice of age 70 as upper limit of "useful" life. Of course, it is possible to choose greater ages such as 75 or 80; according to contemporary views regarding the productivity of the elderly, this approach seems reasonable. As the upper limit increases, however, it becomes harder to justify the fundamental assumption underlying the definition of $PYLL_k$, namely, that if death from cause k had not occurred the individual would have survived to at least the given upper limit. Since the weighted versions of $L^{\cdot rk}(X)$ make no such assumption it is possible to compute with such advanced ages as 75 or 80. It is to be anticipated, however, that the approximate linearity in r observed for our particular choice of upper limit (age 70) will start to vanish as greater ages are selected.

In any given application the choice of mortality index will depend on the questions being asked. The indices proposed in this article, in both weighted and unweighted forms, appear to be as informative as such classical measures of mortality as standardized death rates and potential years of life lost. Further, the global and local indices described here have the advantage of being developed out of the same theoretical framework with a resulting uniformity of presentation.

APPENDIX A

Denote by $h(x)$ the total force of mortality at age x. Then the probability of surviving to age x is given by

$$p(x) = \exp\left[-\int_0^x h(v)dv\right] \tag{A1}$$

and the life expectancy at age x by

$$e(x) = \frac{\int_x^\infty p(v)dv}{p(x)}. \tag{A2}$$

Let $h_k(x)$ be the force of mortality for cause k, and define the cause-reduced force of mortality by

$$h^{\cdot rk}(x) = h(x) - r\,h_k(x) \tag{A3}$$

where r is a constant between 0 and 1. The cause-reduced counterparts to (A1) and (A2) are obtained by replacing $h(x)$ by $h^{\cdot rk}(x)$ in (A1) and $p(x)$ by $p^{\cdot rk}(x)$ in (A2). We observe that

$$p^{\cdot rk}(x) = p(x)\exp[r\,H_k(x)] \tag{A4}$$

where

$$H_k(x) = \int_0^x h_k(v)dv. \tag{A5}$$

The cause-reduced life expectancy may then be written

$$e^{\cdot rk}(x) = \frac{\int_x^\infty p(v) \exp[r\, H_k(v)]dv}{p(x) \exp[r\, H_k(x)]} = \frac{\int_x^\infty p(v) \exp[r\, H_k(v,x)]dv}{p(x)} \tag{A6}$$

where $H_k(v,x) = H_k(v) - H_k(x)$, a positive-valued function since x is less than or equal to v. It follows that the n^{th} derivative of $e^{\cdot rk}(x)$ with respect to r is

$$\frac{\int_x^\infty p(v) \exp[r\, H_k(v,x)]\, [H_k(v,x)]^n\, dv}{p(x)} \tag{A7}$$

Except for trivial cases, all of these derivatives are strictly positive. In particular, the first and second derivatives are, implying that $e^{\cdot rk}(x)$ is an increasing, concave upward function of r. It follows that the line segment joining $(0, e(x))$ and $(1, e^{\cdot k}(x))$ lies entirely above the graph of $e^{\cdot rk}(x)$ on the unit interval. That is,

$$e^{\cdot rk}(x) \leqq e(x) + r[e^{\cdot k}(x) - e(x)] \tag{A8}$$

for r between 0 and 1.

In view of (A6), the above inequality will be approximately an equality when either r or $H_k(\nu,x)$ is small, for all ν greater than x. The latter condition is the same as saying that k is a rare cause of death after age x.

APPENDIX B

From the Taylor series expansion of $e^{(s+t)}$ we have

$$e^{(s+t)} - 1 = \sum_{i=1}^{\infty} \frac{(s+t)^i}{i!}. \tag{B1}$$

If we assume that s and t are positive, then $(s+t)^i \geqq (s^i + t^i)$, for $i \geqq 1$, and so

$$[e^{(s+t)} - 1] \geqq [e^s - 1] + [e^t - 1]. \tag{B2}$$

Since $h_{j+k}(x)$, the force of mortality for cause $j + k$, is equal to $h_j(x) + h_k(x)$, it

follows that $H_{j+k}(v,x) = H_j(v,x) + H_k(v,x)$. Setting $s = r\,H_j(v,x)$ and $t = r\,H_k(v,x)$ in (B2), we have

$$\exp[r\,H_{j+k}(v,x)] - 1 \geqq \{\exp[r\,H_j(v,x)] - 1\} + \{\exp[r\,H_k(v,x)] - 1\}. \quad \text{(B3)}$$

Multiplying both sides of (B3) by $p(v)$, integrating, and dividing by $p(x)$, we find from (A2) and (A6) that

$$[e^{\cdot r(j+k)}(x) - e(x)] \geqq [e^{\cdot rj}(x) - e(x)] + [e^{\cdot rk}(x) - e(x)]. \quad \text{(B4)}$$

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REFERENCES

Arriaga, E. E. 1984. Measuring and explaining the change in life expectancies. *Demography* 21:83–96.

Chiang, C. L. 1968. *Introduction to Stochastic Processes in Biostatistics*. New York: John Wiley.

Greville, T. N. E. 1948. Mortality tables analyzed by cause of death. *Record of the American Institute of Actuaries* 37:283–294.

Hickman, J. C., and R. J. Estell. 1969. On the use of partial life expectancies in setting health goals. *American Journal of Public Health* 59:2243–2250.

Keyfitz, N. 1977. What difference would it make if cancer were eradicated? An examination of the Taeuber paradox. *Demography* 14:411–418.

Manton, K. G., C. H. Patrick, and E. Stallard. 1980. Population impact of mortality reduction: The effects of elimination of major causes of death on the 'saved' population. *International Journal of Epidemiology* 9:111–120.

Manton, K. G., and S. S. Poss. 1979. Effects of dependency among causes of death for cause elimination life table strategies. *Demography* 16:313–327.

Manton, K. G., and E. Stallard. 1982. Temporal trends in U.S. multiple cause of death mortality data: 1968 to 1977. *Demography* 19:527–547.

Manton, K. G., E. Stallard, and S. S. Poss. 1980. Estimates of U.S. multiple cause life tables. *Demography* 17:85–102.

Manton, K. G., H. D. Tolley, and S. S. Poss. 1976. Life table techniques for multiple-cause mortality. *Demography* 13:541–564.

Mitra, S. 1978. A short note on the Taeuber paradox. *Demography* 15:621–623.

———. 1979. The effects of extra chances to live on life table functions. *Theoretical Population Biology* 16:315–322.

Tsai, S. P., E. S. Lee, and R. J. Hardy. 1978. The effect of a reduction in leading causes of death: potential gains in life expectancy. *American Journal of Public Health* 68:966–971.

Wong, O. 1977. A competing risk model based on the life table procedure in epidemiological studies. *International Journal of Epidemiology* 6:153–159.