

## TARGETING LIFESAVING: DEMOGRAPHIC LINKAGES BETWEEN POPULATION STRUCTURE AND LIFE EXPECTANCY \*

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*Abstract.* Life expectancy in a heterogeneous population can be increased by lowering mortality rates or by averting deaths at different ages, from different causes, or for different groups, as well as by changing the proportions of individuals in various risk groups, perhaps by altering the transition rates between groups. Understanding how such changes in population structure affect life expectancy is useful in evaluating alternative lifesaving policies.

*Résumé.* Cibler la réduction de la mortalité. Relations démographiques entre structure de la population et espérance de vie

Dans une population hétérogène, l'espérance de vie peut être allongée par l'abaissement des taux de mortalité ou en évitant certains décès (à divers âges, de diverses causes ou dans divers groupes), ainsi qu'en changeant la façon dont la population est répartie par groupes exposés à des risques différents (par exemple en modifiant les probabilités de transition entre groupes). Pour évaluer différentes politiques de lutte contre la mortalité, il est utile de comprendre comment de tels changements dans la structure de la population affectent l'espérance de vie.

### 1. Introduction

The individuals comprising the typical population of men, mice, or machines face differing mortality chances. This heterogeneity arises, in

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part, from individual characteristics that change or can be changed, like age, behaviour, occupation, or residence. In this paper, we develop some formulae for analyzing how various kinds of changes in population structure, resulting perhaps from policy interventions that alter survivorship patterns, employment patterns, etc., will change the distribution of mortality chances and hence change life expectancy.

Change in life expectancy is a measure of the number of years of life saved (or lost) by an alteration in population structure and hence is a useful measure for policy analysis. In particular, this measure is appropriate for what might be called target analysis. If limited resources are available for lifesaving interventions, how should the resources be targeted? How effective would programmes be that are directed toward different age groups, diseases, risk groups (like cigarette smokers), regions, etc? A complete target analysis would have to include consideration of how difficult it is to focus an intervention on a particular group and how resistant the group is to change. Nonetheless, understanding the benefits of a change, if achieved, in life expectancy gained or life-years saved is clearly a key component of any target analysis.

In addition to such policy applications, the methods and formulae presented in this paper are useful in gaining a deeper demographic understanding of how mortality rates, deaths, risk groups, and life expectancy are interrelated. How, for instance, do mortality rates change if some deaths are averted?

Four different analytical approaches are used in the paper to analyze the demographic linkages between population structure and life expectancy: the comparative-statics approach, the dynamics approach, computer simulation, and a novel method that we call the 'second-chance' approach. The paper provides some discussion and illustration of the strengths, weaknesses, and interrelationships among these alternative methods of demographic analysis.

## **2. The comparative-statics and dynamics approaches**

Consider, first, age structure as characterized by the survivorship function

$$l(x) = e^{-\int_0^x \mu(s) ds}, \quad (1)$$

where  $\mu(x)$  represents the force of mortality at age  $x$ . [Formula (1) and the results that follow can be interpreted as pertaining to either period or cohort calculations.] A change in  $\mu$  will change this age structure and hence life expectancy at birth:

$$e_0 = \int_0^\omega l(x) dx, \tag{2}$$

where  $\omega$  is an age beyond which no one lives.

The effect of a change in  $\mu$  on  $e_0$  can be analyzed by either of two approaches. In the comparative-statics approach, the trajectory of  $\mu$  is assumed to change to  $\mu'$ , where

$$\mu'(x) = (1 - \delta(x))\mu(x); \tag{3}$$

the analyst relates the proportional change  $\delta(x)$  to the change in  $e_0$ , perhaps as measured by

$$\frac{\Delta e_0}{e_0} = \frac{e'_0 - e_0}{e_0}. \tag{4}$$

In the dynamics approach, there is some proportional rate of change in  $\mu(x, t)$  over time  $t$ :

$$\rho(x, t) = \frac{-\partial\mu(x, t)/\partial t}{\mu(x, t)}; \tag{5}$$

the analyst relates this proportional rate of change  $\rho(x, t)$  to the rate of change in  $e_0(t)$ :

$$\frac{de_0(t)/dt}{e_0(t)}.$$

Both approaches are informative and we shall consider both. For notational simplicity, we shall drop the argument  $t$  throughout and write  $\mu(x)$  rather than  $\mu(x, t)$  and  $e_0$  rather than  $e_0(t)$ .

If  $\rho(x)$  is constant over an interval of time of length  $T$  between the two mortality regimes  $\mu'$  and  $\mu$ , then

$$\mu'(x) = \mu(x) e^{-T\rho(x)}. \tag{6}$$

Combining this result with (3) yields the relationship between  $\rho$  and  $\delta$ :

$$\rho(x) = \frac{-\ln(1 - \delta(x))}{T}. \quad (7)$$

If  $\delta$  is small, this reduces to

$$\rho(x) \approx \frac{\delta(x)}{T}. \quad (8)$$

Hence, results concerning  $\rho(x)$  can be derived from results concerning  $\delta(x)$  and vice-versa: the comparative-statics approach and the dynamics approach complement each other. Note that for any specified value  $\delta(x)$ ,  $\rho(x)$  can be arbitrary large, as long as  $T$  is small enough.

