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Dynamics Of Morbidity And Mortality In Africa And Implications For Health Care Planning

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DYNAMICS OF MORBIDITY AND MORTALITY IN AFRICA AND IMPLICATIONS FOR HEALTH CARE PLANNING

by

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ABSTRACT

The dissertation aims to tie together the empirical, theoretical and policy oriented aspects dealing with the dynamics of morbidity and mortality in Africa. On the theoretical aspect, scholars are arguing that the epidemiologic transition theory which provided the framework for understanding African mortality in the past, is actually part of a broader health transition theory which also includes a health care transition. In this theory, the overemphasis on biomedical interventions is down-played. To understand African mortality, it is argued, we have to consider the social and cultural determinants. Also we have to consider morbidity and its relationship to mortality. This thesis is only considering two aspects of these concerns of health transition theory, namely health care planning and the dynamic relationship between morbidity and mortality.

In order to study the dynamics of morbidity and mortality, a multistate demographic model is used. As data availability in Africa is poor, a straight-forward application of the model is not possible because of its high data requirements. The adopted solution is to construct separate submodels for the indirect estimation of morbidity and of mortality. In the mortality submodel, the relevant age-cause-specific death rates are obtained by synthesizing Preston's cause of death model and an extension of the Brass logit model. In the morbidity submodel, the relevant morbidity rates are obtained by modifying Klementiev's degenerative model to handle lethal infectious disease for which limited recovery is possible.

With the data obtained from these submodels, the multistate (dynamic) model is used to investigate the mechanism of the health transition; that is, the effect of changes in risk factors (affecting incidence rates) and that of health care technology (affecting case fatality rates). With changes in the incidence rates, case fatality rates or recovery rates, the model can then be used to obtain estimates of life expectancies in either the illness states or the well state.

A specific application of the dynamic model is in health care planning. In two hypothetical examples given in the thesis, scenarios are constructed and the application of the dynamic model in aiding in decision making is outlined.

DEDICATION

This thesis is dedicated to the Muslim Youth Association of London for all they taught me and for their warm brotherhood.

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CHAPTER I

INTRODUCTION

1.1 Introductory Remarks

Over the past decade, various researchers on mortality, starting from totally different perspectives, are arriving at a consensus that information on morbidity (quantitative as well as substantive) is very vital in understanding changes in mortality trends. These perspectives range from the empirical to the theoretical to the policy oriented. From the empirical perspective, this recognition gave rise to various attempts at the indirect estimation of morbidity rates. From the theoretical perspective, this recognition resulted in attempts at modification and indeed, reconstruction of existing mortality theories. The theory targeted at was the widely accepted epidemiologic transition theory that was used to explain mortality decline. While the theory was being critically modified, it was also being submerged as a component of a newer theory called the health transition theory. This latter theory places more emphasis on substantive aspects of morbidity than on mortality changes. From the policy oriented perspective, opinions started emerging which questioned health care planning programs which were only mainly aimed at reducing death rates but which did not address morbidity.

With the recognition of the importance of morbidity, a few researchers started to study the relationship between morbidity and mortality. However, most of the work done in this field is in the context of more developed countries and the diseases considered are chronic degenerative diseases. This thesis is a bold attempt to apply this research to the African setting, considering lethal infectious diseases instead of degenerative diseases. Because of the paucity of data in Africa, this implies that the relationship between morbidity and mortality cannot be studied prior to the indirect estimation of the relevant mortality and morbidity rates.

Whenever a real phenomenon is unknown but needs to be studied, the use of models is the first step in that direction. This thesis is an effort in that direction. While the main model proposed here deals with the dynamic relationship between morbidity and mortality, submodels are constructed to estimate the relevant morbidity and mortality rates.

1.2 Structure of Thesis

Since widely different models are to be dealt with, this work is divided into seven chapters and is structured to cater for this diversity. The second chapter deals with the review of literature and includes an outline of the objectives and a brief description of the analytical framework to be used later. It also covers the theoretical developments leading to the awareness of the important role of morbidity and its dynamic relationship with

mortality. The third chapter deals with mortality and causes of death in Africa and its indirect estimation. The fourth chapter deals with morbidity in Africa and its indirect estimation. The fifth chapter is the main chapter; it deals with the dynamics of morbidity and mortality. That chapters seeks to tie together the two previous chapters into a multistate demographic model. Chapter six covers health care planning in Africa. The last chapter presents a summary as well as conclusions of this study.

CHAPTER II

REVIEW OF LITERATURE AND OBJECTIVES OF STUDY

2.1 Introduction to Concepts

The crucial elements to be considered in a study of the dynamics of morbidity and mortality are incidence rates and case fatality rates. While the incidence rates are affected by risk factors, case fatality rates are affected by several factors including health care technology. Hence, a study on the dynamics of morbidity must use risk factors and case fatality rates as the linking thread guiding the research. Before proceeding further, there is need to clarify on the concept of 'dynamics'. This has been used in various contexts. In the context of social dynamics, Hanneman (1988:13) explains the concept as follows:

> 'The dynamics studied by ...sociologists... are composed of multiple simultaneous causal processes, operating along multiple dimensions...Such processes are inherently complicated....In effort to cope,...we progressively simplify problems by assumptions until what remains can be dealt with systematically....At present, theorists concerned with dynamics tend to be divided into two methodological camps....One group utilizes "everyday" language for constructing theories...Another large group of scholars utilize formal mathematical language tools (eg. differentials equations, Markov processes, etc.)...'

From the mathematical perspective, one common usage of dynamics is in 'system dynamics' where differential equations are used to model changes. A computer software called DYNAMO was developed by Pugh (1976) using that approach. Another common

usage of dynamics is in the context of transitions between several states. For examaple, in a study on 'the dynamics of female labour force participation' in the United Kingdon, Wright and Hinde (1991) made use of a three-state employment model and studied the impact that socio-economic factors have on transitions among the various states.

In this work, dynamics refers to interactions or interelationships, whether direct or indirect. In the context of morbidity and mortality, one can see that morbidity clearly affects mortality rates, the reverse however is not very obvious. If a mathematical model is used which expresses morbidity as a function of mortality, then it can be seen that mortality rates affect morbidity ones. From another perspective, when health policies are implimented which affect mortality rates, in some cases, these policies might also affect morbidity rates so in the way, the mortality rates are related to morbidity rates. In the dynamic model proposed here, morbidity and mortality are incorporated in a single model so that it would be possible to oberve how either of them changes when the other is altered or how they change when health conditions such as recovery rates are altered.

The dynamics of morbidity and mortality has both socio-cultural and demographic manifestations. On the socio-cultural level, one issue that has been discussed is the cultural inflation of morbidity during the decline of mortality (Johansson, 1991). After studying morbidity trends in Japan, the United States, Britain and Hungary, Riley (1990) observed that the average duration of sickness had moved in a direction opposite to that of death rate for most age and sex groups. In general, morbidity trends are seen to have

important cultural dimensions which call for caution in relating morbidity changes to mortality changes. Some of the cultural factors that influence reporting of morbidity include:

> ' rising health expectations on the part of ordinary people, including their ability to perceive illness and their willingness to seek professional help, and institutional pressures on medical professionals ....' (Johansson, 1991:39)

and

> '...new scientific theories of health and disease, which permit the continual discovery of new diseases; new techniques for diagnosing and treating diseases' (Johansson, 1991:53)

Thus while inflation of morbidity in developed countries is biologically real, it involves a shift from the reporting of diseases which are fatal or relatively severe to those that are less fatal or non-fatal. By extension, developing countries might also experience this inflation if they follow the path of developed countries in their health transition (Johansson, 1991).

In the case of Sri Lanka where mortality is quite low compared to other developing countries, morbidity rates are reported to be high. There is thus a discrepancy between the health status as inferable from mortality-related indicators and those morbidity-related indicators (Parera, 1985). A similar coexistence between low mortality and high morbidity was also found in Kerala (Krishnan, 1985). In an analysis of the contribution of health care facilities to improvement in mortality in China, Jamison (1985) gave a pessimistic view based on the recognition on the dynamics of morbidity and

mortality. In his view,

> 'As the disease profile moves toward chronic and away from communicable diseases, income improvements are likely to cease having the beneficial effects on life expectancy as they have had in the past.' (Jamison, 1985:29).

The demographic manifestation of dynamics of morbidity and mortality refers to the effect of one on the other either directly or through other population characteristics such as population size and structure, rate of population growth, aging, frailty of the population etc.

Having clarified on the concept of the dynamics of morbidity and mortality, the next task addressed in this work is how this can be linked to health care planning. In order to show on this linkage, one needs to clarify the concept of health care planning. The objectives in health care planning are threefold: i) To reduce morbidity through prevention or cure after the incidence of a disease; ii) To postpone mortality to as late as possible; iii) To give people a sense of quality of life especially those with disabilities or whose conditions cannot be cured with the existing medical knowledge.

These objectives cannot be fully achieved only with knowledge of general mortality. They can be partly achieved with knowledge of cause specific mortality. Cause of death data do serve as good proxies for morbidity in the case of chronic diseases. However, in the case of most infectious diseases, cause of death data alone cannot reveal much about the morbidity aspect of the diseases. According to Johansson, (1991:58)

> 'In general, using only cause-of-death data to study the history of morbidity trends is not a good basis for deriving conclusions about the biological magnitude and direction of changes in disease levels during the health transition.'

Compartmentalized knowledge of mortality, cause of death and morbidity can still be useful when dealing with stationary populations. However, such knowledge will not be sufficient when studying rapidly growing populations undergoing both epidemiologic and health transitions. Rather, we need to know the interaction of morbidity and mortality on the one hand and population on the other hand. Such information will be most useful in health care planning and in assessment of health care programmes. According to Pollard (1990:219),

> 'To establish whether medical science has managed to ensure quality of life during the additional years of life now available in many populations requires a relatively complex interactive model of morbidity and mortality.'

2.2 Introduction to the Literature Review

As this study is largely methodological, the literature to be reviewed in this study are of three groups: the first group is technical and consist of reviews of the various estimation techniques to obtain cause of death rates and morbidity rates and to study the dynamics of morbidity and mortality. The second group consists of work that have been done in Africa relating to these subtopics and the estimates that have been obtained from various studies. Both of these groups of reviews have been included under the specific chapters to follow. The existing models are reviewed with the hope of either adopting

them, modifying them or proposing alternative models. The last group of reviews consist of broad theoretical frameworks within which the study could be placed. These reviews have been included in this chapter.

The reviews in this chapter explore the theoretical background of the thesis in a general manner. To put this study in proper perspective, one uses the framework of the epidemiologic transition theory and the emerging health transition theory. The factors affecting mortality change (for better or worse) are described and the different routes to achieve low mortality are outlined.

2.3 Epidemiologic Transition Theory

A useful framework for the analysis of the decline in mortality from high to low levels is the epidemiologic transition theory. The original formulations of this theory used an evolutionary approach in their descriptions. Fredericksen (1969) described the patterns of morbidity, mortality, fertility and health care services as corresponding to four 'stages' of society: 'traditional', 'early transitional', 'late transitional' and 'modern'. Omran (1971) coined the term 'epidemiologic transition' and gave it a more explicit formulation. The epidemiologic transition theory proposed by Omran (1971) focuses on the complex changes in patterns of health and disease in a society and on their demographic, socioeconomic and biologic determinants and consequences. The theory proposes that disease patterns shift over time so that infectious and parasitic diseases are gradually, but

not totally, displaced by degenerative and man-made diseases as the leading causes of death (Omran,1983). Omran proposed *three major successive stages* in the transition: the age of pestilence and famine, the age of receding pandemic and the age of degenerative and man-made diseases. In progressing from one stage to another, mortality declines and life expectancy increases.

Omran further proposed *four models* to account for the different variations in pattern, pace, determinants and consequences of population change in different societies. The *first model* is the classical or Western model which describes the gradual, progressive transition from high to low mortality that accompanied the process of modernization in most western European societies.

The *second model* is the accelerated model which describes the mortality transition similar in pattern to the classical model but was accomplished in a much shorter period, as in countries like Japan, Eastern Europe and the former Soviet Union.

The *third* is the contemporary or delayed model which describes the relatively recent transition still in process in many developing countries, where substantial declines in mortality started mostly after the 1940's and was largely attributed to modern medical technology such as mass use of chemotherapeutic agents, antibiotics and insecticides. Mortality in these countries is still high, socio-economic differentials still persist and recent evidence show marked slow-down in decline in mortality and even stagnation in

some cases (Secretariat of the WHO, 1984).

The *last model* is the transitional variant of the delayed model which describes developing countries, where the rate of decline was initially similar to that in the contemporary model and was sustained especially in the case of infant and child mortality. These countries include, Taiwan, South Korea, Singapore, Hong Kong, Sri Lanka, Mauritius, Jamaica, Kerala state in India, Cuba and Costa Rica.

After Omran's initial formulation and subsequent elaborations of the epidemiologic transition theory, many works were done on applying its theoretical framework to analyzing mortality change in several countries and among several population subgroups. The results of these researches had three outcomes: First, some confirmed the applicability of the framework, with some exceptions (Broudy and May, 1983; Bah, 1993b); second, some called for an additional stage, namely the fourth stage, to be added to Omran's three stage theory (Olshansky and Ault, 1986; Rogers and Hackenberg, 1987); and lastly, some criticized the evolutionary approach employed by all the formulations of the theory and called for a major modification of the theory (Frenk *et al.*, 1989; Soberon *et al.*, 1986; Sepulveda *et al.*, 1987).

Concerning the proposal for an additional stage to Omran's three stages, Olshansky and Ault (1986: 360) give the general characteristics of the fourth stage as including the following:

" (1) rapidly declining death rates that are concentrated mostly at advanced ages and which occur at nearly the same pace for males and females; (2) the age pattern of mortality by cause remains largely the same as in the third stage but the age distribution of deaths for degenerative causes are shifted progressively toward older ages; and (3) relatively rapid improvements in survival being concentrated among the population in advanced ages." They remark that the major characteristic that distinguishes the fourth stage from the third is the unexpected shift in the age pattern of mortality by degenerative causes for the population in advanced ages. In short, "in this fourth stage, the major degenerative causes of death that prevailed during the third stage remain with us as the major killer, but the risks of dying from these diseases are distributed to older ages." (Olshansky and Ault, 1986: 361). They attribute the source of this change to a combination of factors including shift in the age structure toward older ages, advances in medical technology, health care programs for the elderly and reductions in risk factors at population level.

