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II. EPIDEMIOLOGICAL TRANSITIONS IN HUMAN HISTORY

*Shiro Horiuchi**

A. INTRODUCTION

Many aspects of human life have changed entirely from prehistoric, tribal societies to today's global high-technology world. Probably our genetic characteristics have not changed substantially, but the length of human life has. The expectation of life at birth has increased from about 20 years for early man (Acsadi and Nemeskeri, 1970; Howell, 1979; Kaplan, 1997; Preston, 1995) to nearly 80 years in the countries with the lowest mortality levels today.

Underlying this spectacular fall in the *level* of mortality were fundamental shifts in the *pattern* of mortality. The path from 20 years to 80 years of life, and to an even longer life expectancy in the future, can be viewed as a sequence of mortality regimes, each of which has a distinct cause-of-death profile. A shift between two different regimes may be called an "epidemiological transition."

Originally, the concept of "epidemiological transition" was proposed to indicate a particular set of changes in the pattern of mortality and morbidity (Omran, 1971). In this paper, however, the term will be defined more broadly and Omran's epidemiological transition will be considered the second in time among five epidemiological transitions in the human history. Since epidemiology is the study of distribution of diseases, injuries, and their risk factors, an "epidemiological transition" means a long-term change in the overall distribution of diseases, injuries, and their risk factors. In particular, the focus of this paper will be on transitions that significantly alter the distribution of deaths by cause and/or by age.

Major cause-of-death categories used in this paper are "infectious diseases," "degenerative diseases," and "external injuries," together with two main subcategories of degenerative diseases, "cardiovascular diseases" and "cancers." The first three categories essentially represent the three "level-one categories" in the Global Burden of Diseases (GBD) study (Murray and Lopez, 1997): Group 1 includes infectious and parasitic diseases, maternal and perinatal disorders, and nutritional deficiencies; Group 2 comprises non-communicable diseases; and Group 3 consists of injuries, poisoning, toxic effects, and other external causes, whether intentional or accidental.

This paper presents a conceptual framework, in which historical changes of the human mortality pattern in the past and future are summarised as five epidemiological transitions. Three transitions occurred in the past: from external injuries to infectious diseases, from infectious diseases to degenerative diseases, and most recently, the decline of cardiovascular disease mortality. Two more transitions are expected to take place in the near future: the decline of cancer mortality, and slowing of senescence. The typical order of these transitions is shown in figure 1. (Note that the "major cause of death" in figure 1 does not necessarily mean the most frequent cause of death in the population, but rather indicates a cause of death that is considerably more prevalent in the era than in the other eras. Thus the cause of death *characterizes* the mortality pattern of the society).

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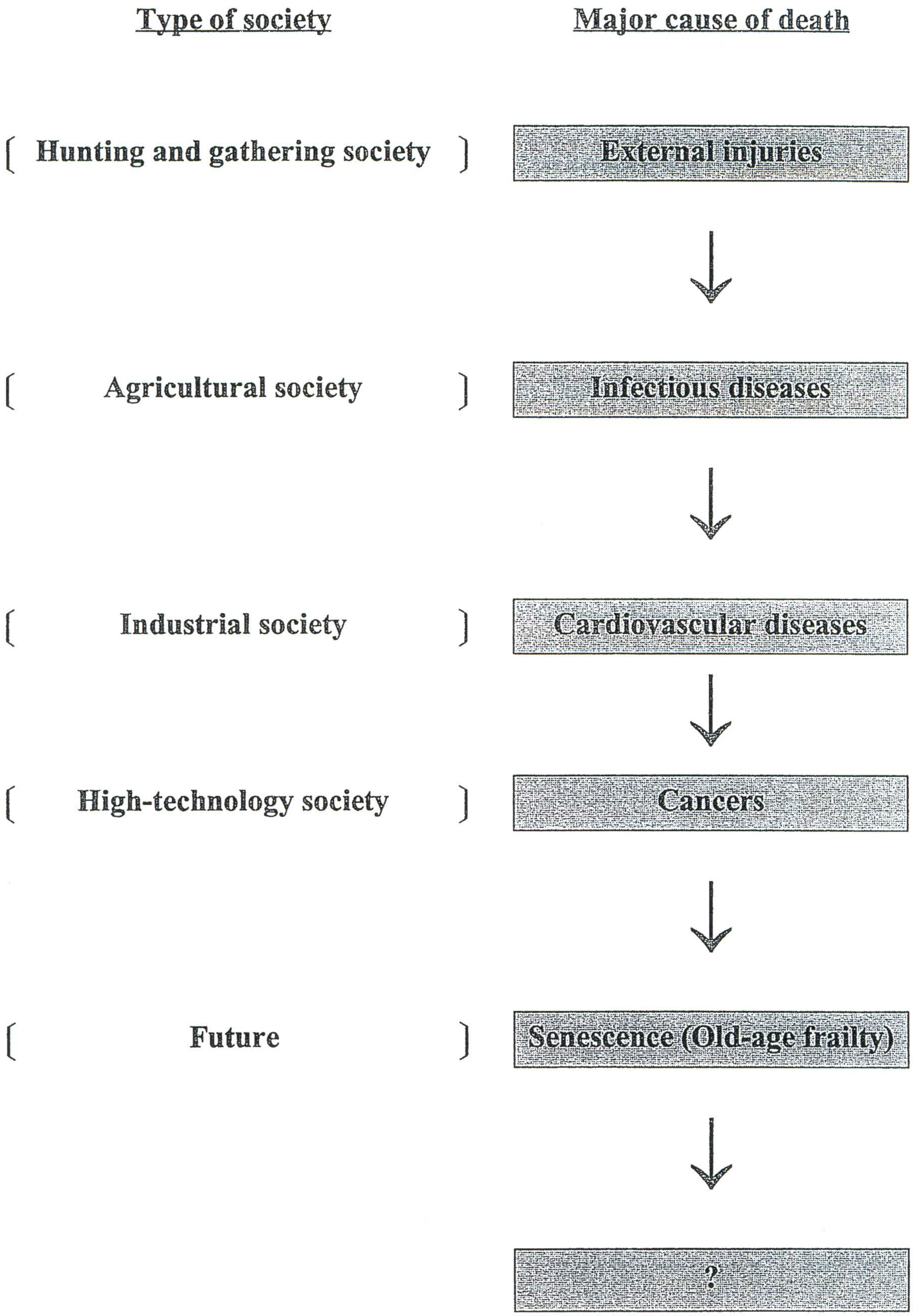
Figure 1. Epidemiological transitions in the human history



B. Epidemiological transitions

1. *First transition: external injuries to infectious diseases*

A number of communicable diseases were endemic or occasionally became epidemic in pre-industrial agricultural societies. Many of them, however, were rare or less serious in small, isolated tribes of early humans, who engaged mainly in hunting and gathering (Black, 1980). It is suspected that a high proportion of the early human population died from external injuries, comprising various kinds of accidents and homicide (Lancaster, 1990, chapter 1; also see Brothwell, 1967). Causes of death that are rare now but probably were not uncommon in those days include attacks by carnivores, drowning, intertribal war (Keeley, 1997), and infanticide. Natural disasters, starvation, and complications of pregnancy and childbirth are also considered to have been prevalent causes of death.