A comparative-statics relationship can readily be derived from (1)–(4):

$$\frac{\Delta e_0}{e_0} = \frac{\int_0^\omega l(x) \left[ e^{\int_0^\delta \delta(s) \mu(s) ds} - 1 \right] dx}{\int_0^\omega l(x) dx}. \quad (9)$$

In the case of a uniform change in mortality at all ages,

$$\delta(x) = \delta, \quad \text{all } x.$$

formula (9) can be rewritten as

$$\frac{\Delta e_0}{e_0} = \frac{\int_0^\omega l(x) [e^{-\delta \ln l(x)} - 1] dx}{\int_0^\omega l(x) dx}. \quad (10)$$

For small  $\delta$ ,

$$e^{-\delta \ln l(x)} - 1 \approx -\delta \ln l(x). \quad (11)$$

Hence

$$\frac{\Delta e_0}{e_0} \approx \delta H, \quad (12)$$

where

$$H = \frac{- \int_0^\omega l(x) \ln l(x) dx}{\int_0^\omega l(x) dx} \tag{13}$$

In the limit, as  $\delta$  approaches zero, formula (12) holds exactly. Consequently, it is apparent that

$$\frac{de_0/dt}{e_0} = \rho H, \tag{14}$$

where  $\rho$  is the uniform rate of progress in reducing mortality rates:

$$\rho = \frac{-d\mu(x)/dt}{\mu(x)}, \quad \text{all } x. \tag{15}$$

Thus, for small changes in  $\mu$ , the comparative-statics approach yields the same formulae as the dynamics approach. Keyfitz (1977) derived (14) and noted that  $H$  is a measure of age heterogeneity; as Demetrius (1979) indicated,  $H$  can be interpreted as the entropy of the age composition of the population. See Pollard (1982) for use of the comparative-statics approach when changes in  $\mu$  are large.

### 3. The second-chance approach

#### 3.1. The basic model

Interventions to reduce mortality (or equipment failure) work by saving lives, i.e., by averting the scythe of death. Suppose that some proportion  $\delta$  of deaths at all ages are averted once. Let  $l(x)$  represent the proportion of the cohort at age  $x$  that is alive and has not been saved and let  $l^+(x)$  represent the proportion of the resuscitated who are alive at age  $x$ . Since the proportion of the cohort surviving at age  $x$  is given by

$$l'(x) = l(x) + \delta l^+(x), \tag{16}$$

the new life expectancy,  $e'_0$ , is given by

$$e'_0 = e_0 + \delta \int_0^{\omega} l^+(x) dx. \quad (17)$$

The relative change in life expectancy is simply

$$\frac{\Delta e_0}{e_0} = \delta \frac{\int_0^{\omega} l^+(x) dx}{\int_0^{\omega} l(x) dx}. \quad (18)$$

An expression for  $l^+(x)$  is readily developed. Assuming that the resuscitated face the same force of mortality as those who have not been saved, the probability of survival to age  $x$  for those whose lives were saved at age  $w$  is given by

$$P(T > x | w) = e^{-\int_w^x \mu(s) ds}, \quad (19)$$

where  $T$  represents age at death. Because the distribution density of  $w$  is  $\mu(w)l(w)$ ,

$$\begin{aligned} l^+(x) &= \int_0^x P(T > x | w) \mu(w) l(w) dw = e^{-\int_0^x \mu(s) ds} \int_0^x \mu(w) dw \\ &= -l(x) \ln l(x). \end{aligned} \quad (20)$$

Substituting (20) in (18) yields

$$\frac{\Delta e_0}{e_0} = \delta \frac{-\int_0^{\omega} l(x) \ln l(x) dx}{\int_0^{\omega} l(x) dx} = \delta H. \quad (21)$$

Note that the  $H$  in (21) denotes the same expression as Keyfitz's  $H$  in (12) and (14). Hence, (21) provides a third interpretation of  $H$  as a measure of the proportional increase in life expectancy if everyone's life were saved once, or alternatively, as the proportional increase in a randomly chosen individual's life span if that individual's life is saved. For Swedish males in 1982,  $H$  was 0.15 and  $e_0$  was 72 years. Conse-

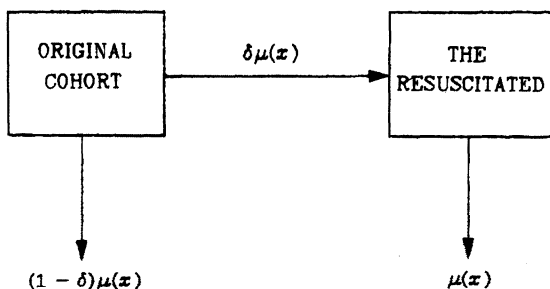


Fig. 1.

quently, at 1982 period mortality rates, averting the first death of a Swedish male would give the resuscitated about 11 years of life expectancy.

The formula for  $\Delta e_0/e_0$  in (21) holds exactly for any  $\delta$ , whereas the analogous formula in (12) only holds approximately, for small  $\delta$ . The reason can be understood by considering some simple diagrams. The model where death is averted only once can be represented as depicted in fig. 1. Individuals are all initially in the left box. A proportion  $\delta$  of those who would have died are saved, but just once: the resuscitated experience the original force of mortality  $\mu(x)$ . On the other hand, the model where mortality rates are decreased by  $\delta$  can be represented as depicted in fig. 2. Because the force of mortality in any state is  $(1 - \delta)\mu(x)$ , the overall force of mortality must also be  $(1 - \delta)\mu(x)$ . What the decomposition into an infinite stream of states reveals is that a reduction in mortality rates may result in some people's lives being saved several times.