The fourth stage suggested by Rogers and Hackenberg (1987) is more descriptive. One of Omran's propositions is concerned with the existence of mortality differentials among several population subgroups. For example, the epidemiologic transition favours females over males, the young over the old and, in the United States, whites over non-whites. Another proposition of Omran's theory is that infectious diseases will decline but will not be entirely eradicated. Rogers and Hackenberg provide evidence from the United States data to show that these propositions need to be changed or modified. They

agree with Olshansky and Ault that the leading causes in this stage are still degenerative and man-made diseases, but disagree with them in identifying the source of this development. They argue that the major source of this development is the increasing influence of individual behaviours and new lifestyles on mortality. This influence may either be positive such as in areas where measures of health promotion are effective or negative such as in areas where potentially destructive lifestyle practices are widespread. On the negative side, individual behaviours and destructive lifestyle practices include sexual orientations and social pathologies like accidents, suicides, homicides, excessive drinking and smoking. Even though Acquired Immunodeficiency Syndrome (AIDS) is an infectious disease, Rogers and Hackenberg consider it a disease belonging to the fourth stage because of its direct relation to an individual's behaviour and lifestyle. They argue that the root cause of these destructive lifestyle practices is "hybris"- an excessive self confidence, a belief that one cannot suffer and that one is invincible. Hence they refer to this fourth stage as the "hybristic stage."

Upon analyzing Canadian epidemiologic transition, Bah and Rajulton (1991) argue that these two formulations of the fourth stage are not exclusive of each other. Rather, they reflect different aspects of this new stage of mortality which is characterised by an interplay between the age-cause patterns of mortality, micro-level determinants such as individual behaviour and social lifestyles, and macro-level determinants such as health care and health promotion programmes. Generally, advances in medical technology help delay death to very old ages. Health promotion programmes, however, affect all ages, not

necessarily the oldest ages. They help reduce preventable deaths and morbidity primarily through modifications of individual behaviours and lifestyles. However, certain causes of death are primarily determined by potentially destructive individual behaviours and lifestyles and are products of man-made environment.

In criticizing Omran's formulations, Frenk *et al.*, (1989) argue that there is a third group which is undergoing a new transition experience quite different from that of the developed nations. They describe such countries as belonging to the 'protracted and polarized' transition model. This model is characterised by the following features: (1) Overlap of eras - the stages of the transition do not follow a sequential order but may co-exist at the same time and place. (2) Counter-transitions- the shift from high mortality to high morbidity occur not only for degenerative diseases but also for infectious diseases as in some developing countries. (3) Protracted transition- the transition process not being clearly resolved as countries exhibit both infectious and chronic diseases. (4) Epidemiologic polarization- a polarization is seen to occur among population subgroups, with the poor and rural populations succumbing to the pre-transitional pathology, the rich and urban populations experiencing post-transitional pathology.

The proposed 'protracted and polarized' model bears close similarity to the transitional variant of Omran's delayed model which has two aspects; i) the evolutionary aspect in which stages are outlined and ii) the differentiation between the paces of mortality and morbidity change. As Frenk *et al.* (1989) have argued, with heterogenous

populations, these two aspects take on more complex courses. The protracted and polarized model proposed can be seen as further qualifications of populations in transition but which cannot be adequately described by Omran's delayed model. Upon studying the epidemiologic transition in Mauritius, Bah (1993b) noted that as Mauritius was largely homogeneous with respect to geographic differentials in health and in access to medical and welfare service, the overlap of epidemiologic eras as observed in Latin America does not occur in Mauritius. Over the period under study, infectious diseases show continuous decline in importance while circulatory diseases showed gain in importance. Similarly, the countertransitions reported in Latin America did not occur in Mauritius. The transition in Mauritius cannot also be described as being protracted. As described above, the era of predominance of infectious diseases is clearly defined as well as that of degenerative diseases. The period over which the transformation took place could be narrowed to within a span of about five years. Lastly, as Mauritius is a small island with no sharp distinctions between urban and rural areas, the epidemiologic polarisation experienced in Latin America is absent in Mauritius.

2.4 Studies on Mortality Change

Mortality change can be of three types; virtually no change, decline of mortality and reversal of mortality decline. It would be very logical to argue that if certain factors cause decline of mortality, the absence of such factors would cause stagnation or reversal of mortality decline. However, this is not always the case. The approaches used by many

countries to initiate mortality change can be broadly grouped into three categories. The **first** is a direct approach of using 'vertical intervention' programmes that are disease-specific; for example, immunization campaigns, spraying of insecticides to reduce malaria incidence etc. This approach is often implemented with the assistance of organizations like the WHO or other inter-governmental organizations. The **second** is an indirect approach, which is based on the assumption that mortality will be reduced indirectly by focusing on social and economic development and social equity. This approach affects factors such as education, income, health care etc. The **third** approach can best be termed as 'restructuring of the society'. This takes the form of increasing egalitarianism and populism, increasing female autonomy, establishment of open political systems and elimination of rigid class structures (Caldwell, 1986).

Part of the differences in the mortality levels achieved by the developing countries which experienced slow mortality decline (as opposed to those which experienced rapid mortality decline) is due to the fact that the former stopped at the first approach (while the latter proceeded to implement the second and third approaches in whole or in parts). According to the WHO (1984:89), the decline in death rates experienced by the 'rapid mortality decline' developing countries is attributable to:

> 'a strong political commitment to improving the health and general welfare of the populations, including an emphasis on comparatively simple preventive measures, health education and the delivery of services to the doorstep of the household in rural areas.'

In explaining the mortality difference between 'rapid decline mortality countries'(RDMC)

and 'slow decline mortality countries' (SDMC), Palloni (1985) argues that the contrast between them can be explained by the difference in economic growth and the unequal success in vertical intervention programs. These two factors however cannot account for the differences in mortality transitions in the two groups of countries. A summary of specific factors influencing mortality decline and those influencing reversal of mortality decline now follows.

2.4.1 Factors influencing mortality decline

Mortality is affected by many factors. However researchers often disagree on which were the most important factors that affected the decline in a specific country. Some of these disagreements have become classic debates, as is shortly reviewed below. Some of the factors that have been argued to influence mortality decline are:

- Developments in technology such as antibiotics and insecticides
- Improved maternal and child health care
- Improvement in nutrition, water and sanitation
- Socio-economic developmental variables such as, education, income and accessibility to health care etc. (Meegama, 1985).

One of the classic debates about the important factors in mortality decline is that between McKeown and Razzel. McKeown *et al.* (1972) argued that the decline in

mortality in the eighteenth century England and Wales was largely attributable to improvements in the environment. During the nineteenth century, the main influences for the decline were a) rising standard of living, b) better hygiene, and c) favourable trends in the relationship between some micro-organisms and the human host. Medical therapy played little role in the reduction of mortality. Most of the decline during the twentieth century is however, associated with decline in infectious diseases.

The arguments of McKeown *et al.* were first challenged by Razzel (1974) who pointed out that the nineteenth century England had experienced decrease in food consumption per head and hence the argument on improvement in diet was a weak one. Instead, it was the improvement in personal hygiene which was responsible for the decline during the first half of the nineteenth century. In a study on infant and child mortality in the United States over the period 1900-1930, Ewbank and Preston (1990) also presented a case for the importance of personal health behaviour on the decline in mortality in infancy and childhood. They argued that the sources of mortality decline cannot be assumed to be independent of household behaviours, hence giving support to Mckweown's argument.

In the twentieth century United States, McKinlay and Mckinlay (1977) argue that general medical measures, both chemotherapeutic and prophylactic, appeared to have contributed little to the overall decline in mortality. The medical interventions were introduced several decades after a marked decline had already set in. Preston (1975) had

pointed out the importance of innovations based on germ theory other than drugs. An argument that drugs were not important in the decline does not rule out other innovations such as improved antiseptic practice, quarantines and segregation of infectious patients and movement for cleaner foods and water, better sanitation and improved infant feeding. In a recent study on child mortality in late nineteenth century America. Preston and Haines (1991) reached the conclusion that McKeown was correct that mortality decline since the middle of the nineteenth century owed little to specific drugs and medicines.

Other researchers have focused on the importance of economic factors on mortality decline. The findings from such studies have been diverse. In one such study, Stolnitz (1955) divided several countries into three categories; he found a close relationship between gross national income per capita (GNI) and age specific mortality rates within each of the three categories but little correlation overall. Using a similar methodology, Hanada (1982) found a strong and positive association between GNI per capita and life expectancy at birth for middle income countries but weak association for higher and lower income countries. In the case of Sri Lanka where the mortality decline after the second world war was very rapid, Fredericksen (1961) argued that rise in standards of living was responsible for virtually all of the post-war mortality decline. In later papers, he extended this conclusion to British Guiana, Mauritius and then to 21 other countries. His arguments regarding Sri Lanka and British Guiana have been refuted by Newman (1970) who argued that malaria control was the main factor for the decline. This debate was picked up again by Gray (1974) who argued that malaria control measures accounted for

only about 23 percent of the mortality decline. Instead, he attributed most of the reduction to 'Improvement in medical services, therapy, nutrition and possibly economic factors...' (Gray, 1974:226). Preston (1975) investigated the relationship between mortality and level of economic development using cross-national data. He found that with increasing levels of GNI per capita the expectation of life at birth initially rose steeply, but became almost a plateau at high levels of income, suggesting a relationship of diminishing returns between the health status of the population and increases in national income levels. He concluded that income had been a trivial factor in recent mortality trends and yet was an important determinant of mortality levels. In the above mentioned work on child mortality in the nineteenth century America, Preston and Haines (1991) concluded that economic development was not the prime force in the mortality decline experienced.

The methodologies employed in many attempts at assigning responsibility for mortality decline have been criticized by several authors. Many use nations as units of analysis and aggregate the factors involved into two major blocks: living standards and medical and public health practices (Ewbank and Preston, 1990). Also, by using cross-sectional approach, drawing from a heterogenous set of countries, the studies overlook the dynamic relationship between mortality and its determinants (Ruzicka and Kane, 1990). As reviewed above, many of the explanations for mortality decline have been shown to be still short. This led Riley (1990) to remark that:

> '...Either we are looking in the wrong direction in exploring nutrition, specific medical measures....,public health, climate, family practice...virulence of pathogenic micro-organisms, the disease or pathogenic profile, and income separately, or that the solution to the puzzle consists of some mixture of these explanations. Perhaps the mix, or rank order of items in the mix, will have changed over time or varied from place to place. Even so, ...a mixture of explanations offers far more promise than the unitary explanations that so many scholars have pursued.'

In sum, while all the factors mentioned above are important, their relative importance differ among countries and from era to era (Grosse and Harkavy, 1980). Further, the experience of one country cannot be easily applied to another country nor can the experience of an era easily be extrapolated into another.

2.4.2 Factors influencing reversal of mortality decline

During the 1980's, research writings started to emerge alluding to deceleration of mortality decline in developing countries (Grosse, 1980; UNECA, 1984; Arriaga, 1981; UNESCAP, 1984; Palloni, 1985). Many reasons have been identified for this deceleration. Arriaga (1981) attributes it to the slow-down in development; Palloni (1985) mainly to distributive programs that were either non-existent or unsuccessful. In general, according to WHO (1984:89):

> 'The reasons for this retardation of mortality decline appear to be associated with slower social and economic development, as well the inadequacies of public health measures and of health care services. The specific causes undoubtedly vary from country to country as does the relative importance of each of the individual factors mentioned above.'

One can probably add to these, the problems of political instability, wars and prolonged famines and droughts.

2.4.3 Routes to low mortality

The challenge faced by many developing countries is how to achieve low mortality in the face of all the constraints that contribute to reversals in mortality decline. One of these constraints, as mentioned above, is low economic development. But the fact that there are some developing countries which have achieved far lower mortality levels than what would be expected at their level of economic development, gives rise to some hope that there could be inexpensive routes to low mortality so that good health could be achieved at low cost.

This interest in achieving good health at low cost led to a conference of health experts in Bellagio, Italy in 1985. The theme of the conference was to explore the factors that have contributed to low mortality in four developing countries in spite of their low level of economic development. These countries were Sri Lanka, China, Costa Rica and Kerala State in India. Assuming these factors can be accounted for, the next issue is whether it is possible to extrapolate the findings to other developing countries. This subsection briefly looks at some of the explanations for these factors and the issue of extrapolation to other poor countries.

Having reviewed the literature on the different factors contributing to low mortality in the four countries, Warren (1985:246) identifies four important elements :

'1. Political and social will.
2. Education for all with emphasis on primary and secondary schooling.
3. Equitable distribution throughout the urban and rural populations of public health measures and primary health care.
4. Assurance of adequate caloric intake for all.'

In the same vein, Rosenfield (1985:175-176) identifies five social and political factors that are shared by all the countries:

'1. Historical commitment to health as a social goal;
2. Social welfare orientation to development;
3. Widespread participation in the political process;
4. Equality of health services coverage for all social groups (equity).
5. Intersectoral linkages for health.'

These are broad generalizations used to describe common features among the four populations. However, for each of those countries there are additional factors which played important roles in health improvement. For example, in three of these populations, China, Sri Lanka and Kerala, there is a well developed indigenous health care system which complements the Western allopathic health care system. In China, other additional factors are: the provision of safe water supplies, sanitary and waste disposal and fertility reduction (Jamison, 1985). In Costa Rica, while Rosero-Bixby (1985) singles out primary health care as the single most important factor in mortality decline in the 1970's, Gonzalez-Vega (1985) argues that no one factor can account for the health improvements. From the point of Gonzalez-Vega, several different but mutually dependent factors have contributed to the decline in mortality. The role of these factors cannot be divorced from their historical context. So the gains in mortality in Costa Rica have resulted from 'a long

gestation period and a sequence of preparatory accomplishments', they should not be viewed as a recent phenomenon (Gonzalez-Vega, 1985). In the case of Sri Lanka, while Gunatilleke (1985) gives importance to the role of the health care system in mortality, he is careful to point out that the health care system was actually a part of a broad social welfare program which included subsidized food and free education.

With regard to the cost aspect of the health efforts in these populations, Joseph (1985) argued that the term 'low cost' is not appropriate as each of the four populations invested huge amount of government expenditure on health. Also, even though mortality is low in the populations, morbidity is still high, so the populations have still not achieved 'good health'. On the question of whether the lessons learnt from these four populations are relevant to other developing countries i.e. whether their examples serve as routes to low mortality, the conference participants agreed that they did and hence made the following recommendations:

> 'A. Equitable distribution and access to public health and health care beginning at the primary level and reinforced by secondary and tertiary systems.
> B. A uniformly accessible educational system emphasizing the primary and then moving to secondary and above.
> C. Assurance of adequate nutrition at all levels of society.'
> (Halstead *et al.*, 1985:248).

Kunitz (1989) disagrees that the experience of these populations could be extrapolated to other developing countries. In his view, these recommendations imply that the third world countries could develop these attributes by choice. These recommendations (which he thinks are standard primary health care and nutrition recommendations) did not

suggest a profound understanding 'of the social and political processes underlying the evolution of different nations.' Kunitz cautions against extrapolations to other cultures without understanding the history and institutions of each culture. This reservations are partly in line with the remark of Gonzalez-Vega mentioned above - that in the case of Sri Lanka, the gains should not be viewed as a recent phenomenom but rather, their historical context has to be taken into account. The conference summary statement did not take historical differences into account.

Perhaps one of the fruitful contributions of the conference was in highlighting the importance of non-economic factors in the reduction of mortality. This opened the way to a new research area known as 'Health Transition' which is concerned about the cultural, social and behavioural determinants of health in preference to biomedical and economic determinants.