Agriculture started some 10,000 to 12,000 years ago. It diffused widely in the period between 8,000 BC and 4,000 BC and was accompanied by various changes in human life, including diet, dwelling and habitat, social structures, and population size (Cohen, 1995, chapter 3). A fundamental shift in the pattern of health and mortality took place as well (Austad, 1997, chapter 3). The change in the major means of survival from hunting and gathering to agriculture reduced risks of various accidents and risks of homicide by other animals. However, some characteristics of agricultural societies made it easier for various pathogens to remain continuously or diffuse effectively in human populations. These factors include greater population size of communities, higher population density in broad geographical areas that have multiple communities, longer periods of residence at the same locations (often with domesticated animals), storage of foods, domestication of some animals, and extended contacts with other communities. The emergence of urban communities must have amplified these risks. In addition, nutritional deficiencies due to decreased dietary diversity in agricultural societies might have made humans more vulnerable to infections than in hunting-gathering tribes. Infectious and parasitic diseases had become the dominant cause of human mortality. This pattern of mortality then predominated until the latter part of the present millennium.

2. *Second transition: infectious diseases to degenerative diseases*

Limited historical data on mortality in the eighteenth century suggest that the expectation of life at birth in European countries was usually in the range of about 25 to 40 years, with substantial regional variations. By the mid-twentieth century, the life expectancy for all European countries combined reached the neighbourhood of 65 years. In particular, the gain in the life expectancy during the first half of the twentieth century was spectacular (United Nations Secretariat, 1962, table III.4).

This gain was largely due to reduction in mortality from infectious and parasitic diseases, as well as maternal, perinatal, and nutritional disorders (Omran, 1971; Preston, 1976; United Nations Secretariat, 1962, chapter 5). In France, the age-standardised death rate due to infectious and parasitic diseases (most notably, tuberculosis) declined between 1925 and 1955[1] by 79 per cent for males and 87 per cent for females, and the age-standardised death rate due to complications of pregnancy and childbirth fell by 71 per cent for females (Vallin and Meslé, 1988).[2]

This transition can be divided into two phases. The first phase is the reduction of crisis mortality. Demographic crises occurred irregularly but not necessarily infrequently in the nineteenth century and before. The mortality level rose suddenly due to disasters such as harvest failures or pandemics. In Europe, episodes of the crisis mortality became less frequent toward the late nineteenth century (Perrenoud, 1991). This reduction might not have had significant impacts on the mortality level in ordinary (non-crisis) years, but lowered the average level of mortality for relatively long periods. The Spanish Influenza epidemic of 1918 may be the last episode of the traditional type of mortality crisis in European countries.

In the second phase, the mortality level of regular years showed a gradual decline. This shift occurred mainly in the second half of the nineteenth century and the first half of the twentieth century, although the exact timing varied greatly among countries. As discussed later, this phase can be split further into two periods that have different age patterns of mortality reduction. The reason for the secular mortality decline was multi-factorial. Some researchers emphasise the impact of the improved standard of living, in particular, nutrition (Fogel, 1994; Fogel and Costa, 1997; McKeown, 1979), whereas others focus on the importance of public health measures and personal hygiene (Morel, 1991; Preston, 1990; Preston and van de Walle, 1978).

The reduction of infectious disease mortality shifted major causes of death from infectious to degenerative diseases including heart diseases, strokes, cancers, diabetes mellitus, chronic liver diseases, and chronic kidney diseases. Generally, death rates due to these degenerative diseases rise steeply with age.

The age-standardised death rate due to all cardiovascular diseases combined declined in France between 1925 and 1955 by 27 per cent for males and 34 per cent for females. This decrease was significantly slower than the reduction of infectious disease mortality. As for neoplasms, the death rate increased by 52 per cent for males, and decreased slightly, only by 4 per cent, for females in the same period. (These cancer mortality trends may be partly attributable to improved diagnosis and partly to increased cigarette consumption.) The growing proportion of population at older ages also helps to increase the proportion of all deaths that are attributable to degenerative diseases.

3. *Third transition: decline of cardiovascular disease mortality*

By the mid-twentieth century, considerable reduction in infectious disease mortality, in particular, among children and adults at reproductive ages, had been achieved. On the other hand, the relative reduction of mortality at old ages was modest. This gave rise to a view that, although mortality at young and middle ages can be reduced significantly, mortality at old ages cannot, because senescence is the inevitable fate of every human organism. It was argued that since mortality rates at young and middle-ages had already fallen to very low levels, the potential of human life prolongation had largely been exhausted. Thus, life expectancy was close to its upper limit (Bourgeois-Pichat, 1978; Fries, 1980; Gavrilov and Gavrilova, 1991).

An unexpected breakthrough occurred, however, in the late twentieth century. Economically developed countries entered a "new stage of epidemiological transition" (Olshansky and Ault, 1986; Rogers and Hackenberg, 1987). The transition is characterised by reduction of mortality due to degenerative diseases, in particular, cardiovascular diseases. The decline of cardiovascular disease mortality was generally slow in the 1950s and 1960s, but accelerated in the 1970s (Uemura and Pisa, 1988). Total (all-cause) death rates at oldest ages (80 and over), which had previously been stagnant (probably for centuries and possibly for millenniums), also commenced a considerable decline. The onset of the oldest-age mortality reduction occurred between the late 1950s and the early 1980s, earlier for females than males (Kannisto, 1994, 1996; Kannisto and others, 1994).

This was not only due to an extension of life of the morbid or disabled elderly, but also due to improvements of health conditions of the elderly. The increase of life expectancy in the United States during the 1980s was mainly due to longer years of life without disability (Crimmins, Saito, and Ingegneri, 1997). The prevalence of disability decreased among the elderly in the United States in the 1980s and the early 1990s (Manton, Corder, and Stallard, 1997).