Let  $\tau_0^i$  represent the expected life years lived by an individual in the

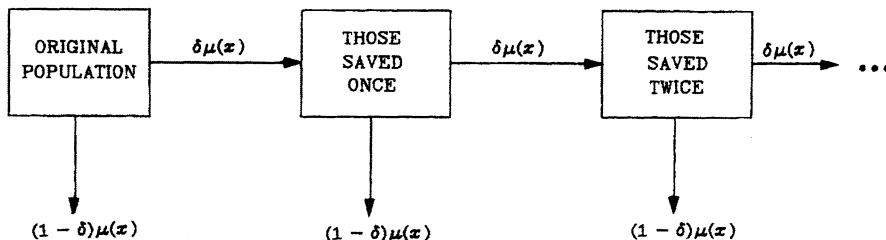


Fig. 2.

$i$ th state, i.e., by an individual whose life has been saved  $i$  and only  $i$  times:

$$\tau_0^i = \int_0^{\omega} p^i(x) dx, \quad (22)$$

where  $p^i(x)$  denotes the probability that a newborn individual is alive and in state  $i$  at age  $x$ . Note that  $\tau_0^0$  is equal to  $e_0$ , the original life expectancy before the lifesaving intervention. Clearly,

$$e'_0 = e_0 + \tau_0^1 + \tau_0^2 + \dots \quad (23)$$

When  $\delta$  is small, it is unlikely that anyone will gain much life expectancy by being saved more than once, i.e., the terms  $\tau_0^2$ ,  $\tau_0^3$ , and so on are unimportant. [We prove and expand on this intuitively plausible result elsewhere, in Vaupel and Yashin (1985).] Hence,

$$e'_0 \approx e_0 + \tau_0^1. \quad (24)$$

In the two-state model, where death is averted only once,

$$e'_0 = e_0 + \tau_0^1. \quad (25)$$

The similarity between (29) and (25) sheds light on why Keyfitz's  $H$  in (12) is identical to the  $H$  in (21).

It is sometimes easier to analyze the two-state model than the many-state model. Since the two models have equivalent implications for life expectancy in the limit for small  $\delta$ , the two-state model may provide a convenient line of attack. We exploit this, and the relationship between  $\delta$  and  $\rho$  discussed earlier, in several subsequent derivations in this paper. We call the method involving the two-state model the 'second-chance' approach, in contrast with the comparative-statics approach and the dynamics approach. Although in this paper the second-chance approach is used only to analyze changes in life expectancy, it has more general applications to any situation, including marriage, divorce, abortion, unemployment, the repair of equipment, etc., where changing some rate can be considered as equivalent to giving some individuals a second chance.



Suppose, as above, that some proportion  $\delta$  of deaths at all ages are averted once. How will the trajectory of mortality rates, as given by  $\mu(x)$ , change? In brief, how does saving lives affect mortality rates? Substituting (20) in (16), taking log derivatives, and then simplifying yields:

$$\mu'(x) = \mu(x) \left[ 1 - \frac{\delta}{1 - \delta \ln l(x)} \right]. \tag{26}$$

At age zero, when  $l(x)$  is one, the formula simplifies to

$$\mu'(0) = \mu(0)[1 - \delta]. \tag{27}$$

As survivorship decreases, however,  $\mu'(x)$  approaches  $\mu(x)$ . Thus, reducing deaths by some proportion  $\delta$  at all ages reduces the force of mortality by less than  $\delta$  at all ages after birth. The distribution of death times, as given by  $\mu(x)l(x)$ , changes to

$$\mu'(x)l'(x) = \mu(x)l(x)[1 - \delta - \delta \ln l(x)], \tag{28}$$

so that a reduction in deaths by  $\delta$  leads to a new distribution of death times shifted to older ages. Since death, as Shakespeare put it, ‘is certain to all’, it is clear that a death averted today is an additional death tomorrow. The mathematics of this adjustment is captured by (26) and (28).

### *3.2. If the resuscitated are different*

The formulae and calculations above assume that a resuscitated person would subsequently face the same force of mortality as a person whose life had not been saved. To generalize the formula, it is useful to consider the following variation on the model discussed above (see fig. 3). Note that now individuals who are saved experience a mortality trajectory given by  $\mu^+(x)$ , rather than by  $\mu(x)$ . Let  $e^+(x)$  be the life expectancy at age  $x$  of the resuscitated:

$$e^+(x) = \int_x^\omega l_x^+(s) ds, \tag{29}$$

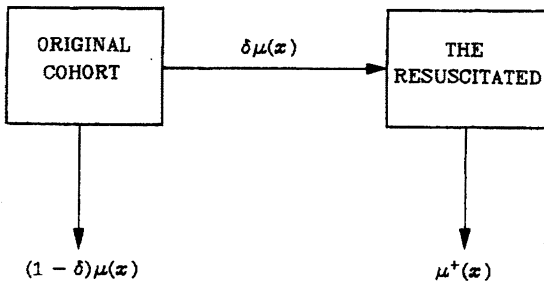


Fig. 3.