2.5 Health Transition Theory

The original formulation of the health transition theory is by Learner (1973). He used the term 'health transition' and employed the evolutionary approach in describing the phases of health changes. The successive stages of health changes are described as: 'low vitality', 'increasing control over mortality', and 'broadened conception of health'.

Recently, the concept of health transition has received great attention. This came

out of research findings which showed that societies with similar levels of health provision and comparable levels of development displayed contrasting levels of mortality and widely different levels of health. The explanations of such anomalies gave rise to the recognition of what is now called 'the health transition factor.' The theory of health transition is still being developed, and one finds in the literature different formulations of the same theory. Health transition is used by Caldwell (1990) to include both the epidemiological transition and the related social changes. It is defined as:

> 'The cultural, social and behavioural determinants of health.' (Caldwell and Caldwell, 1991:3)

These are those determinants other than medical interventions and income. Findlay (1991:382) clarifies further:

> 'of interest in the "health transition" are not the changes in diseases but in the social transformations accompanying these changes.'

These social transformations have been described as follows:

> 'In the early transitional societies, neither the family nor society has developed effective technologies or behaviours to protect itself from disease.....As the transition progresses, communities and families begin to develop a more effective range of measures which protect then from parasitic and infectious diseases. ...In the late transitional settings, local communities add, to their earlier responsibilities for basic environmental sanitation and public health measures to protect the population from infectious and parasitic diseases, responsibilities for reducing the risk of chronic diseases or accidents.' (Findlay, 1991:382-383).

Thus, the aim in health transition research is the focus on other important aspects of health change which have been neglected in favour of biomedical explanations (Van de Walle, 1990).

Although health transition variables are seen as being elusive, they are considered to be of great importance in explaining global mortality decline (Caldwell and Caldwell, 1991). It is even perceived that the most economic route to low mortality is probably to spe' more on seeking complementary behavioural changes rather than increasing direct medical expenditure (Caldwell, 1990). Also, health transition variables help explain the 'role of social and economic changes in initiating and accelerating a societal transition to better health.' (Findlay, 1991:387). Palloni (1990) however cautions that the health transition should not be seen as a unique path that consists of several stages leading from high mortality regimes to low mortality regimes. Instead, the health transition in developing countries is characterized by multiplicity of stages and multiplicity of paths for countries as well as for population subgroups. The other property of the health transition is its 'vulnerability' because of obstacles. This leads to unevenness and slow-downs instead of continuity and acceleration in mortality decline. These obstacles eventually lead to worsening of mortality differentials instead of reduction. Lastly, Palloni pointed out that health transition in developing countries is closely associated with fertility and reproduction.

Drawing on data from developed countries dating back to the 18th. century, Riley

(1990a) concluded that there had been two health transitions which have moved in opposite directions. One consisted of decline in mortality while the other of rise in disease prevalence. This inverse association between mortality and morbidity is seen as a temporary rather than a permanent phenomenon. While this inverse association cannot be denied, it is perhaps more accurate to speak of the health transition going through different stages. Nevertheless, Findlay (1991:387) agrees that 'the social and environmental transformations driving the health transition do not uniformly facilitate both lower mortality and lower morbidity.'

In a later development, Frenk *et al.* (1991) attempted to bring together the diverse works on health and epidemiologic transition to form elements for a coherent health transition theory. They discuss five components of the theory; its concepts, determinants, mechanisms, attributes and consequences. In clarifying the concepts, Frenk *et al.* (1991:23) conceive of health transition as being made up of two components:

> 'The first is the epidemiologic transition strictly speaking, which is defined as the long-term process of change in the health conditions of a society, including changes in the patterns of disease, disability and death. The second component, which may be called the health care transition, refers to the change in the patterns of the organized social response to health conditions.'

In this definition, the epidemiologic transition is treated as being part of a broader health transition. In outlining the determinants of the health transition, a framework is proposed in which the determinants of health status are grouped into three categories: The basic determinants at the systemic level, the structural determinants at the societal level and the

proximate determinants at the institutional and household level. The basic determinants consist of biological risks, the environment and social organization; the structural determinants consist of level of wealth, social stratification, occupational structure and redistribution mechanisms in the society; the proximate determinants consist of working conditions, living conditions, lifestyles and the health care system. In summary,

> 'health status is the final result of the balance between the exposure to disease agents and individual susceptibility resulting from a complex network of risks; this, in turn, is the product of an articulated set of social and biological determinants.' (Frenk *et al.*, 1991:29).

The mechanisms involved in the health transition are three. They are:

> 'fertility decline, which alters the age structure; changes in risk factors, which affect the incidence of diseases; and improvements in health care technology and organization which modify case fatality rates.' (Frenk *et al.*, 1991:31).

In describing the attributes of the health transition, Frenk *et al.* (1991) considered them to be synonymous with those of the epidemiologic transition. Building on previous modifications of the epidemiologic transition theory by Frenk *et al.* (1989), the attributes are described as:

> 'the patterns of change, the sequence of stages, the starting moment, the pace and direction of changes and the distribution of health profiles among different groups.' (Frenk *et al.*, 1991:33).

Lastly, the consequences of the health transition are considered to be manifest in demographic processes such as the change in age structure and population growth rates; in economic processes such as the effect on labour force productivity and retirement

allowance plans and finally in the health care system.

One crucial contribution of the health transition theory is the recognition of the central role played by health care systems toward achieving low mortality. This recognition now gives renewed importance to indigenous health care systems which are not based on the Western model- an area that medical sociologists had been exploring for the past few decades.

2.6 Health Care Planning

Health care system (HCS) refers to the 'formal and informal arrangements that exist in all countries for the primary purpose of treating the ill, caring for the disabled and promoting health.' (Murnaghan, 1981). The planning of the HCS for minimizing costs or for better efficiency or for the achievement of certain objectives and goals is referred to as, 'health care planning.' While health care planning (HCP) is very important, it is a very complex task. This complexity is partly due to its inter-disciplinary nature. It is a field of concern not only for public health workers, health economists and geographers but also for medical sociologists and anthropologists. Indeed one could say that demographers are recent arrivals in the field of health care planning. In studying the health of children, for example, demographers only succeeded in bridging together their socio-economic paradigm with the biomedical paradigm of the epidmiologist after the seminal work of Mosley and Chen in 1984. With such breakthroughs, it makes it easier for demographers

to borrow from other disciplines when addressing health care planning. The demographer's contribution to health care planning is quite meaningful as he or she adds to it the wealth of work done on indicators of morbidity, causes of death, and mortality and also the skills of indirect estimation whenever relevant information are not available.

For HCP to be complete, it has to take into account the needs, wants and demands for health care; the utilization of health care in meeting demands; the resources (material and manpower) available and how they are allocated and also health care delivery.

Any health care programme typically 'starts with an assessment of population needs and demands for health care. This forms the basis of the formulation of objectives for the programme which will be adapted and modified according to the availability of resources. On the basis of the objectives a series of activities (the process) are implemented whose aims are to achieve an outcome which should match the stated objectives.' (Holland, 1983).

In reality, however, health care planning in developing countries involves much more than 'stating objectives' and implementing activities to realise certain outcomes. Health care cannot be planned in isolation from the socio-economic and political systems existing in the country. In societies where the prevailing ideology emphasizes hierarchical relationships or individual responsibility, entrepreneurship or egalitarian social relationship, the health care systems that emerge are different (United Nations, 1984).

Another reality is that in developing countries, one is confronted by two (often conflicting) paradigms for health care: the biomedical paradigm on which the Western health care system is based and the psychosocial paradigm on which many other non-Western medical systems are based (Fabrega, 1977). In spite of these constraints, the challenges facing health care planing is to recognize the wide range of health problems and take into account other problems stemming from changing morbidity and mortality patterns (Okolski, 1986).

2.7 Objectives of the Present Study

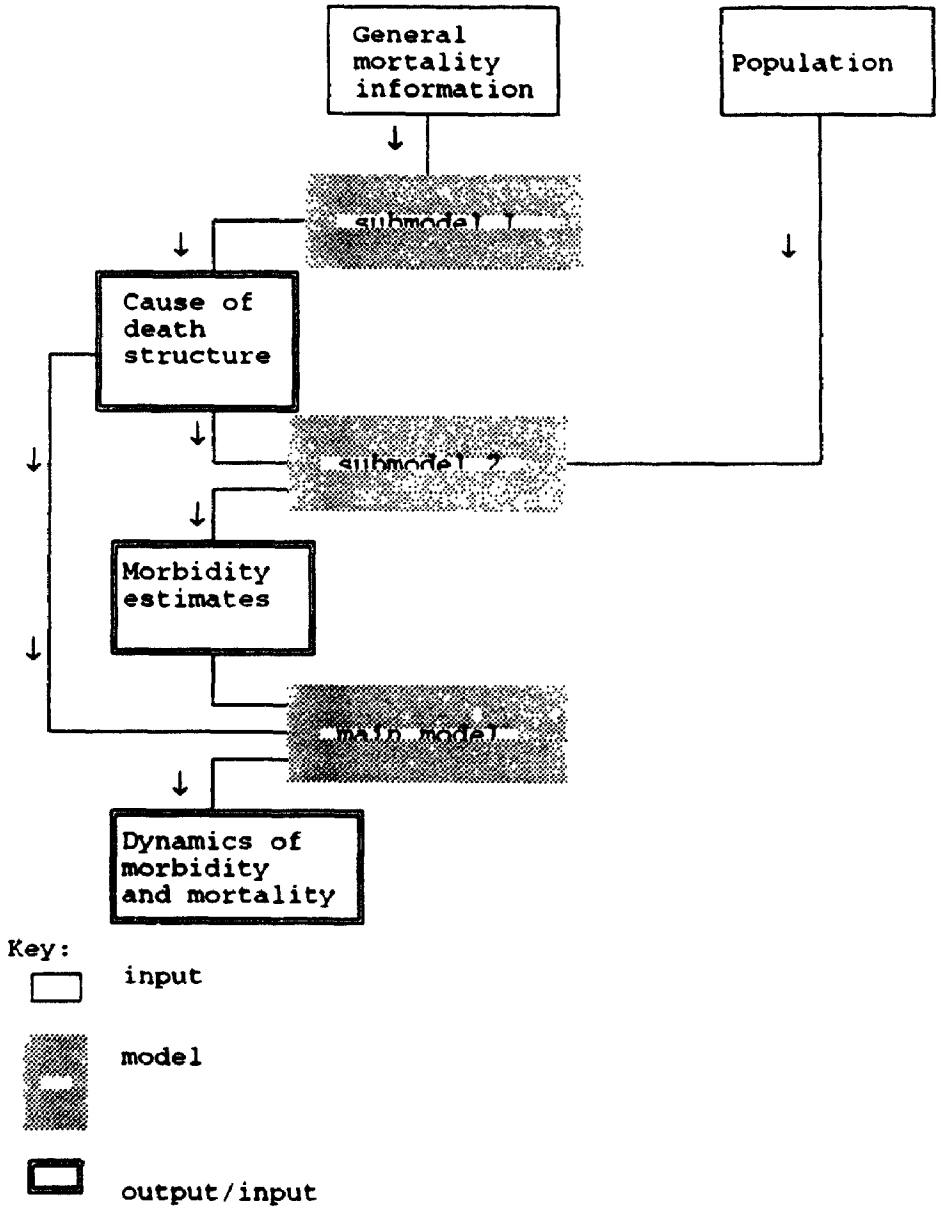
Considering the nature of the study, one part of the thesis is largely methodological while the other is empirical. Both parts of the thesis go towards satisfying the following objectives:

1) To obtain cause of death data from mortality data by applying existing models or modified variants of them.
2) To obtain morbidity data from mortality data by applying existing models or modified variants of them.
3) To investigate the dynamics of morbidity and mortality using a multistate demographic model .
4) To investigate the implications of the dynamics of morbidity and mortality on health care planning.

The analytical framework to be employed in order to achieve the objectives of this study is graphically shown in Figure 2.1. The framework takes into account the data restraints in African countries. Before modelling the dynamics of morbidity and mortality, two submodels are constructed in order to derive the cause of death structure and morbidity rates. Submodel 1 is used to estimate cause of death structure and submodel 2 is used to estimate morbidity rates. Using these data in addition to population data, a multistate model (main model) is used to explore the dynamics of morbidity, cause of death and mortality. It is hoped that the results of the main model might be interpreted in light of health care planning in Africa.

The work might finally conclude by relating the results to the substantive theories of the epidemiologic transition and the health transition. Policy implications can subsequently be extracted from these models.

Figure 2.1 Framework for Studying the Dynamics of Morbidity and Mortality.



CHAPTER III

MORTALITY AND CAUSES OF DEATH IN AFRICA AND THEIR INDIRECT ESTIMATION

3.1 Levels, Patterns and Trends of Mortality in Africa

It is a common lamentation that very little is known about the 'trends and variations in African mortality' (United Nations, 1982). This is largely due to poor vital registration systems, differential coverage of censuses and the reporting errors inherent in both the systems in many African countries. The degree of incompleteness and inaccuracy vary from country to country; being low in Egypt and Island populations like Mauritius and Cape Verde, and very high in certain countries of East and West Africa.

As a result of the shortcoming of these demographic sources of data, most of the information on mortality comes from 'indirect' estimation techniques. The term 'indirect' has been used to describe

> 'any estimation method that depends upon models or uses consistency checks, or indeed uses conventional data in an unconventional way.'(United Nations, 1983:2)

Using these indirect estimation techniques, a great deal has been learnt about infant and child mortality while very little is known about adult mortality. Timæus (1991) cites two reasons for this discrepancy. First, improvements in child mortality are widely seen as a more important public health issue. Second, development of robust indirect estimation

techniques for measuring adult mortality has been proved difficult.

Some of the recent findings about the levels, patterns and trends of childhood and adult mortality in Africa are summarized below.

3.1.1 Infant and child mortality

Given only levels of infant and child mortality observed at arbitrarily different times, the most that could be made of them is regional comparisons or comparison with standard reference model life tables such as those of Coale and Demeny. Upon comparing the age pattern of infant and child mortality in sub-Saharan Africa with reference life tables, the United Nations (1982) found those of Coale-Demeny model life tables inadequate in describing the observed patterns of mortality. Pison and Langaney (1985) arrived at a similar conclusion after studying childhood mortality in Bandafassi in Eastern Senegal. With regard to regional comparisons, it has been observed, as far back as 1974, that within Africa, considerable variations in mortality are found within the same geographic region (Cantrelle, 1974). This has been affirmed again by Blacker *et al.* (1985) and Cantrelle *et al.* (1986). Further, for the same population at different points in time, Blacker *et al.* (1985) note that there appeared 'to be no rule to predict whether infant or childhood mortality falls most rapidly.'