Reasons for this health improvement are not limited to technological advancements in curative medicine. Publicly supported health care systems have developed. Hypertension became less prevalent partly because of antihypertensive drugs but partly because greater availability of fresh foods and diffusion of refrigerators changed diet patterns and reduced salt intake. Regular medical check-ups help detect hypertension and high cholesterol level in early stages. Other relevant health trends with long-term impacts include vitamin D supplementation, thermal inactivation of atherogenic viruses in commercial food processing, and diminished prevalence of virulent streptococcal strains that cause chronic rheumatic heart diseases (Manton, Stallard, and Corder, 1997).

For most developed countries, the period of the second epidemiological transition and that of the third epidemiological transition are fairly distinguishable. In Sweden, the cut-off point of the two transitions was in the late 1950s (Horiuchi and Wilmoth, 1998). In the United States, the second transition virtually ended around 1954 and the third transition started around 1968. During the inter-transition period of 1954-1968, the mortality decline was stalled (Crimmins, 1981). In contrast, the timing of the two epidemiological transitions heavily overlapped in Japan after the Second World War, thereby raising the life expectancy very rapidly (Horiuchi and Wilmoth, 1998). Similar patterns of two *simultaneous* epidemiological transitions probably characterise the fast mortality reductions in the newly industrialised countries in Eastern and Southeastern Asia during the recent decades.

4. *Fourth transition: decline of cancer mortality*

How far can the length of human life be extended in the next century? Will it soon face the practical limit of increase, perhaps around 85 years of the life expectancy, or will it reach 90 years and even approach 100 years? The prospect varies, depending on theories and interpretations of empirical evidence (Manton and Stallard, 1996; Manton, Stallard, and Tolley, 1991; Olshansky and Carnes, 1996; Olshansky, Carnes, and Cassel, 1990; Vaupel, 1997; Wilmoth, 1998). For life expectancy to be lengthened significantly in the future, a continuation of the current transition may not be sufficient. New types of epidemiological transitions need to take place. Cancer's turn should be next.

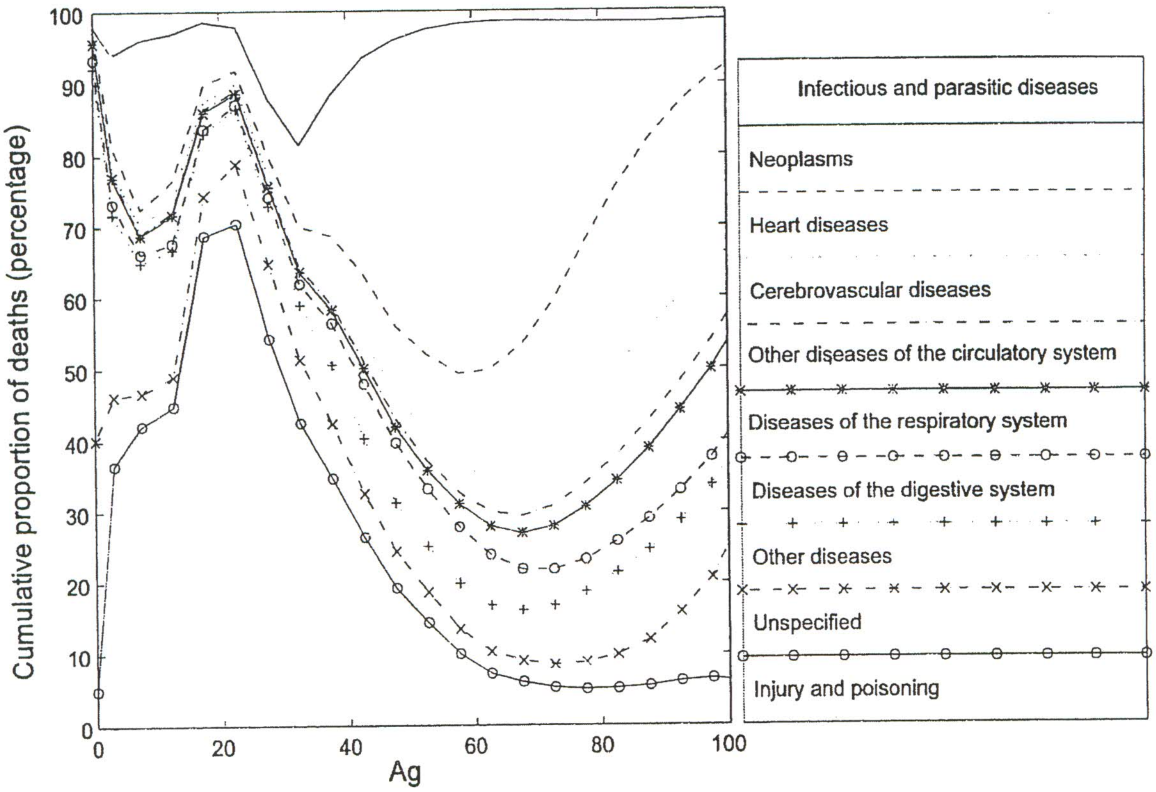
In contrast to the decline in mortality due to cardiovascular diseases, mortality due to cancers did not show a substantial reduction in developed countries during the last few decades. The trend varies considerably by the site of cancer, among countries, and between sexes (Coleman and others, 1993). For some types of cancers, incidence trends and mortality trends exhibit notable discrepancies, and period data and cohort data show different aspects of cancer trends. In general, downward trends seem dominant for cancers of the colon, rectum, bone, cervix uteri, testis, bladder, and thyroid, as well as Hodgkin's disease. The reduction of stomach cancer has contributed significantly to prolongation of the life expectancy. However, trends in cancers of the larynx, lip, tongue, oesophagus, pancreas, and ovary, are either flat, unclear, or divergent among countries. Upward trends prevail among cancers of the mouth, pharynx, skin (melanoma), breast, prostate, and kidney, as well as Non-Hodgkin's lymphoma and multiple myeloma. Lung cancer mortality is still on the rise in many countries, although the trend appears to be changing in countries where smoking is becoming less prevalent.

When causes of death are grouped into very broad categories, cancer is the second most prevalent cause of death, next to heart disease, in the majority of developed countries, and the dominant cause of death in several developed countries. Furthermore, cancer deaths are common at *younger* old ages, as shown later (figure 2). Thus, if the reduction of cancer mortality is substantial, so will be the increase of life expectancy.

A long-awaited decline in total cancer mortality has finally started around 1990 in Canada (McLaughlin and others, 1997), the United States (Cole and Rodu, 1996) and the European Union (Levi and others, 1997). The downward trend is notable for both cancers that can be caused by smoking and cancers that are not closely related to smoking. Whether this is a temporary phenomenon or marks the beginning of a long-term decline remains to be seen.