where

$$l_x^+(s) = e^{-\int_x^s \mu^+(u) du}. \quad (30)$$

Because the density at age  $x$  of the distribution of (first) death is given by  $\mu(x)l(x)$ , the value of  $\Delta e_0$  must be given by

$$\Delta e_0 = \delta \int_0^\omega \mu(x)l(x)e^+(x) dx. \quad (31)$$

Hence

$$\frac{\Delta e_0}{e_0} = \delta H^+, \quad (32)$$

where

$$H^+ = \frac{\int_0^\omega \mu(x)l(x)e^+(x) dx}{\int_0^\omega l(x) dx}. \quad (33)$$

If  $\mu^+(x)$  equals  $\mu(x)$ , so that individuals are, in effect, saved from death once, then  $H^+$  equals  $H$ . If  $\mu^+(x)$  equals  $(1 - \delta)\mu(x)$ , so that death rates are reduced uniformly for everyone, regardless of whether they have been resuscitated or not,  $H^+$  will be close in value to  $H$  as long as  $\delta$  is small. Consequently,

$$\frac{de_0/dt}{e_0} = \rho H, \quad (34)$$

where

$$H = \frac{\int_0^\omega \mu(x)l(x)e(x) dx}{\int_0^\omega l(x) dx} \tag{35}$$

This expression for  $H$ , which is equal in value to Keyfitz's expression for  $H$ , was derived by Vaupel (1986) directly from Keyfitz's formula. The expression clearly indicates how the effect of saving lives on life expectancy depends on both the number of deaths at various ages and on the number of additional years of life a resuscitated person might have.

#### 4. Targeting lifesaving in selected sub-groups

##### 4.1. Reducing death rates in selected age groups

As Vaupel (1986) discusses at length, if death rates are reduced by some proportion  $\delta$  between ages  $\alpha$  and  $\beta$ , then for small  $\delta$ ,

$$\frac{\Delta e_0}{e_0} \approx \delta H_{\alpha\beta}, \tag{36}$$

where

$$H_{\alpha\beta} = \frac{\int_\alpha^\beta \mu(x)l(x)e(x) dx}{\int_0^\omega l(x) dx} \tag{37}$$

Correspondingly, if progress is being made at a rate  $\rho$  against mortality between ages  $\alpha$  and  $\beta$ , then

$$\frac{de_0/dt}{e_0} = \rho H_{\alpha\beta} \tag{38}$$

The values of  $H_{\alpha\beta}$  for various five-year age categories for Swedish males and females in 1982 are given in table 1. Remarkably, it is for males 70 to 75 and for females 75 to 80 that  $H_{\alpha\beta}$  is largest. A one per

Table 1  
 Values of  $H_{\alpha\beta}$  for Swedish males and females in 1982.

Age period	Males	Females
0-5	0.00853	0.00763
5-10	0.00085	0.00068
10-15	0.00060	0.00060
15-20	0.00226	0.00079
20-25	0.00289	0.00120
25-30	0.00344	0.00140
30-35	0.00341	0.00172
35-40	0.00434	0.00243
40-45	0.00530	0.00313
45-50	0.00736	0.00447
50-55	0.00942	0.00591
55-60	0.01258	0.00779
60-65	0.01555	0.00936
65-70	0.01788	0.01202
70-75	0.01869	0.01464
75-80	0.01719	0.01679
80-85	0.01282	0.01650
85-90	0.00675	0.01201
90-95	0.00231	0.00545
95-100	0.00055	0.00142
<i>H</i> (i.e., total for all ages)	0.15270	0.12622

Source: Vaupel (1986).

cent reduction in mortality in those age categories would increase life expectancy at birth by more than twice as much as a one per cent reduction in mortality in infancy and early childhood.

#### 4.2. Averting deaths from selected causes

Let  $\mu_c(x)$  represent the force of mortality from cancer, or more generally any specified cause of death. Suppose that for some proportion  $\delta$  of individuals who would have died from cancer, the (first) death from cancer is averted. Further suppose that these resuscitated individuals then have the same remaining life expectancy as ordinary individuals. Using the second-chance approach and the same kind of reasoning employed to derive formulae (31)–(33), it is clear that

$$\frac{\Delta e_0}{e_0} = \delta \frac{\int_0^{\omega} \mu_c(x) l(x) e(x) dx}{e_0} = \delta H_c \quad (39)$$

If  $\delta$  is small, it is unlikely that an individual would be saved from cancer death more than once. Hence, (39) holds approximately for a reduction  $\delta$  in cancer mortality rates as long as  $\delta$  is small. It follows that

$$\frac{de_0/dt}{e_0} = \rho H_c, \tag{40}$$

where  $\rho$  is the rate of progress in reducing cancer mortality

$$\rho = \frac{-d\mu_c(x)/dt}{\mu_c(x)}. \tag{41}$$

If cancer is independent of other causes of death, then it is possible to derive an alternative expression for  $H_c$  that is similar to Keyfitz's formula for  $H$  in (13). Let  $l_c^+(x)$  represent the proportion of people in the population who are alive at age  $x$  and who have been saved once from cancer death (at any age prior to  $x$ ). By analogy to (20), letting  $w$  denote the age at which cancer death was averted, it follows that

$$l_c^+(x) = \int_0^x P(T > x | w) \mu_c(w) l(w) dw = -l(x) \ln l_c(x), \tag{42}$$

where  $l_c(x)$  can be interpreted as the survival function when cancer is the only cause of death,

$$l_c(x) = e^{-\int_0^x \mu_c(s) ds}. \tag{43}$$

Hence, by the same logic used to derive (21),

$$H_c = \frac{\int_0^\omega l(x) \ln l_c(x) dx}{\int_0^\omega l(x) dx}. \tag{44}$$