In a comprehensive review of trends in childhood mortality in Sub-Saharan mainland Africa, Hill (1987) has made several observations. There had been a major post-war decline in mortality but the magnitudes of the decline varied between countries. While it was dramatic in countries such as Ghana, Kenya and Congo, it was gradual for the rest of the countries. For all periods for which data existed, the variations in mortality level were very impressive. In the late 1950's, the proportion of children dying before age five ranged from 16 to over 40 percent. By the late 1970's, the range was from 12 to 33 percent, with most countries falling below 27 percent. It was also observed that there was a marked and consistent differential between Eastern and Western Africa. According to Hill, the highest levels of childhood mortality existed in West Africa, next highest in Central Africa. The levels in East Africa were low and the lowest were in Southern Africa. This is an echo of previous findings by Adegbola (1977) and Blacker (1979).

Hill classified the African countries into three groups. In the first group are those countries which experienced 'static or rising mortality' namely, Angola, Niger, Nigeria, Mozambique, Ethiopia, Rwanda and Sudan due to socio-political instability and interruptions in socio-economic development. In the second group are those from Western and Central Africa which have achieved levels comparable to those in Eastern Africa due to rapid economic and educational development and heavy urbanization. These are Ghana, Congo and Cameroon. The last group has only one country namely Malawi. Its mortality is considered to be anomalously high compared to the lower levels in the other countries of Eastern and Southern Africa.

Hill's paper did not challenge the widely accepted view of regional differentials in mortality favouring South and Eastern Africa, leaving Western and Central Africa at disadvantage. However, the many exceptions given by Hill make this view questionable. This point has been taken up by Ayeni (1985), who argues that, as survey methodology and analytical techniques have improved since the late 1960's, the early estimates for infant and child mortality in Africa may not be reliable. Using the WFS data to support his argument, he has derived infant and child mortality rates for eight countries in the late 1970's. Two of these countries are taken from Northern Africa, three from West Africa and one each from Southern, Central and Eastern Africa. His results run counter to the accepted view. His observations were as follows:

> 'According to the PRB (Population Reference Bureau) estimates Egypt has the lowest infant mortality in North Africa and the second lowest in the whole of mainland Africa but estimates from the WFS data derived here show that it has the highest rate of the eight countries considered. Similarly, previous estimates had shown that Kenya had the lowest infant mortality rate in mainland East Africa. Estimates from the WFS data now show the Ghana's rate is lower then Kenya's and that Nigeria's rate is only slightly higher. Also Lesotho which has the second lowest rate in Southern Africa is now shown by the WFS data as having about the same level with Nigeria and Sudan. The faint impression from the estimates derived from the WFS data in the eight countries is that West Africa may indeed have lower infant mortality rates than other subregions. In any case the previously held belief that it has substantially higher levels of mortality has not been confirmed.' (Ayeni, 1985:268)

Ayeni's observation has been partly confirmed by Hill and Hill (1988). They argue that while there was East-West differential in mortality in the 1960's, it had disappeared by the 1980's as a result of faster mortality declines in Western and Middle Africa than in the South.

The most recent evidence from the Demographic and Health Surveys (DHS) conducted in the late 1980's also confirm this later position. Out of nine surveys conducted in West, East and South Africa, it was found that Uganda in the East had the highest infant mortality rate at 95.8 per thousand, much higher than the rates in Ghana, Togo, Senegal and Mali in West Africa. The infant mortality rates of Ghana and Togo were close but slightly lower than that of Burundi in the East. Zimbabwe had the lowest infant and child mortality rates and the rates in Kenya were lower than those in Ghana, Togo, Senegal and Mali (Barbieri, 1991).

The observation about the heterogenous nature of mortality in Africa was also confirmed. Within-country differences in mortality rates were found to be at least as large as between-country differences. For Kenya, the DHS data showed that the ratio between the lowest and the highest sub-regional infant mortality rate was close to 1:4 and was nearly 1:10 for child mortality rate (Barbieri, 1991).

3.1.2 Adult mortality

Generally, very little has been done on indirect estimation of adult mortality until lately. This is mostly due to neglect, as work on indirect estimation of adult mortality started as early as the 1970's. Much work could not proceed because of the added problems associated with the estimation of adult mortality. However, from the little work done on adult mortality, a picture emerges which helps us to draw inference on the levels,

patterns and trends of mortality.

Detailed information about age patterns of mortality are available only for few countries in Africa. Some of these include Senegal and Gambia in West Africa, Kenya in East Africa, Algeria, Tunisia and Egypt in North Africa and the Island of Mauritius in South-East Africa. Upon comparing the age patterns of mortality of North Africa with those of Coale-Demeny, the United Nations (1982) observed that the 'South' model fits the North African data better. For sub-Saharan Africa, the mortality patterns were found to vary so widely that it is impossible to a get a single pattern of mortality for sub-Saharan Africa.

After examining recent patterns of mortality in Africa, Blacker et al. (1985) came to several conclusions which have methodological implications: First, no simple association exists between the age-specific mortality schedules of different populations and the overall level of mortality or with their location in Africa. Second, no family of model life tables, either from the Coale-Demeny set or the New United Nations model life table set, has been found to have all of the mortality features common in Africa or even few of them.

In a multiround survey of Bandafassi in Eastern Senegal, the life expectancy over the period 1970-1983 was found to be as low as 31.4 years for both genders. In this case, however, the pattern of adult mortality observed was found to be adequately represented

by Coale-Demeny West model as well as Brass' model life table (Pison and Langaney, 1985).

Timæus (1988) accepts the conclusions of Hill (1987) on child mortality as also holding true for adults to some extent. In the late 1970's the life expectancy for both males and females at age 15 ranges from 44 years in Gambia to nearly 56 years in Northern Sudan. Even within Kenya, life expectancy at age 15 range from around 45 years to 57 years in different regions. These findings further emphasize the heterogenous nature of adult mortality in Africa.

In offering an explanation for the large regional differentials in Africa, many suggestions have been made. These include, 'climate, altitude, ecology, epidemiology, basic diets and patterns of population settlement, as well as various cultural, social, political and historical factors.'(Hill, 1987). Commenting on the slow rate of mortality decline in sub-Saharan Africa, United Nations (1982:96) has this to say:

> 'Among the many conditions that have prevented sub-Saharan African population from emerging into the moderate-mortality zone, perhaps the most important is impoverishment. A distinct lag in food production and economic growth, coupled with an increased emphasis on governmental spending in military areas, have certainly taken their toll in health progress.'

3.2 Trends of Causes of Death in Africa

According to the United Nations (1982), very little is known about the actual structure of causes of death in sub-Saharan Africa. Fortunately, the available examples based on good data come from different parts of Africa so that taken together, they can give a fair picture of cause of death structure in Africa.

In a recent study of mortality in a rural area of Machakos district in Kenya, the cause of death structure over the period, 1975-78 was reported as follows: Disease of the respiratory system (15.8%), congenital anomalies and perinatal conditions (12.8%), intestinal infectious diseases (10.8%), measles (8.1%), disease of the circulatory system (7.2%), tuberculosis (7.1%), other infectious and parasitic diseases (6.6%), external causes of injury and poisoning (6.2%), nutritional and metabolic diseases (4.4%), neoplasms (3.7%), disease of the digestive system (3.5%), malaria (3.1%), all other (4.0%) and unknown (6.6%) (Omondi-Odhiambo and Ginneken, 1990). The reason given for the few deaths due to malaria was the high altitude of Machakos and the abundance and ready availability of anti-malaria drugs. In an earlier study of mortality in Kenya, Ewbank *et al.* (1986) had also estimated the major causes of death around 1979. The two sets of estimates are slightly different but agree that respiratory causes, measles and intestinal diseases are among the four leading causes of death.

From the Machakos study, Omondi-Odhiambo and Ginneken (1990) identified pneumonia, influenza and intestinal disease as the major killer diseases for infants below one year. For ages 1-4 years of age, measles and nutritional deficiencies were found to be the major causes of death. For ages 5 years and above, the causes of death that were identified as being important were external causes of injury and poisoning, other infectious and parasitic diseases, diseases of the circulatory system and tuberculosis.

In a study of mortality in two Senegalese cities over the period 1960-1980, Cantrelle *et al.* (1986) found the leading causes of death as follows:

Infants: congenital disorders, malnutrition and dehydration, measles and diarrhoea.

Ages 1-4: measles, malnutrition and dehydration, diarrhoea, malaria and broncho-pulmonary disorders.

Ages 5-14: malaria, tetanus, diarrhoea, measles and broncho-pulmonary disorders.

It was observed that the major incidence of infant mortality was tied up with climatic factors such as the amount of rainfall, and the incidence of drought and also their consequences on the availability of food.

In a study of mortality in the West African city of Bamako in Mali, the principal causes of death over the period 1974-1985 were found to be: measles (11.9%), malaria (9.3%), diarrhoea (6.6%), prematurity (5.9%), cardiac problems (3.2%), malnutrition (3.2%) and other causes (32.8%) (Bonneuil and Fargues, 1990).

Vital statistics on causes of death from hospital data in East Africa showed that the two leading causes of death in both Kenya and Malawi in 1978 were infectious and parasitic diseases followed by diseases of the respiratory system. For Zambia in 1974, the leading causes of hospital deaths were infectious and parasitic diseases followed by 'certain conditions originating in the perinatal period.' For Gambia in 1981, the leading causes of hospital deaths were infectious and parasitic diseases followed by diseases of the digestive system. For Ghana in 1976 and Liberia in 1980, infectious and parasitic diseases are followed by 'ill defined causes' and the leading causes of hospital deaths (United Nations Economic Commission for Africa (UNECA), 1985).

A category of causes of death which needs special attention is maternal mortality. The expression 'maternal mortality' is generally used to cover both direct and indirect obstetric causes of death. Direct obstetric causes of death include haemorrhage, infection, eclampsia, abortion and obstructed labour. Indirect obstetric causes of death include diseases aggravated by pregnancy, such as viral hepatitis, malaria, anaemia and cardiovascular diseases (Lettenmaier *et al.*, 1988). From the few population-based studies available in Africa, maternal mortality ratios have been found to be 1025 per 100,000 live births in rural Gambia over the period 1951-1975 (Billewicz and McGregor, 1981) and 1286 per 100,000 live births in rural Senegal over the period (1975-87) (Boerma, 1987). Low maternal mortality levels of less than 100 per 100,000 live births have been reported in rural Kenya over the period 1975-1978 (Voorhoeve *et al.*, 1985) and intermediate levels of around 500 per 100,000 live births have been reported in Ethiopia (Kwast *et al.*,

1986) and in Guinea-Bissau, 1982-84 (Boerma, 1987).

3.3 Methodological Implications

The implications of the above findings on mortality levels, age patterns of mortality and causes of death structure for developing the necessary methodologies of indirect estimation are as follows: The first and most important feature of African mortality patterns is the high unstability. Mortality levels found to vary markedly between countries and between regions in the same country. The second is the high level of infant and child mortality relative to adult mortality. Third is the inability of one-parameter-Coale-Demeny life tables, to adequately describe most African mortality patterns. There is need for two or more parameter models to adequately reflect African mortality profiles, or better still, a relational model. Ewbank (1990) applied the logit model of Brass (1975) to data based on indirect estimation of child and adult mortality to derive new information about the age patterns of mortality in East Africa. He found that the age pattern of child and adult mortality in East Africa can be described on the average by transforming the North Model 12 with a value of ß (slope parameter from the logit model) of about .9 or .95 for both sexes combined. He also found that the sex differentials in adult mortality in Africa were larger than what was implied in the Coale-Demeny and UN life tables. Hence he recommended the use of relational model life tables, of at least two parameters so that these parameters could be varied to fit the estimates of the child and adult mortality.

Starting with two-parameter relational life table model of Brass (1975), several other relational models have been proposed (e.g Zaba (1979), Gomez de Leon (1980)). The latest in relational model life tables is the four parameter adaptive model of Ewbank *et al.* (1983). The model is collapsible into one, two or three parameters depending on the available data. The model also comes with a new standard life table which seems to perform better than Brass's general standard life table. Ewbank *et al.* (1983) showed that with the two additional parameters, the adaptive model could describe changes in very young and old ages far better than Brass's model.

The pattern of cause of death in most African countries, with the exception of some island populations like Mauritius where mortality has approached those of developed countries, can be described as belonging to the 'contemporary model' of Omran. They are also mostly in the second stage in their epidemiologic transition, namely the stage of 'receding pandemic' where life expectancy at birth increases steadily from 30 to 55 years. The major causes of death at this stage are:

- Intestinal diseases
- Other infectious diseases
- Airborne diseases
- Maternal causes of death
- Accidents and poisonings
- Behavioural diseases
- Complex and unexplained causes

While these broad groups of causes of death feature in many African countries, there are many disparities between their cause of death structures. These disparities arise due to differences in environment, culture, climate, education, income levels, population distribution, availability and accessibility of health services, the intensity in public health campaigns and a host of other factors. This means that in the estimation of cause of death structure, a very broad regionalization will not be helpful. As much as possible the unit of analysis should be national or sub-national and effort should be directed to including some of the sources of variation in cause of death structures observed in Africa.

3.4 Indirect Estimation of Causes of Death

A good assessment of the cause of death structure is the first step in any planning on mortality reduction. As Lopez and Hull (1983:66) remark:

> 'strategies for reducing mortality should be based on an informed assessment of the underlying structure of causes of death.'

Hakulinen *et al.* (1986) consider statistics on causes of death to be an 'indispensable' part of health information systems on which managerial decisions are based. They summarize the importance and use of cause of death data as follows:

> '(a) contribution towards explaining trends and differentials in overall mortality; (b) relevance for deciding on priorities for health action as well as in bio-medical health systems and behavioral research; (c) role as baseline data for the design of intervention programmes; (d) assessment and monitoring of public health problems; (e) allocation and distribution of resources to and within the health sector; (f) use as an indisputable measure of the effectiveness of health programmes, and (g) clues for epidemiological research.' (Hakulinen *et al.*, 1986:177).

It is hard to obtain mortality data in Africa and even much harder to obtain cause of death data. Several attempts have therefore been made to estimate them through indirect techniques. Some of these techniques are reviewed below.

3.4.1 Preston's regression methodology

Preston and Nelson (1974) made the first comprehensive attempt at indirect estimation of cause of death structure. Their approach was empirical, based on cause of death data compiled in a previous work (Preston *et al.*, 1972). The data comprised of 165 national populations (mostly European or 'overseas European') spanning a period of about 100 years (1861 to 1964) and grouped into 12 broad categories of causes of death. Using the age distribution of the 'West' model female stable population with life expectancy, e^0_0 = 65.0 and growth rate, r=.01 as the standard population (effectively, an old-age standard), Preston and Nelson computed age-standardized cause-specific death rates for each of the 12 broad groups of causes as well as for all causes combined.

Using these standardized death rates, Preston and Nelson estimated for each of the 12 groups of causes of death, linear regression equations of the form

$$M^i = a^i + b^i . M \qquad (1)$$

Where M^i is the age-standardized cause-specific death rate for cause i, M is age-standardized death rate for all causes and a^i and b^i are the fitted regression coefficients (given in Appendix I).