Breakthroughs may occur when the progress in basic research on cancer (for example, studies on such subjects as telomere shortening, angiogenesis around tumor cells, DNA damage, and oncogenes and tumor suppresser genes), which is greatly enhancing our understanding of the cancer mechanisms on the cellular and molecular levels, leads to new medical technologies. The challenge of cancers, however, seems biologically more fundamental than the challenge of cardiovascular diseases. Although coronary heart diseases are peculiar to humans and rare in other animals, cancers at old ages are found widely among mammalian species.

Figure 2. Age-related changes in the distribution of death by cause in France, both sexes combined, 1990-1994



Source: Institut National d'Études Démographiques mortality database

5. *Fifth transition: slowing of senescence*

What will become the most frequent causes of death if we succeed in reducing both mortality from cardiovascular diseases and from cancers to fairly low levels? The list of likely candidates includes acute pneumonia and bronchitis, influenza, acute gastro-enteritis, and congestive heart failure. Deaths that are difficult to classify by specific medical cause may also increase. Such deaths will occur mostly at very old ages.

The distribution of deaths by cause varies with age for both sexes in France, 1990-1994, as shown in figure 2. The age pattern helps us to reflect on major causes of death after the third and fourth epidemiological transitions. Around age 20, some 70 per cent of deaths are due to external injuries (including poisoning) in accidents, homicide, and suicide. The relatively high proportion of deaths due to infectious diseases in the late 20's and 30's suggests substantial impacts of acquired immuno-deficiency syndrome (AIDS) on mortality in this age group. Cancers dominate the "younger old ages". Nearly half of deaths in the 50's and 60's are due to neoplasms. The most frequent cause shifts during the 70's from cancers to cardiovascular diseases, which dominate "older old ages." Indeed, it has been observed that cancers are quite rare among centenarians (Allard and others, 1996). More than 40 per cent of deaths in the 80's and 90's are attributed to cardiovascular diseases.[3]

Figure 2 indicates that the proportion of deaths without specifically identified causes, including "senility without mention of psychosis" (797 in ICD9), increases steeply with age from 3 per cent in 65-69 to 14 per cent in 95-99. This may be partly due to the tendency for persons at older ages to die from multiple diseases, which makes it difficult to select the primary cause of death. However, it also seems to indicate that a number of deaths at very old ages may occur without clear manifestations of any specific diseases. In a review of autopsy findings for two-hundred persons above age 85, no specific cause of death could be identified for more than 30 per cent of the cases (Kohn, 1982). It should be noted that deaths without identifiable causes in autopsy are not necessarily "natural" deaths without diseases. Schneider and Brody (1983) argue that for even a very old person to die there must be "a specific pathologic insult". At very old ages, the insult could be too small for the physician to detect.

Figure 2 also shows that the proportion of deaths due to diseases of the respiratory system rises steeply with age, from 4 per cent in 60-64 to 12 per cent in 95-99. Included in this category are pneumonia, bronchitis, and influenza. This is consistent with the finding of a recent study on age-patterns of cause-specific mortality (Horiuchi and Wilmoth, 1997). The study revealed that although death rates due to most diseases rise with age, the pattern of age-related increase differs considerably between diseases. Three major types of age pattern are identified. First, for most cancers, the death rate rises with age very steeply in the middle years, but slows down markedly at older ages. Second, death rates due to ischemic heart disease and infarctive stroke tend to increase with age at relatively constant high rates, though the rises eventually slow down at very old ages. Third, death rates due to some diseases rise slowly in the middle years, but markedly accelerate at later ages. These diseases include pneumonia, bronchitis, influenza, gastro-enteritis, and heart failure. The increased importance of these diseases is consistent with the observation that the hospitalisation cost declines with age after the peak in the 70s (Perls and Wood, 1996). Treatment of these diseases is generally less expensive than that of coronary heart disease or cancer. At the oldest ages, the rate of relative mortality increase with age ("life table ageing rate") is higher for these diseases than most others.[4]

Deaths at very old ages due to these diseases or without specified causes may be considered direct manifestations of senescence, or the "state of non-specific vulnerability" of old age (Gavrilov and Gavrilova, 1991). Persons with favourable endowments and healthy life styles have better chances to survive the risks of atherosclerosis, hypertension, and malignancy. However, as "normal ageing" proceeds, some immune functions deteriorate, thereby making the persons more vulnerable to infections that they would control easily at younger ages (Miller, 1995). The ability to pump the sufficient amount of blood out of the heart weakens with age, leading to congestive heart failure. Many other physiological functions also deteriorate with age (Masoro, 1995). If these declines result in a death without clear manifestation of any disease, the cause of the death is difficult to specify.

Can the senescent processes be slowed and deaths due to "old-age frailty" be delayed? Although some data suggest health conditions of the elderly have markedly improved in recent decades, this transition is considered to be in a very early stage, if it has indeed even started. The proportional mortality decline during the last few decades has been substantial at old ages in general, but relatively small in the 90s. For ages 75 and above, there has been a notable tendency for the decline to be smaller at older ages (Kannisto, 1996, figure 5; Wilmoth, 1997, figure 3.9).