Keyfitz (1977) derives formula (44) using a different approach. In addition, he presents some illustrative examples. For instance, for Italian females in 1964,  $H_c$  for deaths from neoplasms was 0.0300, compared with a total  $H$  of 0.1631. Thus, a one per cent reduction in

cancer mortality would increase life expectancy at birth by about three per cent of one per cent, or by about eight days given Italian female life expectancy of 72.9 years in 1964. By way of comparison,  $H_c$  for deaths from cardiovascular diseases was 0.0564, almost twice as high as the  $H_c$  for deaths from cancer, whereas  $H_c$  for deaths from influenza, pneumonia and bronchitis was 0.0122, or less than half as great as the  $H_c$  for deaths from cancer.

### 4.3. Averting deaths in high-risk groups

Consider now a population that is structured according to race, sex, socio-economic status, region or some other classification. Adopting the line of attack of the second-chance approach, suppose that a proportion  $\delta_i$  of the first deaths in group  $i$  are averted. What will the effect be on the life expectancy of the entire population? Letting  $\mu_i(x)$ ,  $l_i(x)$ , and  $e_i(x)$  denote the force of mortality, survivorship function, and remaining life expectancy at age  $x$  of the  $i$ th group, then

$$\Delta e_0 = \delta_i \pi_i(0) \int_0^{\omega} \mu_i(x) l_i(x) e_i(x) dx, \quad (45)$$

where  $\pi_i(0)$  is the initial proportion of the population in the group  $i$ . Hence,

$$\frac{\Delta e_0}{e_0} = \delta_i H_i \quad (46)$$

and

$$\frac{de_0/dt}{e_0} = \rho_i H_i, \quad (47)$$

where

$$H_i = \frac{\pi_i(0) \int_0^{\omega} \mu_i(x) l_i(x) e_i(x) dx}{\int_0^{\omega} l(x) dx} \quad (48)$$

and

$$\rho_i = \frac{-\partial\mu_i(x)/\partial t}{\mu_i(x)}, \quad \text{all } x. \quad (49)$$

The US male population, for example, might be classified as white and non-white. The value of  $H_i$  for US non-white males in 1950 was about 0.038. So reducing non-white male mortality by one per cent would add about nine days to the overall US male life expectancy of 65.5 years. By comparison, this reduction in non-white male mortality would add about 75 days to non-white male life expectancy. The difference is largely explained by the proportion of non-whites at birth, about 12.6 per cent.

The US population as a whole can be divided into male and female groups. The value of  $H$  for males at 1980 mortality rates was 0.193, the value for females was 0.155. If the two groups are given equal weight, then  $H$  for the entire population is 0.179 and  $H_i$  is 0.096 for males and 0.077 for females. Suppose there are three alternative interventions. The first reduces male mortality by two per cent, the second reduces female mortality by two per cent, and the third reduces total mortality by one per cent. The male strategy would save about 11 per cent more years of life than the total strategy which, in turn, would save about 15 per cent more years of life than the female strategy.

## 5. Targeting lifesaving by changing high-risk behaviour

### 5.1. Reducing the proportion in high-risk groups

#### A. Two groups

Suppose that a population consists of two subpopulations with age-specific mortality rates  $\mu_1(x)$  and  $\mu_2(x)$ , where  $\mu_2(x) > \mu_1(x)$  and where two groups might be residents of urban vs. rural areas, smokers vs. non-smokers, blue-collar workers vs. white-collar workers, people in the south of a country vs. people in the north, people who are overweight vs. people who are not, etc. How will changes in the distribution of the population between these two groups affect life expectancy?

Consider an intervention that changes  $\pi(x)$ , the proportion of the population in the high-risk group, by some proportion  $\delta$  at all ages after some initial age  $x_0$ :

$$\pi'(x) = (1 - \delta)\pi(x). \quad (50)$$

It is convenient to consider age  $x_0$  as the age at 'birth', so that  $e_0$  refers to remaining life expectancy at age  $x_0$  and  $x$  refers to years of age since  $x_0$ . The force of mortality for the population as a whole is given by

$$\bar{\mu}(x) = (1 - \pi(x))\mu_1(x) + \pi(x)\mu_2(x) \quad (51)$$

and

$$\bar{\mu}'(x) = (1 - (1 - \delta)\pi(x))\mu_1(x) + (1 - \delta)\pi(x)\mu_2(x). \quad (52)$$

Hence,

$$\bar{\mu}'(x) - \bar{\mu}(x) = \delta(\mu_1(x) - \bar{\mu}(x)). \quad (53)$$

It follows that

$$\Delta e_0 = \int_0^{\omega} l(x) \left[ e^{\int_0^x \delta(\mu_1(s) - \bar{\mu}(s)) ds} - 1 \right] dx. \quad (54)$$