Using the same data, Preston and Nelson also fitted a series of second-degree polynomials of the form;

$$M^i = a^i + b^i . M + c^i . M^2 \qquad (2)$$

where a^i, b^i and c^i are the fitted regression coefficients.

However, after experimenting with different non-linear curve fitting methods, they observed that there was not much gain in explanatory power over the linear regression model.

These two regression equations formed the basis for further indirect estimation of cause of death structures. Using the values of the coefficients as inputs, the aggregate cause-specific death rates were estimated with the knowledge of the overall mortality rate.

Preston (1976) extended the regression methodology to include age patterns of mortality. Representing age patterns of mortality by life table survivorship proportions, he related them to overall level of mortality by the relation

$$\log(l_{x+n}/l_x) = a_x + b_x . M \qquad (3)$$

where (l_{x+n}/l_x) is the survivorship proportion, M is the age-standardized death rate, a_x and b_x are the regression coefficients (given in Appendix II). This loglinear relationship was also extended to causes of death using the following equation:

the coefficients of a^*_x and b^i_x are given in Appendix III.

$$\log(l_{x+n}/l_x) = a^*_x + \sum_i b^i_x . M^i$$
$$\text{where } a^*_x = \sum_i a^i_x \qquad (4)$$

One of the early applications of Preston's linear regression model was done by Hull and Rhode (1978) on Javanese data. In their work, they used the linear regression coefficients obtained by Preston (1976) and showed that the cause of death structure can be estimated with some confidence using the regression methodology. It was later argued by Lopez and Hull (1983) that for high mortality populations with 'young' age structures, the regression coefficients derived using the 'old' standard population might not be applicable. They proceeded to derive new sets of regression coefficients using the age-standardised death rates of Preston *et al.* (1972) (these rates were obtained using a younger age population, the West female stable population with e^0_0 =45.0 and r=.02, as the standard). The coefficients are given in Appendix IV. They applied the coefficients to Javanese data and showed that the cause structure derived from mortality rates based on the young standard was much more consistent with the expected cause of death structure of high infectious and parasitic diseases. They further refined the model by deriving separate coefficients for age 0-1 and ages 1-4. These new coefficients are given in Appendix V.

In studying the changes in the structure of causes of death in Latin America over the period 1950-1975, Palloni and Wyrick (1981) also employed Preston's regression methodology. Using data for both genders, they estimated the regression coefficients from the Latin American data instead of using Preston and Nelson's regression coefficients.

regression model of Hakulinen *et al.* the model specifications are unavailable. Beside that, Hakulinen *et al.* used very broad regionalization which is not much helpful in Africa. The model of Blum and Fargues (as admitted by Blum in personal communication) needs further clarification before it could be replicated.

In the light of these shortcomings, the method adopted here for estimating the cause of death structure in Africa is a mixture of Preston and Nelson's regression methodology and Brass's logit system. The proposed methodology runs as follows:

1) Given the crude death rate, the regression coefficients of Lopez and Hull given in Appendices IV and V are used to obtain the cause specific death rates for all ages combined and for age groups 0-1 and 0-4 years for males and females separately and for both genders together. The percentage of the rates for each broad cause of death to the total rates for all causes combined are computed. Similarly, the age- cause-specific death rates are expressed as percentage of the total death rates in these age groups.

2) Using the cause-specific death rates obtained above, the multiple regression coefficients of Preston (1972, 1976) relating the log of life table survivorship to cause-specific death rates are used to obtain life table l_x values for all causes combined for selected ages.

3) From the life table l_x values the next task is to obtain cause-specific death rates by indirect means. This was done by means of Brass logit model and regression of the cause-

for broad age groups) were then estimated using equations similar to equation (1). Through this approach, Hakulinen *et al.* (1986) estimated global patterns of causes of death around 1980.

3.4.2 Lundy's factor analytic methodology

Using the same data of Preston *et al.* (1972), Lundy (1978) used factor analysis to examine the relationship between age-patterns of mortality and cause of death structure. Lundy justified this approach by pointing out the problem of multicollinearity inherent in regression models. He considered the special case of incomplete data. He suggested the following method:

> '(If) a researcher is presented with a set of data covering the population and total deaths, and wishes to estimate the cause structure. He may compute the factor values for the total age pattern, and use them in turn to estimate the factor values for the individual cause groups. From these, in turn, can be generated estimates of the age-specific death rates for each cause group.' Lundy (1978:85).

Using this factor analytic model, he obtained age-standardized cause-specific death rates M'. He also obtained M' using Preston and Nelson's regression model (equation 1). Lundy then compared these two set of estimates with the observed ones. He found that the predictability increased considerably using the factor model. This is mainly because the factor model includes information of age while this is absent in the regression model of equation 1.

3.4.3 Brass' one parameter life table methodology

Brass' (1978) model for estimating cause of death used a standard life table approach but without a rigorous formulation and testing procedure. Brass proposed that mortality from a specific cause should be divided into two categories: 'resistant' and 'avoidable.' In the 'resistant' category it is assumed that the incidence remains constant and in the 'avoidable' category it is assumed that the incidence varies in the same proportion at each age.

Brass' methodology still remains to be refined. At present it leaves many questions unanswered especially with regard to countries with poor data on cause of death. How can one categorize deaths from a specific cause into avoidable and resistant ? Also, when one has only crude death rates, how can the model assist one to get a rough estimate of cause of death structure? Brass' methodology still needs to be worked out in order to answer some of these questions.

3.4.4 Parametric approaches

While the parameterization of mortality has a long history, that of cause of death had a late start and only really got serious attention after Heligman and Pollard's (1980) parameterization of the general age pattern of mortality. The model consists of eight

parameters (A to H) and takes account of infant and child mortality, middle age mortality and old age mortality. According the model, the probability of dying at age x is given by the expression

$$q(x) = A^{(x+B)^C} + \exp[-E(\ln X - \ln F)^2] + \frac{GH^x}{(1 + GH^x)} \qquad (5)$$

Another parameterization of the general mortality profile is the double-exponential model of Rogers and Planck (1983). The model has nine parameters (A_0, A_1 A_2, A_3, α_1,α_2,α_3,μ_1 and μ_2) and is given by the expression:

$$q(x) = A_0 + A_1 e^{-\alpha_1 x} + A_2 e^{-\alpha_2(x-\mu_2) - e^{-\alpha_2(x-\mu_2)}} + A_3 e^{\alpha_3 x} \qquad (6)$$

Using these models, it has been shown that they can be fitted well on certain causes of death (McNown and Rogers, 1990; Rogers and Gard,1991).

3.4.5 Sisterhood method for estimating maternal mortality

The Sisterhood method proposed by Graham *et al.* (1989) was specifically developed for the estimation of maternal mortality. It is unlike the orphanhood and widowhood methods which were developed to estimate adult mortality. The method is built upon the sibling survivorship technique proposed by Hill and Trussel (1977) and is also based on census or survey responses. It derives indicators of maternal mortality from the reported proportions of sisters who reached the age of exposure to the risk of pregnancy-related death, and who are either alive or dead during pregnancy (Graham *et*

al. 1989).

In the sisterhood method, maternal mortality is assumed to follow a relational Gompertz model and together with a fixed distribution of the difference between the ages of the respondent and his/her sister, adjustment factors Ai are derived. These factors are used to convert the proportion of adult sisters dying of maternal causes to probabilities of dying. The method has however had only a limited testing.

3.4.6 The methods of Blum and Fargues for estimating maternal mortality

Blum and Fargues (1990) proposed two methods for estimating maternal mortality:

a) 'Sex ratio' of mortality method

This method assumes that any excess mortality of females over males over the reproductive ages 15-49 is due to maternal mortality. The sex-ratio of mortality i(x) is given by:

$$i(x) = m_f(x) / m_m(x) \qquad (7)$$

Where $m_f(x)$ and $m_m(x)$ are the age-specific mortality rates for females and males respectively, obtained from civil registration sources that do not specify cause of death. The age-specific maternal rate $t_1(x)$ is given as:

$$t_1(x) = m_f(x) - i^*(x)\, m_m(x) \qquad (8)$$

where $i^*(x)$ is the expected value of the ratio of female to male mortality in the absence

of maternal mortality. It is obtained by interpolating between ages just before (10-14 years) and just after childbearing period (50-54) (Blum and Fargues, 1990).

b) Method based on slope of women's mortality curves

This method is based on the application of Gompertz law to mortality rates (Blum and Fargues, 1990). The Gompertz law applies well for adult and older ages beginning at age 30 and particularly for men. For women, however, the Gompertz curve shows a discontinuity at the end of the childbearing period (around age 45). It is assumed that the discontinuity is caused by maternal mortality. Two methods are proposed in order to adjust for this discontinuity. The first method uses backward extrapolation of mortality rates from age 45 and a series of interpolation between the extrapolated and observed rates. The second method also uses interpolation but assumes that the logarithm of maternal mortality rate (in the absence of maternal mortality) is a quadratic function of age.

On application to data from developing countries, the method produced plausible estimates. The sources of bias are abnormal age and sex mortality differentials and high non-pregnancy-related female mortality outside the childbearing ages. There is also a source of bias where the quadratic function proposed fails to hold.

3.5 The Method Adopted in the Present Study

The parametric methods of Heligman and Pollard and that of Rogers and Planck cannot be used at present for indirect estimation of cause of death structure. Before it can be done, the parameters have to be estimated using a long series of cause of death data such as those of Preston *et al.* (1972) and the values of the parameters can then be related to different levels of mortality. One can envisage that once the parameter values have been obtained for different causes at different levels of mortality, then the whole age profile of the cause of death can be estimated given the level of mortality. This assumes that the mortality pattern in the study population is similar to that in the reference population. This is a heavy task; a parallel work that has been done in this direction, though in another field, is the work of Rogers and Castro (1982). The work was based on a model migration equation of eleven parameters. They proceeded to fit the model on a set of migration data and obtained estimates of the various parameters. They established relation between several of the parameters using regression. From these regression equations and other mathematical relation between the parameters, they sought to estimate migration rates using a few input equations. Upon applying the indirect estimation technique to the data for Filipino males, they observed that the result was not very satisfactory.

Brass's method has not been fully developed yet. Lundy's factor model would have been very useful if the model specifications are available. Similarly for the

regression model of Hakulinen *et al.* the model specifications are unavailable. Beside that, Hakulinen *et al.* used very broad regionalization which is not much helpful in Africa. The model of Blum and Fargues (as admitted by Blum in personal communication) needs further clarification before it could be replicated.

In the light of these shortcomings, the method adopted here for estimating the cause of death structure in Africa is a mixture of Preston and Nelson's regression methodology and Brass's logit system. The proposed methodology runs as follows:

1) Given the crude death rate, the regression coefficients of Lopez and Hull given in Appendices IV and V are used to obtain the cause specific death rates for all ages combined and for age groups 0-1 and 0-4 years for males and females separately and for both genders together. The percentage of the rates for each broad cause of death to the total rates for all causes combined are computed. Similarly, the age- cause-specific death rates are expressed as percentage of the total death rates in these age groups.

2) Using the cause-specific death rates obtained above, the multiple regression coefficients of Preston (1972, 1976) relating the log of life table survivorship to cause-specific death rates are used to obtain life table l_x values for all causes combined for selected ages.

3) From the life table l_x values the next task is to obtain cause-specific death rates by indirect means. This was done by means of Brass logit model and regression of the cause-

specific α and ß (α_i and β_i) on α and ß values for general mortality. The idea of applying Brass logit model to causes-of-death has been briefly discussed by Chackiel (1990). The parameters obtained through such an application would be cause-specific parameters. Hence if standard mortality rates are available for both general mortality and for cause specific mortality, then for populations with data for general mortality as well as the specific causes of death, it would be possible to fit both the general Brass logit model as well as its cause specific version. For populations with only general mortality data and not cause of death ones, a way has to be found to obtain cause specific α and ß values (α_i and β_i) from the general α and ß values.

The method used to link these α and ß values is to use data from a developing country to fit Brass logit model on three sets of causes of death- all causes combined, an infectious disease and 'all other causes'. Supposing that the pairs of parameters obtained are given respectively as: α and ß; α_1 and β_1 and finally α_2 and β_2. These parameters could be related by the following multiple regression equations:

$$\beta_1 = a_1 + c_{11}*\beta + c_{21}*\alpha \qquad (9)$$

$$\alpha_1 = a_2 + c_{12}*\beta + c_{22}*\alpha \qquad (10)$$

$$\beta_2 = a_3 + c_{13}*\beta + c_{23}*\alpha \qquad (11)$$

$$\alpha_2 = a_4 + c_{14}*\beta + c_{24}*\alpha \qquad (12)$$

With these regression equations, it would then be possible to obtain cause specific α and β values given α and β values for all causes. With these cause specific α and β values together with the standard rates, age-cause-specific rates can then be obtained. While this empirical approach has some weakness, it is one of the most logical ways to proceed to obtain cause specific death rates when one has only general mortality information in parameterized form.

To accomplish the tasks outlined above, three FORTRAN programs were used, CAUSE.FOR, BRASSCH.FOR and SURVCH.FOR. The source listings of these programs are given in Appendix VIII. For the program CAUSE.FOR, the input data used are the set of crude death rates, the infant mortality rates (age 0-1) and the child mortality rates (ages 1-4) for about 40 African countries for two periods, 1965 and 1983. The mortality levels in 1965 covered a wide range; from the low life expectancy at birth of 37 years experienced in Guinea in West Africa to a high life expectancy of 61 experienced in Botswana in the South.

As a first approximation, the three rates are assumed to be the same for both males and females. These were then applied to gender specific models. The results obtained include: the cause- specific death rates for all ages combined and for age groups 0-1 and 0-4 years for both males and females and for both genders, the percentage of the rates for each broad cause of death to the total rates for all causes combined, the life table l_x values and Brass' parameters- α and β. In addition to Brass's parameters, a parameter for

rectangularization similar to the one proposed by Anson (1991) is also used. These are done separately for each gender. One must note that in the interpretation of the results, not much attention will be given to gender differentials in mortality and causes of death. The two sets of results however will be useful in showing the range within which the true mortality pattern and cause of death structure could lie.

The program BRASSCH.FOR was used to obtain general and cause specific α and β values for a developing country with available cause of death data. Multiple regression models were then fitted to the series of α and β values. The coefficients obtained together with the technical details, are given in Appendix VI.

Finally, using the regression coefficients and standard rates for general mortality as well as cause specific mortality, the program SURVCH.FOR was used to obtain the age-cause specific death rates. Since Carrier and Goh (1972) have shown that Brass logit model performs best when fitted to l_x values rather than to q_x, all the fitting was done via l_x values.

3.6 Discussion of Results

From the output of the program CAUSE.FOR, results were obtained for about 40 African countries that give estimates of levels of causes of death, distribution of causes

of death in age groups 0-1 and 1-4 years and mortality parameters that take the estimated cause of death information into account. Even though estimates were obtained for these countries, the discussion of results will initially focus on the two extremes of Guinea and Botswana. First, the cause of death structure is discussed followed by the discussion on the mortality parameters obtained using information on the estimated cause of death structure.

