NOTES

1 Schwenk, M. A., Reality Orientation for the Institutionalized Aged: Does It Help? *The Gerontologist*, 1979, 19, 373–7.

2 Holden, U. P. and Woods, R. T., *Reality Orientation. Psychological Approaches to the 'Confused' Elderly*, Churchill Livingstone, 1982.

Epidemiology and Community Medicine — John Bond

K. G. Manton, Changing concepts of morbidity and mortality in the elderly population. *Milbank Memorial Fund Quarterly/Health and Society*, **60** (1982), 183–244.

In the previous edition of *Ageing and Society* Bromley, Isaacs and Bytheway[1] reviewed the important book by Fries and Crapo[2] in which they developed a theory of ageing describing the human survival curve as approximately rectangular. The reviewers were not entirely complimentary to the Fries and Crapo thesis and in this lengthy article Manton continues the attack, showing that the rectangular survival curve theory and other existing models and theories of human mortality are inconsistent with national mortality and morbidity data.

Mortality is both a *public issue*[3] and a *private trouble*.[4] For society mortality is a major factor in determining the age structure of the population, knowledge of which is essential in planning health and welfare services. To the individual mortality determines the number of years of life that a person can expect to live, which will influence the way he or she plans a career, retirement and investment goals. The importance and interest surrounding theories of human ageing cannot therefore be under-estimated. This article reviews such theories; examines them in the light of our perception of both the quantitative and qualitative aspects of human ageing; and suggests an agenda for future epidemiological research into the nature and implications of current mortality patterns in the U.S.A.

Current theories of human ageing predict that life expectancy is unlikely to increase much beyond present levels. Two explanations have been proposed. The first suggests that the limitation on life span is due to the cellular processes of senescence and the second suggests that there is increased societal risk of mortality from chronic diseases.

Manton highlights four types of evidence put forward to support the view that senescence will soon limit life expectancy. First, historically, the maximum human life span has not been observed to change except

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in populations where age documentation is poor and the literacy rate is low. Secondly, the risk of death seems to increase as an exponential function of age. Thirdly, standard actuarial computations indicate that the elimination of cancer and heart disease would increase average life expectancy only minimally. Finally, there is experimental evidence which suggests that certain human cells are internally programmed for only a limited number of reproductions.

Within this school of thought there exist two different views on the expected changes in morbidity and mortality patterns. The optimistic view, illustrated by Fries and Crapo's rectangular curve, sees the possibility of postponing the onset of chronic disease. However, as Isaacs[5] indicated in his review, British data do not support this view.[6] In contrast, the pessimistic view suggests that the prevalence of chronic disease and disability will increase as life expectancy is increased. Despite these differences, they share the common perspective that chronic illness and not mortality should be the prime focus of public health efforts.

Manton only briefly reviews the second theoretical perspective which suggests that the limitation on life span is due to increased societal risk of mortality from chronic disease. The historical evidence for this view is the fact that major declines in mortality have resulted from the reductions in infectious diseases due to improvements in life style, hygiene, nutrition and other public health factors and not because of innovations in medical technology. Like the pessimists' view of the biological model this perspective sees little chance of life expectancy increasing through the intervention of medical science in the treatment of chronic disease.

Having outlined these different theoretical perspectives Manton reviews the evidence to determine both the logical and empirical consistency of the models of human mortality described.

The crucial evidence presented in support of the biological model of human ageing concerns the fact that repeated sub-culturing of human fibroblasts eventually produced cells incapable of further division. This phenomenon has been labelled the *Hayflick limit*.[7] However, Manton points out that Hayflick himself was cautious about generalising these data to all human cells, arguing that this total senescence phenomenon need not have a simple relationship to the ageing of the organisms from which the cells were obtained. Manton reports other studies[8] which suggest that the theoretical and experimental results are not conclusive in determining the age at which a Hayflick type of limit becomes operational in the human population. If this is so then the validity of

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biological theories of human ageing rests upon evidence about current patterns of mortality and upon actuarial analysis.

Manton suggests that recent changes in mortality patterns also conflict with the models of human ageing described. In particular he shows that for white males and females there is no evidence of a ceiling on life expectancy for the period 1900–77. He could find no evidence for rectangularised survival curves in these data.

Turning to the prevalence of chronic diseases and disability Manton found that 'though more persons were surviving to advanced ages where chronic diseases should be more prevalent, little evidence existed to suggest that elderly persons of a given age were more disabled than in earlier decades' (p. 198). He recognises that this conclusion is based on national data about disability and health which lack adequate age detail to examine possible mechanisms underlying health changes at more advanced ages.