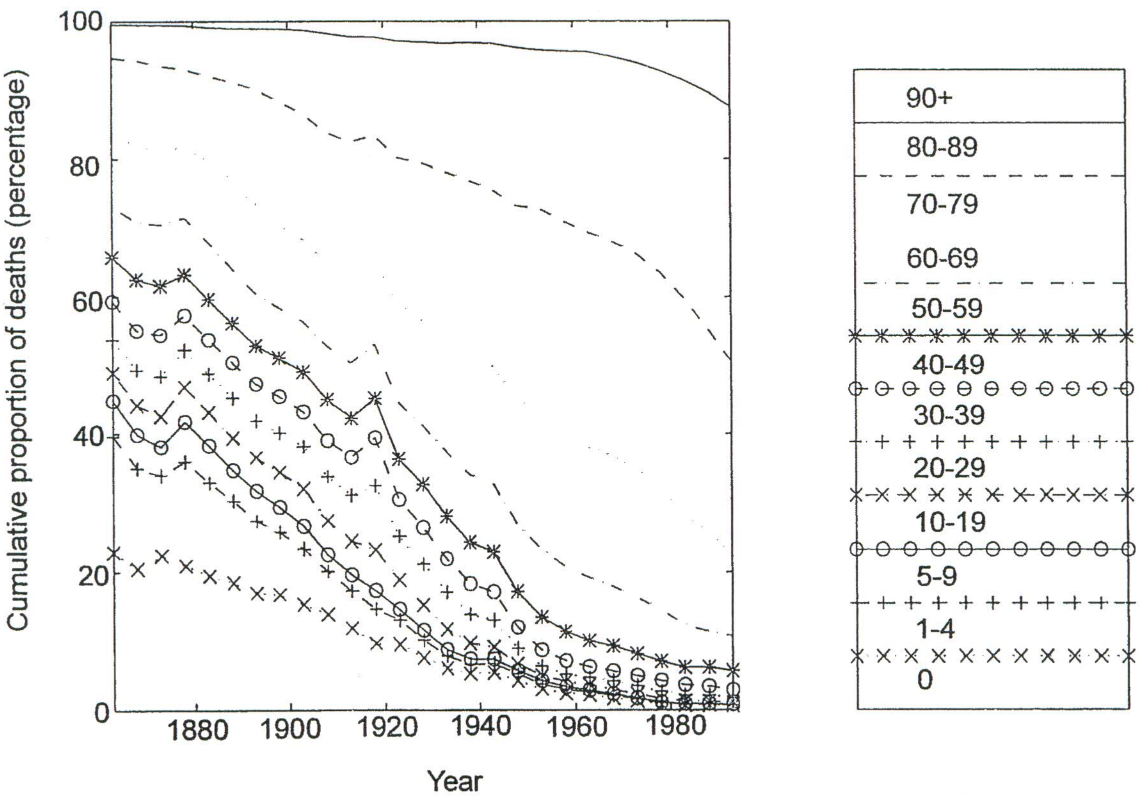
Nevertheless, there are at least three reasons for optimism. First, the slowing-down may be achieved, though probably to a more limited extent, by adopting and keeping healthy life styles. There is much direct and indirect epidemiological evidence that exercise, a low-calorie and low-fat diet that includes a variety of vegetables and fruits, no smoking and moderate drinking, avoidance of excessive mental stress, and active participation in social interactions help to slow the age-related physiological deterioration and/or keep the person free of diseases and disabilities of old ages. Second, benefits are expected from further development, wider availability, and more efficient use of medical technologies for preventing incidence and progression of degenerative diseases (Butler and Brody, 1995). Thirdly, gerontological research is exploring the fundamental biological mechanisms of senescence (for example, free-radical damages, replicative senescence, cross-linking of collagen, and senescence-related genes). Findings of the research may eventually lead to entirely new types of medical technologies that slow senescent processes (Banks and Fossil, 1997; Miller, 1997).

In the traditional evolutionary biology of ageing, it has been theorised that genes that have deleterious effects at old ages may not be eliminated by the force of natural selection (Medawar, 1952), particularly if those genes contribute to reproductive success at younger ages (Williams, 1957). These theories imply the inevitability of senescence. However, recent laboratory studies using nematodes, fruit flies, and rodents show that the length of life of these animals can be extended significantly by experimental manipulations, including caloric restriction, genetic intervention, and temporary exposure to non-lethal levels of radiation, heat, and toxic substance. These results may indicate that in the course of evolution, animals have developed certain mechanisms to raise the ability to resist stress and/or repair damages under special circumstances (Masoro and Austad, 1996; Johnson, Lithgow, and Murakami, 1996; Richardson and Pahlavani, 1994). These mechanisms are inactive in the usual life course, but are activated by some stimuli in the above-mentioned experimental manipulations. Although these laboratory results are not directly applicable to humans, this emerging theory suggests that senescent processes in the human organism may be slowed by activating a latent capacity that is already present in the human body.

C. DEMOGRAPHIC PATTERNS OF THE EPIDEMIOLOGICAL TRANSITIONS

The mortality decline due to the second and third epidemiological transitions shifted the age distribution of deaths to older ages. The reduction in the proportion of children due to fertility decline added to this shift. Figure 3 shows that the age composition of deaths in Sweden changed markedly between 1861 and

Figure 3. Trends in the age distribution of deaths, Sweden, both sexes combined, 1861-199



Source: University of California, Berkeley, mortality database.

1995. In 1861-1865, nearly half of all deaths occurred at ages under 20, but in 1991-1995, almost half of all deaths were at ages 80 and over.

The age pattern of mortality decline differs markedly between the second and third epidemiological transitions (Wilmoth, 1997, figure 3.9). This is reflected in the trends of life expectancies at different ages, as shown in figure 4. The expectation of life at birth in Sweden increased progressively from about 46 years in 1861-1865 to 81 years in 1991-1995 for females and 44 years to 76 years in the same period for males, with the exception of the sudden fall due to the pandemic of Spanish Influenza in 1918 (figure 4A). Life expectancies at other ages, however, did not necessarily follow similar trajectories.

For the following analysis, it is useful to consider the temporary expectation of life, which is the number of years between any two ages, x and y, that are expected to lived by a person at exact age x. Its maximum value is $x-y$.

Mortality of young children continued to fall, as reflected in the trend of temporary life expectancy between age 0 and 10, as shown in figure 4B. The apparent deceleration of the rate of increase in the second half of the twentieth century is simply due to the fact that age-specific mortality rates of young children were approaching zero, leaving little room for further significant improvements.

The trend of the temporary life expectancy between age 10 and 60 (figure 4C) was very different from that between age 0 and 10. It increased slowly in the late nineteenth century and early twentieth century, then rose very steeply during the second quarter of the twentieth century, reflecting the substantial reduction of tuberculosis mortality among young adults. After the mid-century, the progress slowed down. The temporary life expectancy between age 10 and 60 even decreased slightly for males in the 1960s and 1970s.