3.6.1 Estimates of cause of death structure

The cause of death structures estimated for Guinea and Botswana for the year 1965 are shown in Tables 3.1 and 3.2. Leaving aside for now the residual causes 'all others', one sees from Panel 1 in Table 3.1 that the estimated cause of death structure for Guinea for both genders shows that the four major causes of death are; tuberculosis (8.12%), other infectious diseases (13.61%), respiratory diseases (20.35%) and diarrhoeal diseases (10.69%). Panel 2 shows that for infancy, tuberculosis is negligible but the two other causes are still the major killers, in addition to 'certain diseases of infancy.' On the other hand, for Botswana, the major causes of death are; respiratory diseases (15.36%), cardiovascular diseases (14.99%), other infectious diseases (9.82%) and diarrhoeal diseases (8.19%). For infancy, the leading cause of death is diseases of infancy followed by respiratory diseases. These estimates are roughly consistent with expectations based on the epidemiologic transition theory.

Table 3.1 Estimation of cause of death structure for African country, # 18 (Guinea), Using 1965 data.

CRUDE DEATH RATE/000=.0300
INFANT MORTALITY RATE/000= .1970
CHILD MORTALITY RATE /000 (AGE 1-4)=.0530

Panel 1

CAUSE OF DEATH STRUCTURE
(PERCENTAGE OF DEATHS FOR ALL AGE GROUPS)

CAUSES	MALES	FEMALES	BOTH
TUBERCULOSIS	8.02	8.27	8.12
OTHER INFEC	13.53	14.05	13.61
NEOPLASMS	.83	1.59	1.26
CARDIOVASC	6.86	7.56	7.39
INF-PNEU-BRO	20.83	20.10	20.35
DIARRHOEAL	10.53	11.10	10.69
DEGENERATIVE	2.00	1.72	1.91
VIOLENCE	4.30	1.52	3.37
ALL OTHERS	24.91	24.50	24.60
INFANCY	8.19	7.61	7.99
PREGNANCY	.00	1.98	.70
TOTAL	100.00	100.00	100.00

Panel 2

CAUSE OF DEATH STRUCTURE
(PERCENTAGE OF DEATHS FOR INFANCY AND CHILDHOOD- BOTH GENDERS)

CAUSES	0-1	1-4
TUBERCULOSIS	.49	2.08
OTHER INFEC	10.31	26.73
NEOPLASMS	.04	.01
CARDIOVASC	1.52	1.13
INF-PNEU-BRO	19.27	24.29
DIARRHOEAL	17.70	19.20
DEGENERATIVE	.16	.75
VIOLENCE	.96	1.79
ALL OTHERS	23.54	24.03
INFANCY	26.00	.00
TOTAL	100.00	100.00

Source: Output from author's program CAUSE.FOR

Table 3.2 Estimation of cause of death structure for African country, # 33 (Botswana), Using 1965 data.

CRUDE DEATH RATE/000=.0120
INFANT MORTALITY RATE/000= .1080
CHILD MORTALITY RATE /000 (AGE 1-4)=.0210

Panel 1

CAUSE OF DEATH STRUCTURE
(PERCENTAGE OF DEATHS FOR ALL AGE GROUPS)

CAUSES	MALES	FEMALES	BOTH
TUBERCULOSIS	6.02	6.79	6.42
OTHER INFEC	8.83	10.76	9.82
NEOPLASMS	6.04	5.50	5.81
CARDIOVASC	16.21	13.76	14.99
INF-PNEU-BRO	14.53	16.04	15.36
DIARRHOEAL	7.43	8.96	8.19
DEGENERATIVE	3.10	2.56	2.86
VIOLENCE	7.21	2.55	5.07
ALL OTHERS	21.32	22.34	21.85
INFANCY	9.29	8.57	8.99
PREGNANCY	.00	2.17	.65
TOTAL	100.00	100.00	100.00

Panel 2

CAUSE OF DEATH STRUCTURE
(PERCENTAGE OF DEATHS FOR INFANCY AND CHILDHOOD- BOTH GENDERS)

CAUSES	0-1	1-4
TUBERCULOSIS	.42	2.02
OTHER INFEC	9.17	26.10
NEOPLASMS	.08	.30
CARDIOVASC	1.17	1.19
INF-PNEU-BRO	18.36	24.09
DIARRHOEAL	16.63	18.71
DEGENERATIVE	.17	.84
VIOLENCE	1.10	2.91
ALL OTHERS	22.60	23.85
INFANCY	30.31	.00
TOTAL	100.00	100.00

Source: Output from author's program CAUSE.FOR

Table 3.3 Estimation of cause of death structure for African country, # 18 (Guinea), Using 1983 data.

CRUDE DEATH RATE/000=.0270
INFANT MORTALITY RATE/000= .1580
CHILD MORTALITY RATE /000 (AGE 1-4)=.0360

Panel 1

CAUSE OF DEATH STRUCTURE
(PERCENTAGE OF DEATHS FOR ALL AGE GROUPS)

CAUSES	MALES	FEMALES	BOTH
TUBERCULOSIS	7.87	8.16	7.99
OTHER INFEC	13.18	13.81	13.33
NEOPLASMS	1.22	1.88	1.60
CARDIOVASC	7.55	8.02	7.96
INF-PNEU-BRO	20.36	19.80	19.98
DIARRHOEAL	10.30	10.94	10.50
DEGENERATIVE	2.08	1.79	1.98
VIOLENCE	4.52	1.60	3.50
ALL OTHERS	24.65	24.34	24.39
INFANCY	8.27	7.69	8.06
PREGNANCY	.00	1.99	.70
TOTAL	100.00	100.00	100.00

Panel 2

CAUSE OF DEATH STRUCTURE
(PERCENTAGE OF DEATHS FOR INFANCY AND CHILDHOOD- BOTH GENDERS)

CAUSES	0-1	1-4
TUBERCULOSIS	.47	2.06
OTHER INFEC	9.97	26.53
NEOPLASMS	.05	.10
CARDIOVASC	1.42	1.15
INF-PNEU-BRO	19.00	24.23
DIARRHOEAL	17.38	19.05
DEGENERATIVE	.16	.78
VIOLENCE	1.00	2.13
ALL OTHERS	23.26	23.97
INFANCY	27.29	.00
TOTAL	100.00	100.00

Source: Output from author's program CAUSE.FOR

Table 3.4 Estimation of cause of death structure for African country, # 33 (Botswana), Using 1983 data.

CRUDE DEATH RATE/000=.0090
INFANT MORTALITY RATE/000= .0740
CHILD MORTALITY RATE 000 (AGE 1-4)=.0130

Panel 1

CAUSE OF DEATH STRUCTURE
(PERCENTAGE OF DEATHS FOR ALL AGE GROUPS)

CAUSES	MALES	FEMALES	BOTH
TUBERCULOSIS	4.91	5.97	5.47
OTHER INFEC	6.22	8.95	7.71
NEOPLASMS	8.93	7.65	8.34
CARDIOVASC	21.42	17.19	19.21
INF-PNEU-BRO	11.04	13.79	12.58
DIARRHOEAL	5.71	7.78	6.80
DEGENERATIVE	3.72	3.02	3.39
VIOLENCE	8.82	3.12	6.02
ALL OTHERS	19.33	21.15	20.32
INFANCY	9.91	9.09	9.54
PREGNANCY	.00	2.27	.62
TOTAL	100.00	100.00	100.00

Panel 2

CAUSE OF DEATH STRUCTURE
(PERCENTAGE OF DEATHS FOR INFANCY AND CHILDHOOD- BOTH GENDERS)

CAUSES	0-1	1-4
TUBERCULOSIS	.36	1.96
OTHER INFEC	8.00	25.45
NEOPLASMS	.13	.59
CARDIOVASC	.81	1.25
INF-PNEU-BRO	17.44	23.88
DIARRHOEAL	15.54	18.21
DEGENERATIVE	.18	.93
VIOLENCE	1.23	4.05
ALL OTHERS	21.63	23.68
INFANCY	34.68	.00
TOTAL	100.00	100.00

Source: Output from author's program CAUSE.FOR

By 1983, the mortality level in Guinea had shown only slight improvement while for Botswana, the improvement was appreciable. The estimated cause of death structures for the year 1983 are shown in Tables 3.3 and 3.4. The leading causes of death in 1983 for Guinea were still the same but with only slight reduction in the percentage contribution of the causes of death. The percentage contribution for tuberculosis reduced to 7.99%, 'other infectious diseases' reduced minimally to 13.33% and diseases of the respiratory system also minimally reduced to 19.98%. Correspondingly, for the age groups 0-1, the leading causes of death are still respiratory diseases, diarrhoeal disease and certain diseases of infancy, and have only minimally changed in their percentage distribution. For age 1-4 years, the leading causes have remained the same as in 1965 with only very slight changes in their percentage distribution. On the contrary, Botswana on Table 3.4 shows appreciable changes from the percentages in 1965 shown in Table 3.2. The percentage distribution of the three leading causes of death in Botswana in 1983 were: cardiovascular diseases (19.21%), respiratory causes of death (12.58%) and certain diseases of infancy (9.54%) and neoplasms (8.34%). For the age groups 0-1 and 1-4, slightly more reductions were obtained for age group 0-1 than for the latter age group, while the leading causes of death in 1965 still remained the same.

In between these two extremes (Guinea and Botswana), various estimates of cause of death structure have been obtained. It is hard to verify the accuracy of these estimates except for those countries for which there are independent estimates of cause of death structure. Even for those countries where there are estimates, in most cases the data are

only a sample of registered hospital deaths which are hardly representative of national cause of death structure. Besides, there is the problem of the non-standard way of grouping the causes and hence there is mismatch between those used in the observed and the estimated data.

As a first assessment of the plausibility of the model, we could use an internal consistency check. This could be applied in two ways for two or more countries at different levels of mortality at the same point in time and for the same country at different levels of mortality at two or more points in time. Considering that Guinea and Botswana are at widely different levels of mortality in 1965, one would expect significant difference in the estimates of their cause of death structures. Tables 3.1 and 3.2 reveal that some estimates do show marked differences, but some others like 'pregnancy related causes' hardly show any difference. In addition to infectious and parasitic diseases, this is one group of causes of death that one would expect to be sensitive to improvement in health. This would suggest that the regression coefficients for this group of causes of death are not quite sensitive enough.

As far as temporal change in cause of death structures is concerned, the estimates discussed above are acceptable in that the cause of death structure showed minimal change in Guinea whose reported life expectancy changed only slightly. The improvement in health over 1965-1983 in Botswana caused some reduction in the percentage distribution of the leading causes of death and even a change in their ranks.

As a second assessment of the plausibility of the model, we could use an external consistency check by comparing the estimated results with observed data. In the first case, we use the data on registered causes of death in urban areas of Ghana in 1966. The urban population comprised 15% of the total population and the study period was from July to September. Table 3.5 shows a comparison between the observed (urban) and the estimated (national) cause of death structure using a cause of death grouping that allows for comparability. It would have been better to compare estimate for urban area with the observed rates for the same area. For that to be done, estimate is needed for mortality in the urban area which was not available for this analysis. Taking into account that there is marked seasonality in mortality in Africa and that mortality patterns in rural areas are quite different from those in urban areas, the reported data could hardly be said to be representative of the national population. Nevertheless, it can still allow rough comparisons. Table 3.5 shows a good agreement in the estimate for infectious diseases with the observed. For neoplasm, respiratory diseases and cardiovascular diseases, the model tends to overestimate.

As a second illustration, the observed cause of death structure in rural Kenya is compared with the estimated cause of death structure in Table 3.6. The observed data was obtained from the demographic surveillance system in the Machakos project in rural Kenya. The estimated cause of death data was obtained using 1983 data. The table shows even better agreement between the observed data and the estimated results than in the Ghanaian case. Diseases of the respiratory system which appeared to be overestimated in

Table 3.5 Comparison between estimated cause of death structure in Ghana (1965) and observed cause of death structure in Ghana (1966) based on reported hospital deaths

Cause of death	Percentage observed (Ghana, 1966)	Percentage estimated (Ghana, 1965)
Infectious and parasitic diseases	26.0	27.76
Neoplasms	2.7	3.92
Diseases of the circulatory system	5.1	11.83
Diseases of the respiratory system	9.9	17.44
Accidents and poisoning	6.6	4.37
Maternal causes of death	2.1	.67
Certain diseases of early infancy	12.4	8.57
All others (residual)	35.2	25.44
Total	100.0	100.00

Sources:

1) Miltenyi (1971) (for the observed data)
2) Output from author's program CAUSE.FOR (for the estimated data)

Table 3.6 Comparison between estimated cause of death structure and observed cause of death structure in rural Kenya (1975-78)

Cause of death	Percentage observed (Kenya, 1975-1978)	Percentage estimated (Kenya, 1983)
Tuberculosis	7.1	6.42
Other infectious and parasitic diseases	17.6[a]	11.68
Neoplasms	3.7	5.81
Diseases of the circulatory system	7.2	14.99
Diseases of the respiratory system	15.8	15.36
Diarrhoeal diseases	10.8	9.42
Accidents and poisoning	6.2	5.07
Certain diseases of early infancy	12.8	8.99
All others (residual)	18.8	22.26
Total	100.0	100.00

a- Includes measles and malaria

Sources:

1) Omondi-Odiambo *et al* (1990) (for the observed data)
2) Output from author's program CAUSE.FOR (for the estimated data)

Ghana now show very good agreement with the data. However, cardiovascular diseases appear to be overestimated just as in the Ghanaian case. This is partly reflects the differences in mortality between African countries and those used in the data base on which the regression model was based. This problem is often encountered in indirect demographic estimation for Africa.

3.6.2 Estimates of mortality parameters incorporating information on causes of death

The life table survivors (l_x values) obtained from the output of CAUSE.FOR have taken into account information on cause of death structure. The values were found to be more realistic when compared to estimates that do not take cause of death structure into account. When the equation (3) was used to estimate l_x values using another program (AGEDEATH.FOR), the values obtained tended to be very high. For example, for country 1 (Mali) in 1965, the life table survivorship l_1/l_0 was estimated at .758 and .771 for males and females respectively with the model incorporating cause of death (model 1). For model 2 (not incorporating cause of death information), the same survivorship proportion were .804 and .808 for males and females respectively.