If  $\delta$  is small,

$$\frac{\Delta e_0}{e_0} \approx \delta H_1, \quad (55)$$

so

$$\frac{de_0/dt}{e_0} = \rho H_1, \quad (56)$$

where



$$H_1 = \frac{\int_0^{\omega} l(x) \ln [l_1(x)/l(x)] dx}{\int_0^{\omega} l(x) dx}, \quad (57)$$

$$l(x) = (1 - \pi(0))l_1(x) + \pi(0)l_2(x), \quad (58)$$

and

$$\rho = \frac{-\partial \pi(x)/\partial t}{\pi(x)}. \quad (59)$$

As an example of the use of these formulae, suppose that the population consists of non-smokers and smokers, and that the population is being studied starting at age 35 (so that  $e_0$  refers to life expectancy at age 35). Further suppose that the force of mortality for non-smokers is  $0.001e^{0.1x}$  ( $x$  being age minus 35), that the force of mortality for smokers is twice as high, and that half the population smokes at age 35. Remaining life expectancy for non-smokers in this case is about 40.8 years and remaining life expectancy for smokers about 34.2 years. Then  $H_1$  turns out to equal 0.077. If the proportion of the population that smokes is reduced by one per cent, then life expectancy (at age 35) will increase by 0.077 per cent, or by about 11 days, given the average remaining life expectancy for the population as a whole of 37.5 years.

More generally, it is interesting to investigate the values of  $H_1$ , and of expected days of life saved, at different starting ages, i.e., at different ages of intervention. Table 2 presents some sample calculations. Note that  $H_1$  increases with age: a reduction in smoking yields a greater proportional increase in life expectancy at the ages with the highest mortality rates. The absolute increase in life expectancy, however, as measured by days added, falls off with age. Because it falls off slowly, at least before age 55 or 65, it may be optimal to target anti-smoking interventions toward older people – if it is easier to induce older smokers to quit. The calculations in table 2 are merely illustrative, but some empirical analysis of this sort could shed light on the effectiveness of targeting various kinds of health programmes toward individuals in different age classes.

Table 2

Values of  $H_1$ , life expectancy, and days added to life expectancy if the proportion of a population that smokes is reduced by one per cent, at various ages.

Age	$H_1$	Remaining life expectancy (in years)			Days added to total life expectancy if proportion that smoke is reduced by one per cent
		Non-smokers	Smokers	Total population	
35	0.077	40.8	34.2	37.5	10.5
45	0.095	31.4	25.2	28.3	9.9
55	0.120	22.6	17.1	19.9	8.7
65	0.150	14.9	10.5	12.7	7.0
75	0.184	8.8	5.6	7.2	4.8
85	0.215	4.5	2.7	3.6	2.8

### B. More than two groups

The results in the previous section can be generalized to the case where the population consists of  $N$  subpopulations with age-specific mortality rates  $\mu_i(x)$  and in proportions  $\pi_i(x)$ , where

$$\sum_{i=1}^N \pi_i(x) = 1 \quad (60)$$

and

$$\bar{\mu}(x) = \sum_{i=1}^N \pi_i(x) \mu_i(x). \quad (61)$$

As before, let the first subpopulation be the healthiest,  $\mu_1(x) < \mu_i(x)$  for all  $x$  and for all  $i > 1$ , and let  $\delta_i$  denote the change in proportions at starting age 0:

$$\pi_i'(x) = (1 - \delta_i) \pi_i(x), \quad i > 1, \quad x \geq 0. \quad (62)$$

Clearly,

$$\pi_1'(x) = 1 - \sum_{i=2}^N \pi_i(x) = \pi_1(x) + \sum_{i=2}^N \delta_i \pi_i(x). \quad (63)$$

For simplicity, assume that

$$\delta_i = \delta, \quad \text{all } i > 1. \tag{64}$$

Then it is not difficult to show that

$$\bar{\mu}'(x) - \bar{\mu}(x) = \delta(\mu_1(x) - \bar{\mu}(x)). \tag{65}$$

This formula is identical to (53). Consequently,

$$\frac{\Delta e_0}{e_0} \approx \delta H_1 \quad \text{for small } \delta, \tag{66}$$

and

$$\frac{de_0/dt}{e_0} = \rho H_1, \tag{67}$$

where  $H_1$  is defined as before by (57) and

$$\rho = \frac{-\partial \pi_i(x)/\partial t}{\pi(x)}, \quad \text{all } i > 1. \tag{68}$$

That  $H_1$  is the same as before may, at first glance, seem puzzling but, on closer thought, it is reasonable because the assumptions group the sub-populations into two parts. Other formulae can readily be derived for other special cases.

As an illustration of the use of (66), consider a population of males with a high prevalence of alcoholism. In particular, assume that 50 per cent of the population drink moderately or not at all, that 30 per cent drink heavily, and that the remaining 20 per cent drink very heavily. Further, assume that the heavy drinkers have twice the mortality and the very heavy drinkers have four times the mortality of the first group. Finally, as in the previous example, suppose that the population is being considered starting at age 35 and that the force of mortality follows a Gompertz curve with  $\alpha = 0.001$  and  $b = 0.1$  for the healthy subpopulation.