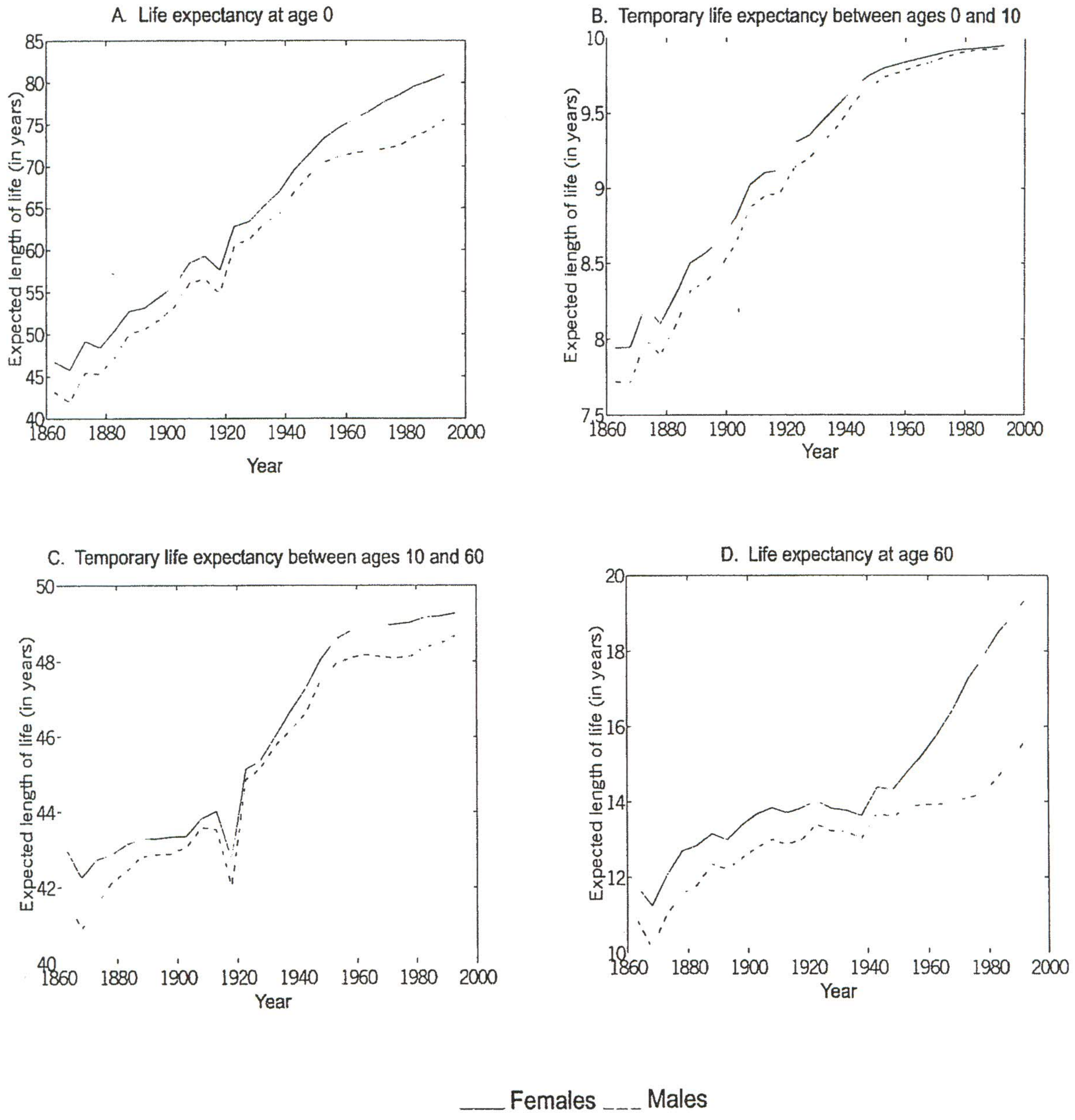
The expectation of life at age 60 followed another, entirely different trend (figure 4D). It was relatively constant, around 14 years for females and 13 years for males, during the first half of the twentieth century. In females, it suddenly began to increase in the 1950s. The steep rise continued during the second half of the twentieth century, the era of the third epidemiological transition. Now it exceeds 19 years. In contrast, the life expectancy of males at age 60 remained nearly constant around 14 years during the 1950s, 1960s, and 1970s, then began to ascend notably in the 1980s.

In summary, the differential time trends of age-specific life expectancies in Sweden suggest that: (1) in early stages of the second epidemiologic transition, mortality reduction was pronounced for infants and young children, but not necessarily for young and old adults; (2) in later stages of the second transition, the mortality level decreased substantially among adults at reproductive ages, mainly due to the reduction of tuberculosis mortality; (3) in the third epidemiological transition, a marked mortality decline was seen among the elderly, mainly due to the reduction of cardiovascular disease mortality, though with a notable difference between females and males.

Why did this sex differential emerge? International time-series data show that the excess of male over female mortality has increased substantially in the twentieth century. The sex difference (female minus male) in the expectation of life at birth in developed countries was typically about 2 to 3 years around the beginning of the century, but 5 to 8 years in recent decades (United Nations Secretariat, 1988). The discrepancy expanded rapidly after 1960, in particular, during the 1970s (Vallin, 1983). Figure 5 indicates that sex ratios (male over female) of age-specific death rates in Sweden increased steeply between 1931-1935 and 1981-1985.

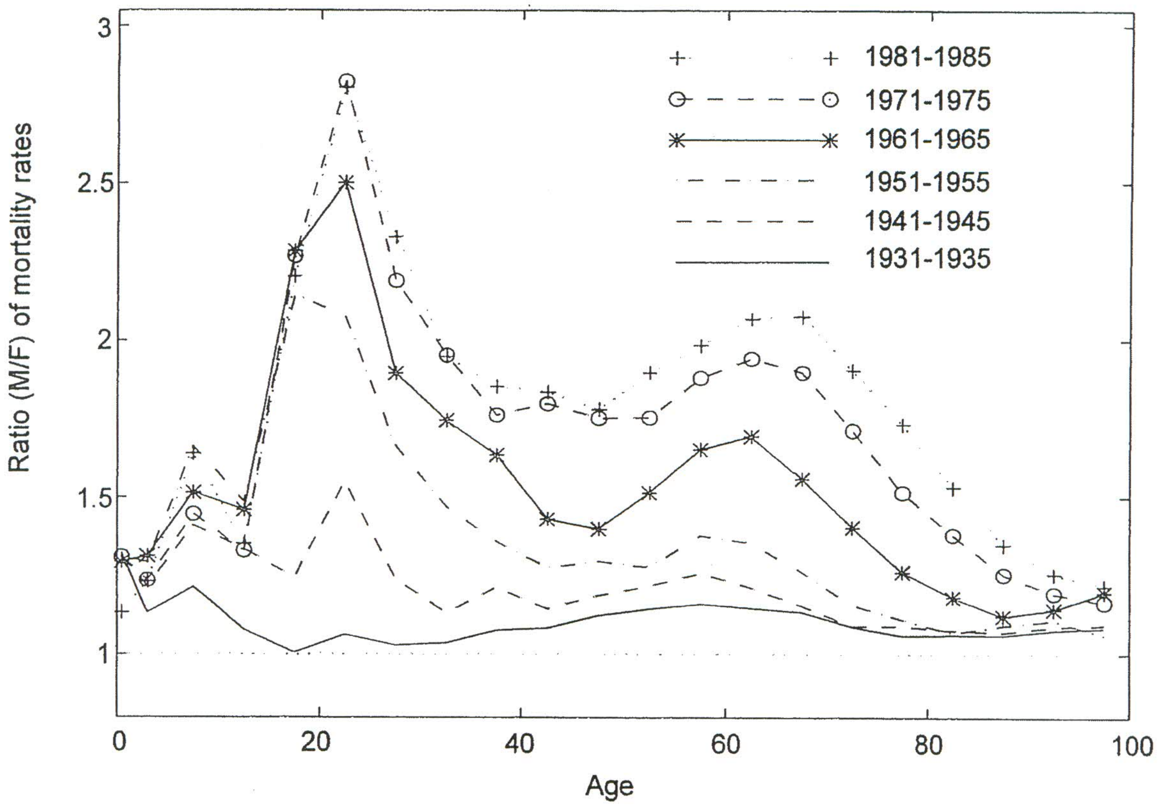
Life table ageing rate analysis reveals that the age pattern of old-age mortality differs considerably between males and females in industrialised countries during the late twentieth century (Horiuchi, 1997, figures 1-3). In the model schedules of old aged mortality for industrialised countries constructed by Himes, Preston and Condran (1994), notable differences are found between the male and female patterns. Swedish data indicate, however, that females and males had similar age patterns of mortality in the nineteenth century. The sex difference evolved as the age pattern of male mortality changed appreciably (Horiuchi and Wilmoth, 1998, figure 4D).