The middle sections of this article are concerned with reviewing several possible cause-specific measures of disease severity for which age-specific rates are available. Manton (after six pages of complex epidemiological arguments) rejects the standard measure because it confounds 'the survival distribution, either observed or modified by cause elimination, of individuals afflicted with the disease with the survival distribution of persons dying of all other causes of death' (pp. 206–7). He also argues that models of cause-specific mortality need to take more account of the increasing importance of chronic disease.

Multiple-cause mortality data have been obtainable from the U.S. standard death certificate since 1968. These data can be used to examine, at specific ages, the changes over time in the role of chronic disease from an underlying cause of death to an associated cause of death. Such changes could be used to estimate the reduction in the severity of the chronic disease process when associated with a decreasing risk as an underlying cause of death. Manton describes the way in which changes might be measured, using the ratio of the number of death certificates on which the chronic disease was recorded to the number of death certificates on which the disease was the underlying cause of death. His analysis suggests that there is a decrease in the severity of chronic illness, a decrease which occurs at all ages. He concludes that along with 'evidence on longitudinal ageing changes and analysis of the age trajectory of mortality risks, that mortality reductions extend the productive life span of individuals not by eliminating chronic disease, but by reducing its severity at any given age' (p. 217).

This article highlights the absence of suitable models of human

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mortality which adequately explain all the relevant data. Manton suggests therefore that appropriate models of human mortality should be based on several simple principles.

1. The human organism is a complex multicomponent system, each component having its own ageing rate. The death of the individual organism therefore will be determined by the fastest ageing rate of the individual components.

2. The failures of components can be identified with major chronic degenerative diseases. One of the factors in chronic degenerative diseases is the effect of cellular senescence on the physiological capacity of a given component or major organ system.

3. The propensity for failure of individual component systems is only partly dependent on the propensity for failure of other components. This suggests that if an effective intervention can be made to delay the failure of a given component, death of the organism can be delayed. Since the diseases are partially interdependent, reduction in the rate of failure of one component may also help to retard the rate of failure of another component.

4. Mortality is a property of individuals, yet data on the risk of death are always based on the behaviour of populations. Hence, analyses of mortality must link the biological mechanisms of individual ageing and mortality with the measures of population risk (pp. 223–4).

These four principles simply highlight a commonsense judgement that human ageing and mortality are complex phenomena. They illustrate the need to up-date our disease concepts in order to reflect ageing processes. Manton introduces the concept of *dynamic equilibrium* in order to understand the relation of morbidity and mortality as life expectancy increases. This concept challenges the epidemiological law that prevalence is a function of incidence and duration and which suggests that if incidence remains constant, a reduction in mortality leading to increases in life expectancy can only occur as a result of an increase in the duration of the disease. Manton suggests that chronic disease can violate this law since, though prevalence might increase, the average severity of the disease might decrease. In contrast, the concept of equilibrium implies 'that the severity and rate of progression of chronic disease are directly related to mortality changes so that, correlated with mortality reduction, there is a corresponding reduction in the rate of progression of the ageing of the vital organ systems of the body' (p. 227). The concept has important policy implications because mortality reductions are directly linked to the long-term management of chronic disease.

Changes in our understanding of human mortality and of longevity have a number of implications. First, it will affect the way in which demographers undertake population forecasts. Manton's analysis chal-

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lenges the assumption that population ageing is determined by differential birth rates between cohorts. His examination of recent efforts to forecast the size of the elderly population shows that assumptions about mortality have serious implications for the size of the population and projections about rate of increase. Secondly, as this article emphasises throughout, human life span can be extended by controlling chronic diseases and there is increasingly a belief that the process of ageing may itself be controlled. The implications of these trends are that biomedical research should be directed towards identifying ways of preventing ill health and maintaining health in the elderly population. Thirdly, increased longevity has considerable implications for social policy and particularly in relation to retirement policy. Manton forecasts the time when different stages of the life career will require different kinds of jobs or careers.

COMMENT

I enjoyed reading this article because it attempts to relate the various theoretical perspectives of biology, medicine, and demography to social policy. My problem in abstracting this article, however, is that I am unable to examine critically all the ideas presented because of its wide theoretical coverage. Common sense tells me that I should not take all at face value because traditionally we have been led to believe that our life span is based on the biblical threescore and ten. Yet some of the ideas explored in this article, if substantiated, will expose the fallacy of common sense. To believe in common sense might be to believe in the flat earth.

If we can accept Manton's thesis that the human life span will shortly be empirically extended then the public issues surrounding social definitions of old age will be quite considerable. For example, using our present administrative definitions of old age we can foresee a future where perhaps over three-quarters of the population will be old. As Manton rightly points out, the implications for pensions and retirement policies are considerable. My private trouble is, how do I prepare for this millennium?

NOTES

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3 Mills, C. W., *The Sociological Imagination*. Penguin Books, Harmondsworth, 1970.
4 *Ibid.*
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