On application of Brass' logit model to the derived l_x data, values were obtained for α, β and $-\alpha/\beta$ for both males and females for both periods, 1965 and 1983. Another index for level of mortality similar to Anson's (1991) rectangularity (U) was also computed. Rectangularity (U) is defined here as:

$$U = \frac{[{}_{20}p_{40}]}{[{}_{20}p^{2}_{20}]}, \text{ where } [{}_{n}p_{x}] = \frac{l_{x+n}}{l_{x}} \qquad (13)$$

Anson had actually used ${}_{25}p_{35}$ in place of ${}_{20}p_{40}$ and ${}_{20}p_{15}$ in place of ${}_{20}p_{20}$. The measure relates middle age mortality to young adult mortality and was found to correlate strongly with level of mortality. The computed values for α, β, $-\alpha/\beta$ and U are shown in Tables 3.7 through 3.10 for the 40 countries. In 1965, the level of mortality, given by $-\alpha/\beta$, ranges from -.63 to .513 for males and from -.628 to .378 for females. For 1983, the values range from -.513 to .777 for males and from -.521 to .603 for females. Bearing in mind that low values of $-\alpha/\beta$ correspond to high levels of mortality and that high values of $-\alpha/\beta$ correspond to low levels of mortality, one can make the following general observations: While a general improvement in mortality is noted over the period 1965-1983, the disparity among countries between the highest and lowest mortality levels widened over time. More improvement occurred in the lower mortality end than in the higher mortality end of the spectrum.

For the measure of rectangularity, the lower the values, the lower the middle age mortality compared to young adult mortality. With this in mind, one observes that the measures show slight improvement over the period for both males and females. The highest values for males in 1965 was 1.049 compared to 1.039 in 1985 and the smallest in 1965 was .987 compared to .98 in 1985. The changes for females is similar, The highest values of rectangularity was 1.114 in 1965 compared to 1.095 in 1985 and the smallest value in 1965 was 1.007 compared to .995 in 1985.

The geographical distribution shows marked heterogeneity. Nevertheless, one notes that the countries showing the highest values of $-\alpha/\beta$ and rectangularity comprise mostly of West African countries and those showing the lowest values of these parameters comprise mostly of Southern and East African countries. This has been the case for both the periods.

3.6.3 Estimates of age-cause-specific death rates

Using the program SURVCH.FOR, age-cause-specific death rates were obtained for all 33 African countries included in the data base. Two causes are considered, tuberculosis (a lethal infectious disease) and all other causes of death beside TB (described as 'all others'). As was done in an earlier subsection, discussion is restricted to the two countries at the low and high ends of mortality levels, namely Guinea and Botswana, for the two periods 1965 and 1983. The estimates for these countries are given in Tables 3.11 through 3.18. For both TB and 'all others,' the data in all the tables show a general rise with age. Also due to the smoothing effect of the logit model, the rates show gradual rise rather sudden increase.

As age-cause-specific death rates are hardly available in most African countries, comparison of the estimated results with external sources is not possible. One can however make some internal consistency checks for the plausibility of the estimates. First, the tables show that for both periods, the rates for TB and 'all others' is higher in Guinea

than in Botswana for both males and females. Secondly, for Guinea as well as for Botswana, there was considerable decline in the rates from 1965 to 1983. For example in Guinea, the age-cause-specific death rate for TB for 15 year old males show a decline of about 27% from 26 per 100,000 to 19 per 100,000. For Botswana, death rate for the same age group for the same cause show a much higher decline of about 64%.

Since the age-cause-specific death rates obtained made use of α and β parameters obtained by indirect estimation, one can see a weakness here. There is the possibility of accumulation of errors if errors were present in the first indirect estimates. In the same vein, the regression coefficients used to relate the α and β parameters with their cause specific variants may not be representative for all African countries. Lastly, there is also the valid argument that a two-parameter model could not adequately describe cause-specific and general mortality for all African countries. In defence, one may question the appropriateness of higher parameter models when one only has a fragmentary data base. Notwithstanding this reservation, an attempt was made to fit the eight parameter Heligman-Pollard model to cause specific data and to find relationship between the parameters. Not only was the fit found to be poor, there was hardly any relationship between the parameters.

3.7 Conclusion

This chapter has dealt with the scarce literature on mortality and causes of death in Africa. The various approaches that hold potential for indirect estimation of cause of death structure were also reviewed.

The method opted for obtaining the cause of death structure is one based on the synthesis of Preston's regression methodology and Brass logit model. In the first part, general mortality information was used with Preston's model to estimate cause of death structure. The results obtained were compared with some rough observed estimates. While the model is broadly consistent with expectations, it overestimates for certain causes of death for example, circulatory diseases. This exercise could have been improved if the model specifications of Lundy were available. Also, were the model specifications of Blum and Fargues available, maternal mortality rates could have been independently estimated and used to refine the other cause of death estimates for females.

In the second part, using Preston's regression equations relating life table functions to cause of death structure, life table survivorship values were estimated which were then summarized in parametric form. The parameters were finally used in another model to obtain age-cause-specific death rates.

Part of the idea of using data for several African countries at two points in time was to assess the behaviour of the model. The ultimate aim however was to obtain age-cause-specific death rates to be used later in the work.

Table 3.7 Estimated mortality parameters for several African countries, males, 1965

COUNTRIES	ALPHA	BETA	-ALPHA/BETA	U
GUINEA-B	0.49	0.778	-0.630	1.049
GUINEA	0.49	0.778	-0.630	1.049
ANGOLA	0.456	0.769	-0.593	1.046
MALAWI	0.456	0.769	-0.593	1.046
SOMALIA	0.421	0.76	-0.554	1.042
GAMBIA	0.421	0.76	-0.554	1.042
MALI	0.386	0.752	-0.513	1.039
MOZAMBIQUE	0.386	0.752	-0.513	1.039
CHAD	0.35	0.744	-0.470	1.035
BENIN	0.313	0.735	-0.426	1.032
NIGER	0.313	0.735	-0.426	1.032
MAURITANIA	0.313	0.735	-0.426	1.032
BURUNDI	0.275	0.727	-0.378	1.028
SUDAN	0.275	0.727	-0.378	1.028
BURKINA	0.275	0.727	-0.378	1.028
C.A.R.	0.275	0.727	-0.378	1.028
NIGERIA	0.236	0.719	-0.328	1.025
SENEGAL	0.236	0.719	-0.328	1.025
ZAIRE	0.236	0.719	-0.328	1.025
TOGO	0.236	0.719	-0.328	1.025
GABON	0.196	0.711	-0.276	1.021
COTE d'IV.	0.196	0.711	-0.276	1.021
LIBERIA	0.196	0.711	-0.276	1.021
TANZANIA	0.196	0.711	-0.276	1.021
MADAGASCAR	0.154	0.703	-0.219	1.018
SWAZILAND	0.154	0.703	-0.219	1.018
CAMEROON	0.111	0.695	-0.160	1.014
ZAMBIA	0.111	0.695	-0.160	1.014
UGANDA	0.066	0.687	-0.096	1.011
LESOTHO	0.02	0.68	-0.029	1.007
RWANDA	-0.03	0.672	0.045	1.004
KENYA	-0.03	0.672	0.045	1.004
NAMIBIA	-0.082	0.665	0.123	1
GHANA	-0.082	0.665	0.123	1
CONGO	-0.195	0.651	0.300	0.994
ZIMBABWE	-0.195	0.651	0.300	0.994
BOTSWANA	-0.328	0.64	0.513	0.987

Source: Output from author's program CAUSE.FOR

Table 3.8 Estimated mortality parameters for several African countries, females, 1965

COUNTRIES	ALPHA	BETA	-ALPHA BETA	U
GUINEA-B	0.515	0.82	-0.628	1.114
GUINEA	0.515	0.82	-0.628	1.114
ANGOLA	0.482	0.811	-0.594	1.108
MALAWI	0.482	0.811	-0.594	1.108
SOMALIA	0.448	0.802	-0.559	1.101
GAMBIA	0.448	0.802	-0.559	1.101
MALI	0.414	0.794	-0.521	1.095
MOZAMBIQUE	0.414	0.794	-0.521	1.095
CHAD	0.38	0.785	-0.484	1.089
BENIN	0.344	0.777	-0.443	1.083
NIGER	0.344	0.777	-0.443	1.083
MAURITANIA	0.344	0.777	-0.443	1.083
BURUNDI	0.308	0.768	-0.401	1.077
SUDAN	0.308	0.768	-0.401	1.077
BURKINA	0.308	0.768	-0.401	1.077
C.A.R.	0.308	0.768	-0.401	1.077
NIGERIA	0.27	0.76	-0.355	1.071
SENEGAL	0.27	0.76	-0.355	1.071
ZAIRE	0.27	0.76	-0.355	1.071
TOGO	0.27	0.76	-0.355	1.071
GABON	0.232	0.751	-0.309	1.065
COTE d'IV.	0.232	0.751	-0.309	1.065
LIBERIA	0.232	0.751	-0.309	1.065
TANZANIA	0.232	0.751	-0.309	1.065
MADAGASCAR	0.192	0.743	-0.258	1.059
SWAZILAND	0.192	0.743	-0.258	1.059
CAMEROON	0.151	0.735	-0.205	1.053
ZAMBIA	0.151	0.735	-0.205	1.053
UGANDA	0.109	0.727	-0.150	1.047
LESOTHO	0.065	0.718	-0.091	1.041
RWANDA	0.019	0.71	-0.027	1.035
KENYA	0.019	0.71	-0.027	1.035
NAMIBIA	-0.029	0.702	0.041	1.03
GHANA	-0.029	0.702	0.041	1.03
CONGO	-0.134	0.685	0.196	1.018
ZIMBABWE	-0.134	0.685	0.196	1.018
BOTSWANA	-0.253	0.669	0.378	1.007

Source: Output from author's program CAUSE.FOR

Table 3.9 Estimated mortality parameters for several African countries, males, 1983

COUNTRIES	ALPHA	BETA	-ALPHA/BETA	U
GUINEA-B	0.386	0.752	-0.513	1.039
SIERRA L.	0.386	0.752	-0.513	1.039
GUINEA	0.386	0.752	-0.513	1.039
MALAWI	0.236	0.719	-0.328	1.025
GAMBIA	0.236	0.719	-0.328	1.025
ANGOLA	0.196	0.711	-0.276	1.021
MALI	0.154	0.703	-0.219	1.018
CHAD	0.154	0.703	-0.219	1.018
BURKINA	0.154	0.703	-0.219	1.018
NIGER	0.111	0.695	-0.160	1.014
SOMALIA	0.111	0.695	-0.160	1.014
BURUNDI	0.066	0.687	-0.096	1.011
SENEGAL	0.066	0.687	-0.096	1.011
MOZAMBIQUE	0.066	0.687	-0.096	1.011
RWANDA	0.066	0.687	-0.096	1.011
MAURITANIA	0.066	0.687	-0.096	1.011
MADAGASCAR	0.02	0.68	-0.029	1.007
LIBERIA	0.02	0.68	-0.029	1.007
BENIN	0.02	0.68	-0.029	1.007
TOGO	0.02	0.68	-0.029	1.007
SUDAN	-0.03	0.672	0.045	1.004
NIGERIA	-0.03	0.672	0.045	1.004
C.A.R.	-0.03	0.672	0.045	1.004
GABON	-0.03	0.672	0.045	1.004
UGANDA	-0.03	0.672	0.045	1.004
ZAMBIA	-0.082	0.665	0.123	1
ZAIRE	-0.082	0.665	0.123	1
TANZANIA	-0.082	0.665	0.123	1
LESOTHO	-0.137	0.658	0.208	0.997
CAMEROON	-0.137	0.658	0.208	0.997
COTE d'IV.	-0.195	0.651	0.300	0.994
SWAZILAND	-0.195	0.651	0.300	0.994
ZIMBABWE	-0.259	0.645	0.402	0.99
KENYA	-0.328	0.64	0.513	0.987
NAMIBIA	-0.404	0.635	0.636	0.983
GHANA	-0.49	0.631	0.777	0.98
BOTSWANA	-0.589	0.63	0.935	0.977

Source: Output from author's program CAUSE.FOR

Table 3.10 Estimated mortality parameters for several African countries, females, 1983

COUNTRIES	ALPHA	BETA	-ALPHA/BETA	U
GUINEA-B	0.414	0.794	-0.521	1.095
SIERRA L.	0.414	0.794	-0.521	1.095
GUINEA	0.414	0.794	-0.521	1.095
MALAWI	0.27	0.76	-0.355	1.071
GAMBIA	0.27	0.76	-0.355	1.071
ANGOLA	0.232	0.751	-0.309	1.065
MALI	0.192	0.743	-0.258	1.059
CHAD	0.192	0.743	-0.258	1.059
BURKINA	0.192	0.743	-0.258	1.059
NIGER	0.151	0.735	-0.205	1.053
SOMALIA	0.151	0.735	-0.205	1.053
BURUNDI	0.109	0.727	-0.150	1.047
SENEGAL	0.109	0.727	-0.150	1.047
MOZAMBIQUE	0.109	0.727	-0.150	1.047
RWANDA	0.109	0.727	-0.150	1.047
MAURITANIA	0.109	0.727	-0.150	1.047
MADAGASCAR	0.065	0.718	-0.091	1.041
LIBERIA	0.065	0.718	-0.091	1.041
BENIN	0.065	0.718	-0.091	1.041
TOGO	0.065	0.718	-0.091	1.041
SUDAN	0.019	0.71	-0.027	1.035
NIGERIA	0.019	0.71	-0.027	1.035
C.A.R.	0.019	0.71	-0.027	1.035
GABON	0.019	0.71	-0.027	1.035
UGANDA	0.019	0.71	-0.027	1.035
ZAMBIA	-0.029	0.702	0.041	1.03
ZAIRE	-0.029	0.702	0.041	1.03
TANZANIA	-0.029	0.702	0.041	1.03
LESOTHO	-0.08	0.693	0.115	1.024
CAMEROON	-0.08	0.693	0.115	1.024
COTE d'IV.	-0.134	0.685	0.196	1.018
SWAZILAND	-0.134	0.685	0.196	1.018
ZIMBABWE	-0.191	0.677	0.282	1.012
KENYA	-0.253	0.669	0.378	1.007
NAMIBIA	-0.32	0.66	0.485	1.001
GHANA	-0.393	0.652	0.603	0.995
BOTSWANA	-0.475	0.643	0.739	0.99

Source: Output from author's program CAUSE.FOR

Table 3.11 Estimated age-cause specific death rates for tuberculosis (TB) and for 'all other causes,' Guinea, males, 1965.