Figure 4. Trends in the expectation of life in Sweden, for five-year periods from 1861-1865 to 1991-1995



Source: University of California, Berkeley, mortality database.

Figure 5. Trends in the sex ratio (male/female) of age-specific death rate, Sweden, selected year



Source: University of California, Berkeley, mortality database.

The contribution of specific causes of death to the increasing sex mortality differential varies among countries, but typically the divergence is due mainly to cardiovascular diseases and cancers (most notably, lung cancers), and to lesser extents, liver cirrhosis and diabetes mellitus, as well as external injuries (Lopez, 1983; United Nations Secretariat, 1988; Wingard and Cohn, 1990). Mortality trends by sex, age, and cause, together with epidemiological data on sex differences in risk factors, suggest that a major reason for the increasing sex mortality differentials is less healthy life styles (in particular, smoking, and possibly to a lesser extent excessive drinking) of males than those of females (Lopez, 1983; Waldron, 1985, 1986).

D. Reverse transitions

The epidemiological transitions, which generally lower total mortality, may meet counteracting trends that keep the mortality level from declining or even raise it. Such health trends are referred to as "reverse transitions." Two major reverse transitions in the recent history and three potential reverse transitions in the near future merit attention.

1. *Early stages of the industrial revolution*

In the early stages of industrialisation, the poor and sometimes dangerous working conditions of factories and mines and the low standard of living among industrial workers posed new types of health risks (Szreter, 1997). Industrialisation induced further urbanisation and expanded urban slums, which were not necessarily followed by measures to deal with the health problems arising there (Wrigley, 1969, chapter 5). "The positive results which were achieved during the second half of the eighteenth and the beginning of the nineteenth century in reducing the mortality from traditional diseases were cancelled by the rise in mortality from certain diseases associated with the laissez-faire policies of the industrial revolution" (Caselli, 1991). In France, the crude death rate declined appreciably between 1750 and 1845, but the decline stagnated in the following 40 years, the most intense period of early industrialisation (Vallin, 1991).

Although the spectacular decline of tuberculosis mortality in the twentieth century was a major component of the second epidemiologic transition, data from Sweden and Finland suggest that death rates from tuberculosis were relatively low in the eighteenth century and then increased markedly in the nineteenth century (Puranen, 1991). Tuberculosis mortality among adults increased in Norway in the late nineteenth century, and geographical variations of tuberculosis mortality in Sweden during the nineteenth century indicate its strong linkage with urbanisation (Lancaster, 1990).

2. *Unhealthy life styles in the wealthy society*

Economic development in the twentieth century improved the health conditions of the population through various direct and indirect pathways. The increased productivity and the wider and faster distribution of products, however, had some adverse "side effects" as well. Alcoholic beverages and cigarettes are produced in large amounts, distributed widely, and affordable to most people. This makes excessive drinking and habitual smoking possible. The reduction of intense manual labour encourages more sedentary life styles. The high-calorie and high-fat diet that was previously a luxury, has become common. A number of epidemiological studies report that these factors raise risks of a variety of degenerative diseases (for example, Butler and Snowdon, 1996; Cockerham, 1997).

Among the adverse health effects of the modern life styles, the most evident and most detrimental seem to be those of cigarette smoking. It is estimated that 24 per cent of all male deaths and 7 per cent of female deaths in developed countries during the year of 1990 are due to smoking (Peto and others, 1994). Lung cancer, which was relatively rare in the 1930s, has now become one of the leading causes of death. In developed countries, the age-standardised death rate from lung cancer has risen by 170 per cent on average between 1950-1954 and 1988-1990 for men and by 230 per cent for women (Lopez, 1995). Currently, tobacco consumption per adult is declining in developing countries, but increasing rapidly in many developing countries (World Health Organization, 1997).

3. *Emergence and re-emergence of infectious diseases*

The reduction of mortality due to infectious diseases has lengthened human life expectancy considerably. Recent decades, however, witnessed increasing episodes of sudden and unexpected new infectious diseases (Levine and Thacker, 1996; U.S. Center for Disease Control and Prevention, 1994). The most threatening was AIDS; other recent examples include hantavirus pulmonary syndrome, Lyme disease, Legionnaire's disease, haemorrhage colitis, haemolytic uremic syndrome, and Ebola haemorrhagic fever. In addition, some old infectious diseases, including tuberculosis and malaria, have become more virulent, more prevalent, or less controllable.

There are a number of reasons for this emergence or re-emergence of infectious diseases (Morse, 1995; Olshansky and others, 1997). First, a number of infectious diseases were once highly prevalent but then brought under control by effective drugs. For some of these old diseases, new strains of the pathogens that are resistant to those drugs have evolved (Levy, 1998). Some vectors of these pathogens also have developed resistance to drugs for controlling them (typically, insecticides).

Second, technological developments and socio-economic changes make it possible for infectious diseases to spread more quickly than before. Some infectious diseases, if they had emerged previously, would have been contained in a local area for many decades or would have expanded their reach only gradually. However, now they are able to become global epidemics in a relatively short period of time. The major reason for the more rapid disease diffusion is the increased, faster, and wider range of trade, tourism, and migration. More frequent sexual contacts between persons from diverse geographical areas as well as some medical technologies (for example, blood transfusion) may contribute to the disease diffusion.