Remaining life expectancy for the three groups turns out to be 40.8, 34.2, and 27.9 years and, for the population as a whole, 36.2 years. The

value of  $H_1$  is 0.108; a one per cent reduction in the proportion of heavy and of very heavy drinkers, would add two weeks to the population's life expectancy.

### 5.2. Changing the rate of transition to low-risk behaviour

Now consider a population that consists of various subpopulations, with individuals making transitions from one subpopulation to another, such that the transition rates are changing or can be changed. For instance, the population may consist of smokers and non-smokers, with some smokers who stop and some non-smokers who start. If either of these transitions could be influenced, what would the effect be on life expectancy? This question is similar to the question considered in the previous two sections, except that the policy lever or control parameter is not the proportion of the population who smoke, but the transition rates between the non-smoking and smoking states. Changing the transition rates will change the proportions and hence life expectancy.

For a cohort, the change in the proportion of individuals in state (or group)  $j$  at age  $x$  is given by the equation

$$\frac{d\pi_j(x)}{dx} = \sum_{i=1}^N \lambda_{ij}(x) \pi_i(x) + \pi_j(x) \left[ \sum_{i=1}^N \pi_i(x) \mu_i(x) - \mu_j(x) \right],$$

$$j \in \overline{1, N}, \quad (69)$$

where  $\lambda_{ij}(x)$  are the transition rates from state  $i$  to state  $j$  at age  $x$ , with the initial proportions  $\pi_j(0)$  given.

In the simplest case of a two-state population with mortality rates  $\mu_1(x)$  and  $\mu_2(x)$  and transition rate  $\lambda(x)$  from state 1 to state 2, the proportion  $\pi(x)$  of individuals in state 2 is the solution of the following equation:

$$\frac{d\pi(x)}{dx} = \pi^2(x)(\mu_2(x) - \mu_1(x)) - \pi(x)(\mu_2(x) - \mu_1(x) + \lambda(x))$$

$$+ \lambda(x). \quad (70)$$

with  $\pi(0)$  given. Let the rate of progress in reducing  $\lambda(x)$  be given by  $\rho(x)$ :

$$\rho(x) = \frac{-\partial\lambda(x)/\partial t}{\lambda(x)}. \tag{71}$$

Straightforward calculations show that

$$\frac{de_0/dt}{e_0} = \frac{\int_0^\omega l(x) \int_0^x \rho(y)(\mu_2(y) - \mu_1(y))\lambda(y)q(y) dy dx}{\int_0^\omega l(x) dx}. \tag{72}$$

where  $q(x)$  is the solution of the differential equation

$$\begin{aligned} \frac{dq(x)}{dx} &= 2\pi(x)q(x)(\mu_2(x) - \mu_1(x)) - q(x)(\mu_2(x) - \mu_1(x) + \lambda(x)) \\ &\quad - \pi(x) + 1. \end{aligned} \tag{73}$$

with  $q(0) = 0$ . Note that this equation has to be solved together with eq. (70) for  $\pi(x)$ . If the rate of progress in decreasing  $\lambda(x)$  does not depend on age, then (72) reduces to

$$\frac{de_0/dt}{e_0} = \rho H_\lambda, \tag{74}$$

where

$$H_\lambda = \frac{-\int_0^\omega l(x) \int_0^x (\mu_2(y) - \mu_1(y))\lambda(y)q(y) dy dx}{\int_0^\omega l(x) dx}. \tag{75}$$

### 6. Resort to simulation

Solving (75) for  $H_\lambda$  is not easy, since  $q(y)$  is the solution of a differential equation (73) that depends on another differential equation

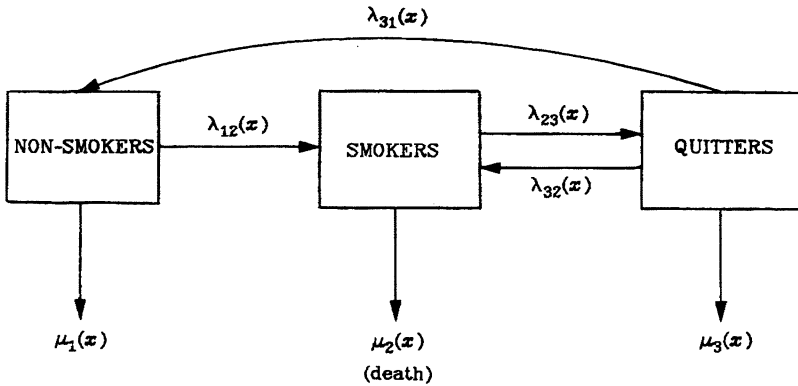


Fig. 4.

(70). When mathematical solutions become as this, they may not only lose elegance but also usefulness for either insight or computation. It may then be fruitful to take a different tack and rely on numerical, computer simulation.

Consider, for instance, the following illustrative model (fig. 4). The population is divided into three groups – non-smokers, smokers, and quitters. The starting point of the analysis is age 10:  $x$  represents age minus 10. For non-smokers, the force of mortality is given by

$$\mu_1(x) = 0.001 e^{0.1x}, \quad (76)$$

for smokers it is

$$\mu_2(x) = 2\mu_1(x), \quad (77)$$

and for quitters,

$$\mu_3(x) = 1.5\mu_1(x). \quad (78)$$

At the start all individuals are non-smokers:

$$\pi_1(0) = 1, \quad (79)$$

$$\pi_2(0) = \pi_3(0) = 0. \quad (80)$$

The transition intensities are

$$\lambda_{12}(x) = 0.06 e^{-0.1x}, \tag{81}$$

$$\lambda_{23}(x) = 0.02 e^{0.05x}, \tag{82}$$

$$\lambda_{32}(x) = 0.5 e^{-0.02x}, \tag{83}$$

$$\lambda_{31}(x) = 0.1. \tag{84}$$

These transition intensities imply that:

- about six per cent of non-smokers start smoking at age 10, about two per cent at age 20, and less than one per cent at age 30;
- the proportion of smokers who quit smoking rises from about two per cent per year at age 10 to 10 per cent per year at age 50 and 22 per cent per year at age 70;
- the recidivism rate of quitters resuming smoking falls from 50 per cent per year at age 10 to 33 per cent at age 30 and 15 per cent at age 70;
- 10 per cent of quitters become non-smokers each year, implying that it takes ten years, on average, for a former smoker to return to the health status of a non-smoker.

The following formulae and approximations can be used to analyze this model:

$$e_0 = 0.5 + \sum_{k=1}^{100} e^{-\sum_{j=0}^{k-1} \bar{\mu}(j)}, \tag{85}$$

where

$$\bar{\mu}(j) = \sum_{i=1}^3 \pi_i(j) \mu_i(j), \tag{86}$$

where

$$\pi_i(j) = \frac{p_i(j)}{[p_1(j) + p_2(j) + p_3(j)]}, \tag{87}$$

where the  $p_i(j)$ , the proportions of the original cohort that are in state  $i$  at time  $j$ , are given by

$$p_1(j) = p_1(j-1)[1 - r_{12}(j-1) - q_1(j-1)] \\ + p_3(j-1)[1 - r_{31}(j-1)], \quad (88)$$

$$p_2(j) = p_2(j-1)[1 - r_{23}(j-1) - q_2(j-1)] \\ + p_1(j-1)[1 - r_{12}(j-1)] + p_3(j-1)[1 - r_{32}(j-1)], \quad (89)$$

$$p_3(j) = p_3(j-1)[1 - r_{31}(j-1) - r_{32}(j-1) - q_3(j-1)] \\ + p_2(j-1)[1 - r_{23}(j-1)], \quad (90)$$

where

$$r_{ik}(j) = 1 - e^{-\lambda_{ik}(j)} \quad (91)$$

and

$$q_i(j) = 1 - e^{-\mu_i(j)}. \quad (92)$$

With the parameter values given above, remaining life expectancy at age 10 is 61.5 years. The proportion of the surviving population that smokes rises to 33 per cent at age 30 and then falls off to 23 per cent at age 50 and six per cent at age 70.

The model can be used to explore various kinds of interventions. If no one ever smoked, or if the health hazards of smoking were eliminated, life expectancy would increase by 1.4 years. If the rate at which people began to smoke were cut in half, life expectancy would increase by 0.6 years. If the rate at which people gave up smoking doubled, the gain would be 0.4 years. If the rate of recidivism could be halved, 0.3 years would be gained; if recidivism could be eliminated, the increase in life expectancy would be 0.7 years. If the duration of the lingering excess risks faced by former smokers could be cut from an average of 10 years to an average of five year, 0.3 years would be added to life expectancy. Finally, if the excess risk of smoking were cut in half, so that  $\mu_2$  equalled  $1.5\mu$ , rather than  $2\mu$ , about half a year would be gained.



This example provides a simple illustration of how micro-simulation can shed light on models that are difficult to analyze formally. More elaborate, more realistic models for target analysis can be handled in the same general way.

## **7. Conclusion**

The life expectancy of individuals (or units) in a heterogeneous population can be increased by numerous strategies, including

- lowering overall mortality (or failure) rates,
- reducing mortality rates in specific age categories,
- lessening mortality rates from some cause,
- diminishing mortality rates in some region or for some population group,
- decreasing the proportion of individuals in high-risk groups, and
- changing transition rates between risk groups.

As the various formulae derived in this paper illustrate, these changes affect life expectancy in different ways.

The formulae, and various extensions or adaptations of them, may be useful to policy-makers in target analyses of the benefits of alternative interventions intended to save lives. In addition, the formulae describe the linkages that exist between population structure and life expectancy. Individuals differ on numerous dimensions that are related to mortality chances, including age, sex, race, socio-economic status, occupation, place of residence, and personal behaviour. A change in population structure along any of these dimensions will change life expectancy.

The first three approaches we used – the comparative-statics, the dynamics, and the ‘second-chance’ approaches – yield analytical solutions that are general and that may facilitate insight. In the limit, when  $\delta$  is small, the three approaches produce equivalent formulae, so which approach to adopt is to some extent a matter of taste and convenience. The three approaches may not be equivalent, however, when  $\delta$  is not small, and each approach may yield a different insight and provide a different perspective. The fourth approach – computer simulation – is useful in attacking complex models that do not yield to the other three

approaches. The answers produced by simulation pertain to particular realizations of a model in which the coefficients are specified: they are thus not general or elegant, but they are answers.

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