Thirdly, such rising rates of disease transmission may help the pathogens to evolve to more virulent forms (Ewald, 1994). Only rapidly diffusing diseases can be deadly and still continue to expand at the same time. (Otherwise, the host population of the virulent disease will diminish.)

Lastly, ecological disruptions accompanying economic developments may help new diseases to appear. Economic activities in wild areas (for example, forestry in tropical rain forests) expose humans to the risk of contact with pathogens that are rare outside the areas. Construction of roads, bridges, dams, etc. may transform the geographical distribution of animals that carry infective agents and parasites, thereby creating new chances of their close contacts with humans.

Thus, the recent emergence and re-emergence of infectious diseases are rooted in the interactions among technological advancement, economic development, and biological evolution. In this sense, AIDS may not be an isolated episode of unforeseeable disaster. The risk of a second AIDS-type outbreak seems worthy of serious consideration. Whether this new trend will significantly reverse the second epidemiological transition remains to be seen.

4. *Pollution*

Pollutants from industry, agriculture, transportation, and household activities may contaminate air, water, and soil, and accumulate in edible animals and plants. In houses and other buildings, humans may be exposed to radon, lead, asbestos, and possibly toxic materials in pesticides, paints, and other chemicals used for building maintenance (Ott and Roberts, 1998). The depletion of atmospheric ozone ("ozone hole") increases the danger of exposure to intense ultraviolet radiation.

Effects of pollution on mortality have been documented in a number of situations: examples include some seriously polluted areas such as Minamata and Chernobyl (Harada, 1995; Hubert, 1997), occupational exposure to certain chemicals (Johanson and Olsen, 1998), and daily variations in air pollution (Kelsall and others, 1997). Nevertheless, evidence of significant effects of pollution on national mortality trends of populous countries has been far from solid. The geographical correlation between all-cause mortality and environmental contamination is usually insignificant. For example, although it was suspected that the recent rise of mortality in Russia had been partly attributable to pollution, geographical mortality data failed to support this conjecture (Chen, Wittgenstein, and McKeon, 1996). Nevertheless, notable large-scale mortality effects of pollution may occur in the future if long-term accumulation of environmental contamination exceeds some threshold.

In the United States, the rate of cancer incidence for children under age 15 has been increasing since the early 1970s. This upward trend is difficult to attribute to improved diagnosis and more complete reporting alone. It has been conjectured that the rise may be due to toxic chemicals in the environment (United States Environmental Protection Agency, 1997).

5. *Social alienation*

Various social, economic, and political problems arise in the course of industrial and technological development. These problems sometimes lead to the emergence of socially alienated population segments. A "socially alienated" individual is here defined to be someone lacking (1) firm feelings of belonging to the community and the larger society (other than to criminally oriented groups), (2) values to which he/she strongly commits him/herself, and (3) goals that guide his/her life in constructive manners. The causes of social alienation are complicated and beyond the scope of this paper.

Social alienation might raise the level of mortality through at least four mechanisms.

First, lack of ethical values, lower levels of self-control, depression, self-destructive attitudes, and lack of long-term life plans may increase the incidence of external injuries including homicide, suicide, and accidents. Second low concerns about health care may result in under-usage of medical services and unhealthy life styles, including substance addiction, alcoholism, heavy smoking, nutritional imbalance, and poor personal hygiene. Third, unhealthy life styles during pregnancy and improper health care of young children may raise infant and child mortality, and leave some long-term adverse effects on the health of the surviving children. Finally, any long-term positive effects on health associated with positive-mindedness and constructive attitudes that could enhance neuro-endocrine and neuro-immune functions may be reduced.[5] It may also be noted that the mortality effects of social alienation may not at all be limited to urban slum populations.

These effects of social alienation on the mortality level are not necessarily small. Some cases of unexpectedly high mortality seem to be partly attributable to social alienation: homicide rates are high in some developed countries, in spite of their economic wealth. The age-standardised death rate due to homicide among males in the United States doubled between 1960 and 1974. The level of total mortality is high in some urban slums. A recent study shows that a boy in Harlem, New York City, who reaches the age of 15 has only a 37 per cent chance of surviving to the age of 65 (Geronimus and others, 1996; McCord and Freeman, 1990). The mortality decline stagnated in a number of countries in Eastern Europe and the former Union of Soviet Socialist Republics during the 1970s and 1980s, when various socio-economic problems accumulated, leading to a delay of political reforms (Jozan, 1996; Rychtarikova, Vallin, and Meslé, 1989; Watson, 1995). Mortality due to suicide, homicide, alcoholism, and poisoning in some countries of the former Union of Soviet Socialist Republics rose steeply during the transition period of the early 1990s, when rapid socio-economic changes might have loosened the existing systems of social integration (Shkolnikov, Meslé, and Vallin, 1996).

In the relation between social alienation and mortality, of particular importance seems to be the extent to which members in relatively lower positions in the socio-economic hierarchy find firm, positive "meanings" of their lives.[6] Although this factor is difficult to measure in internationally comparable ways, its effects on the average mortality level of the entire population through various indirect pathways may be significant. In general, more gain in the life expectancy of the population can be achieved by improving the mortality of population segments that have relatively high mortality, rather than by lowering further the mortality level of population segments that already have lowest death rates.

It is not easy to achieve a very high level of life expectancy if members of significantly large segments of the society have difficulties in finding firm, positive meanings of their lives. For a life to be fully lived, it should be worthy of living.

E. Conclusion

This paper has presented a five-stage model of the epidemiological transition of human mortality, and also described some possible reverse transitions. The model is a combination of a generalisation of past patterns and a prospect of future changes. Since the actual pattern of historical mortality change varies among countries and regions, it may not be difficult to find specific cases that do not fit the model well. The main purpose of this model, however, is to provide a bird-eye view of a *typical* course of mortality history in human societies. The model can be used as a conceptual framework for describing, analysing, and predicting actual mortality trends in the past and future.

Notes

[1]1925 is the earliest year for which the time-series of cause-specific mortality estimates in France are available. 1955 is the year of the onset of significant decline in old-age mortality in France (Kannisto, 1994, chapter 9), thus may be considered the beginning of the next transition.

[2]Long-term trends in cause-specific mortality are difficult to estimate, because of relatively frequent changes in the classification of diseases. A notable exception is France, where a considerable amount of research has been done on the adjustment of vital statistics for coding changes (Vallin and Mesle, 1990; Mesle and Vallin, 1996).

[3]Because figure 2 does not reflect the age distribution of deaths, it does not show the actual importance of cardiovas-