Cause-specific logit model parameters

For TB, α_i= -0.0509 β_i= 0.4755
For ALL OTHERS, α_i= 0.4180 β_i= 0.7367

Estimated age-cause-specific death rates (Mx)

AGE	Mx (TB)	Mx (ALL OTHERS)
15	0.0002647	0.0005100
16	0.0002610	0.0005879
17	0.0005144	0.0006465
18	0.0005008	0.0007237
19	0.0004882	0.0008015
20	0.0007121	0.0008979
21	0.0006883	0.0009944
22	0.0008849	0.0010513
23	0.0010588	0.0010885
24	0.0012093	0.0011062
25	0.0013356	0.0011239
26	0.0014463	0.0011611
27	0.0015332	0.0011976
28	0.0016112	0.0012920
29	0.0016726	0.0014052
30	0.0017252	0.0015174
31	0.0017675	0.0016294
32	0.0018052	0.0017402
33	0.0018339	0.0018315
34	0.0018589	0.0019201
35	0.0017701	0.0020098
36	0.0017971	0.0020985
37	0.0017209	0.0022061
38	0.0018429	0.0023495
39	0.0018581	0.0024921
40	0.0019587	0.0026519
41	0.0020465	0.0028283
42	0.0020426	0.0029843
43	0.0019622	0.0031201
44	0.0018900	0.0032523
45	0.0017518	0.0033849
46	0.0016269	0.0035340
47	0.0016481	0.0037347
48	0.0016670	0.0039694
49	0.0018120	0.0042522
50	0.0019430	0.0045857
51	0.0020630	0.0049119
52	0.0021110	0.0052162
53	0.0020442	0.0054603
54	0.0019282	0.0057015
55	0.0018210	0.0059529
56	0.0017221	0.0062507
57	0.0016806	0.0066264
58	0.0017402	0.0071116
59	0.0018426	0.0076832
60	0.0019382	0.0083391
61	0.0020253	0.0089928
62	0.0020617	0.0095944
63	0.0020517	0.0100793
64	0.0019999	0.0105180

65	0.0019112	0.0109580
66	0.0018269	0.0115037
67	0.0017884	0.0123497
68	0.0017900	0.0135383
69	0.0017541	0.0149559
70	0.0017563	0.0164574
71	0.0017589	0.0178727
72	0.0017255	0.0191467
73	0.0016934	0.0202729
74	0.0016285	0.0212596
75	0.0015667	0.0221805
76	0.0015082	0.0230793
77	0.0014523	0.0240184
78	0.0013991	0.0250339

Source: Output from author's program SURVCH.FOR

Table 3.12 Estimated age-cause specific death rates for tuberculosis (TB) and for 'all other causes,' Guinea, females, 1965.

Cause-specific logit model parameters

For TB, α_i = 0.0850 β_i = 0.5365
For ALL OTHERS, α_i = 0.4432 β_i =0.7766

Estimated age-cause-specific death rates (Mx)

AGE	Mx (TB)	Mx (ALL OTHERS)
15	0.0002428	0.0005308
16	0.0002397	0.0006119
17	0.0004737	0.0006728
18	0.0004625	0.0007532
19	0.0004523	0.0008342
20	0.0006620	0.0009348
21	0.0006423	0.0010354
22	0.0008294	0.0010946
23	0.0009975	0.0011334
24	0.0011458	0.0011521
25	0.0012732	0.0011707
26	0.0013877	0.0012094
27	0.0014805	0.0012477
28	0.0015662	0.0013460
29	0.0016365	0.0014642
30	0.0016990	0.0015816
31	0.0017513	0.0016984
32	0.0017997	0.0018142
33	0.0018394	0.0019097
34	0.0018752	0.0020024
35	0.0017951	0.0020965
36	0.0018321	0.0021894
37	0.0017627	0.0023019
38	0.0018967	0.0024522
39	0.0019215	0.0026017
40	0.0020351	0.0027688
41	0.0021363	0.0029539
42	0.0021422	0.0031178
43	0.0020670	0.0032605
44	0.0019991	0.0033997
45	0.0018599	0.0035390
46	0.0017331	0.0036961
47	0.0017612	0.0039075
48	0.0017871	0.0041541
49	0.0019487	0.0044514
50	0.0020964	0.0048025
51	0.0022339	0.0051461
52	0.0022939	0.0054671
53	0.0022289	0.0057252
54	0.0021091	0.0059806
55	0.0019975	0.0062472
56	0.0018942	0.0065626
57	0.0018532	0.0069601
58	0.0019235	0.0074735
59	0.0020422	0.0080783
60	0.0021534	0.0087726
61	0.0022562	0.0094656
62	0.0023032	0.0101048
63	0.0022978	0.0106221
64	0.0022457	0.0110911

65	0.0021511	0.0115623
66	0.0020610	0.0121458
67	0.0020218	0.0130472
68	0.0020276	0.0143126
69	0.0019910	0.0158224
70	0.0019973	0.0174240
71	0.0020042	0.0189368
72	0.0019697	0.0203027
73	0.0019365	0.0215136
74	0.0018655	0.0225784
75	0.0017976	0.0235742
76	0.0017332	0.0245474
77	0.0016714	0.0255645
78	0.0016121	0.0266633

Source: Output from author's program SURVCH.FOR

Table 3.13 Estimated age-cause specific death rates for tuberculosis (TB) and for 'all other causes,' Botswana, males, 1965.

Cause-specific logit model parameters

For TB, α_i = -1.7333 β_i = 0.3746
For ALL OTHERS, α_i = -0.3050 β_i = 0.6747

Estimated age-cause-specific death rates (Mx)

AGE	Mx (TB)	Mx (ALL OTHERS)
15	0.0000163	0.0001711
16	0.0000161	0.0001973
17	0.0000316	0.0002170
18	0.0000306	0.0002430
19	0.0000297	0.0002692
20	0.0000431	0.0003017
21	0.0000414	0.0003343
22	0.0000528	0.0003534
23	0.0000628	0.0003662
24	0.0000711	0.0003724
25	0.0000779	0.0003784
26	0.0000836	0.0003912
27	0.0000876	0.0004037
28	0.0000915	0.0004358
29	0.0000940	0.0004742
30	0.0000962	0.0005125
31	0.0000975	0.0005506
32	0.0000990	0.0005886
33	0.0000996	0.0006198
34	0.0001002	0.0006505
35	0.0000947	0.0006815
36	0.0000954	0.0007122
37	0.0000909	0.0007494
38	0.0000967	0.0007990
39	0.0000968	0.0008484
40	0.0001016	0.0009038
41	0.0001055	0.0009652
42	0.0001045	0.0010197
43	0.0001000	0.0010677
44	0.0000957	0.0011144
45	0.0000883	0.0011616
46	0.0000818	0.0012147
47	0.0000824	0.0012857
48	0.0000831	0.0013690
49	0.0000900	0.0014692
50	0.0000961	0.0015877
51	0.0001016	0.0017043
52	0.0001035	0.0018141
53	0.0000999	0.0019036
54	0.0000938	0.0019927
55	0.0000884	0.0020863
56	0.0000833	0.0021966
57	0.0000810	0.0023356
58	0.0000838	0.0025146
59	0.0000884	0.0027259
60	0.0000927	0.0029696
61	0.0000967	0.0032150
62	0.0000980	0.0034450
63	0.0000972	0.0036355
64	0.0000946	0.0038119

65	0.0000902	0.0039911
66	0.0000859	0.0042116
67	0.0000840	0.0045463
68	0.0000839	0.0050138
69	0.0000820	0.0055755
70	0.0000820	0.0061800
71	0.0000820	0.0067648
72	0.0000803	0.0073091
73	0.0000785	0.0078097
74	0.0000755	0.0082683
75	0.0000725	0.0087127
76	0.0000698	0.0091601
77	0.0000670	0.0096355
78	0.0000644	0.0101552

Source: Output from author's program SURVCH.FOR

Table 3.14 Estimated age-cause specific death rates for tuberculosis (TB) and for 'all other causes,' Botswana, females, 1965.

Cause-specific logit model parameters

For TB, α_i = -1.5425 β_i = 0.4089
For ALL OTHERS, α_i = -0.2374 β_i = 0.6969

Estimated age-cause-specific death rates (Mx)

AGE	Mx (TB)	Mx (ALL OTHERS)
15	0.0000199	0.0001917
16	0.0000195	0.0002210
17	0.0000386	0.0002432
18	0.0000374	0.0002723
19	0.0000363	0.0003017
20	0.0000530	0.0003382
21	0.0000509	0.0003747
22	0.0000652	0.0003963
23	0.0000777	0.0004105
24	0.0000882	0.0004174
25	0.0000970	0.0004244
26	0.0001044	0.0004386
27	0.0001101	0.0004528
28	0.0001150	0.0004886
29	0.0001186	0.0005318
30	0.0001218	0.0005747
31	0.0001241	0.0006176
32	0.0001262	0.0006601
33	0.0001275	0.0006954
34	0.0001287	0.0007297
35	0.0001220	0.0007645
36	0.0001234	0.0007991
37	0.0001177	0.0008407
38	0.0001256	0.0008965
39	0.0001262	0.0009520
40	0.0001325	0.0010143
41	0.0001382	0.0010831
42	0.0001373	0.0011446
43	0.0001315	0.0011984
44	0.0001264	0.0012512
45	0.0001168	0.0013041
46	0.0001083	0.0013639
47	0.0001093	0.0014439
48	0.0001105	0.0015376
49	0.0001198	0.0016502
50	0.0001282	0.0017837
51	0.0001359	0.0019148
52	0.0001388	0.0020386
53	0.0001340	0.0021394
54	0.0001262	0.0022399
55	0.0001190	0.0023454
56	0.0001124	0.0024698
57	0.0001096	0.0026264
58	0.0001133	0.0028281
59	0.0001197	0.0030665
60	0.0001259	0.0033412
61	0.0001314	0.0036180
62	0.0001334	0.0038773
63	0.0001327	0.0040925
64	0.0001292	0.0042919

65	0.0001233	0.0044944
66	0.0001179	0.0047437
67	0.0001151	0.0051215
68	0.0001153	0.0056491
69	0.0001128	0.0062830
70	0.0001129	0.0069654
71	0.0001129	0.0076259
72	0.0001108	0.0082408
73	0.0001085	0.0088062
74	0.0001044	0.0093245
75	0.0001004	0.0098267
76	0.0000966	0.0103316
77	0.0000930	0.0108683
78	0.0000895	0.0114543

Source: Output from author's program SURVCH.FOR

Table 3.15 Estimated age-cause specific death rates for tuberculosis (TB) and for 'all other causes,' Guinea, males, 1983.

Cause-specific logit model parameters

For TB, $\alpha_1 = -0.2837$ $\beta_1 = 0.4497$
For ALL OTHERS, $\alpha_2 = 0.3254$ $\beta_2 = 0.7203$

Estimated age-cause-specific death rates (Mx)

AGE	Mx (TB)	Mx (ALL OTHERS)
15	0.0001937	0.0004517
16	0.0001908	0.0005205
17	0.0003757	0.0005725
18	0.0003655	0.0006408
19	0.0003559	0.0007098
20	0.0005184	0.0007951
21	0.0005002	0.0008808
22	0.0006423	0.0009311
23	0.0007671	0.0009641
24	0.0008742	0.0009798
25	0.0009634	0.0009958
26	0.0010409	0.0010286
27	0.0011009	0.0010612
28	0.0011541	0.0011447
29	0.0011955	0.0012451
30	0.0012303	0.0013448
31	0.0012576	0.0014442
32	0.0012821	0.0015424
33	0.0012997	0.0016234
34	0.0013151	0.0017022
35	0.0012499	0.0017821
36	0.0012669	0.0018609
37	0.0012113	0.0019564
38	0.0012953	0.0020840
39	0.0013040	0.0022108
40	0.0013727	0.0023527
41	0.0014322	0.0025098
42	0.0014274	0.0026486
43	0.0013695	0.0027696
44	0.0013174	0.0028877
45	0.0012199	0.0030058
46	0.0011318	0.0031388
47	0.0011456	0.0033178
48	0.0011576	0.0035273
49	0.0012572	0.0037791
50	0.0013469	0.0040770
51	0.0014287	0.0043680
52	0.0014607	0.0046399
53	0.0014132	0.0048587
54	0.0013319	0.0050748
55	0.0012570	0.0053006
56	0.0011879	0.0055677
57	0.0011586	0.0059044
58	0.0011989	0.0063395
59	0.0012688	0.0068521
60	0.0013338	0.0074405
61	0.0013928	0.0080278
62	0.0014171	0.0085697
63	0.0014094	0.0090081
64	0.0013731	0.0094059

65	0.0013114	0.0098056
66	0.0012532	0.0103008
67	0.0012261	0.0110660
68	0.0012267	0.0121403
69	0.0012016	0.0134228
70	0.0012028	0.0147842
71	0.0012041	0.0160715
72	0.0011808	0.0172359
73	0.0011585	0.0182705
74	0.0011137	0.0191828
75	0.0010713	0.0200385
76	0.0010310	0.0208773
77	0.0009925	0.0217558
78	0.0009559	0.0227066

Source: Output from author's program SURVCH.FOR

Table 3.16 Estimated age-cause specific death rates for tuberculosis (TB) and for 'all other causes,' Guinea, females, 1983.

Cause-specific logit model parameters

For TB, α_1= -0.1428 β_1= 0.5103
For ALL OTHERS, α_2= 0.3532 β_2= 0.7600

Estimated age-cause-specific death rat · (Mx)

AGE	Mx (TB)	Mx (ALL OTHERS)
15	0.0001808	0.0004717
16	0.0001785	0.0005439
17	0.0003522	0.0005980
18	0.0003436	0.0006696
19	0.0003357	0.0007416
20	0.0004904	0.0008310
21	0.0004753	0.0009205
22	0.0006127	0.0009732
23	0.0007354	0.0010078
24	0.0008430	0.0010244
25	0.0009346	0.0010411
26	0.0010162	0.0010755
27	0.0010817	0.0011097
28	0.0011414	0.0011972
29	0.0011899	0.0013025
30	0.0012324	0.0014069
31	0.0012678	0.0015111
32	0.0012999	0.0016141
33	0.0013258	0.0016993
34	0.0013490	0.0017820
35	0.0012892	0.0018659
36	0.0013133	0.0019488
37	0.0012617	0.0020494
38	0.0013555	0.0021833
39	0.0013711	0.0023167
40	0.0014500	0.0024661
41	0.0015201	0.0026313
42	0.0015220	0.0027776
43	0.0014666	0.0029054
44	0.0014166	0.0030300
45	0.0013165	0.0031548
46	0.0012257	0.0032955
47	0.0012445	0.0034845
48	0.0012615	0.0037055
49	0.0013744	0.0039716
50	0.0014775	0.0042860
51	0.0015727	0.0045940
52	0.0016135	0.0048818
53	0.0015664	0.0051141
54	0.0014810	0.0053440
55	0.0014017	0.0055841
56	0.0013283	0.0058680
57	0.0012988	0.0062261
58	0.0013474	0.0066880
59	0.0014296	0.0072325
60	0.0015067	0.0078579
61	0.0015778	0.0084830
62	0.0016095	0.0090611
63	0.0016049	0.0095304
64	0.0015677	0.0099577

65	0.0015010	0.0103873
66	0.0014375	0.0109190
67	0.0014096	0.0117377
68	0.0014132	0.0128862
69	0.0013871	0.0142578
70	0.0013911	0.0157160
71	0.0013955	0.0170980
72	0.0013710	0.0183515
73	0.0013476	0.0194689
74	0.0012977	0.0204575
75	0.0012504	0.0213869
76	0.0012052	0.0222992
77	0.0011619	0.0232547
78	0.0011206	0.0242880

Source: Output from author's program SURVCH.FOR

Table 3.17 Estimated age-cause specific death rates for tuberculosis (TB) and for 'all other causes,' Botswana, males, 1983.

Cause-specific logit model parameters

For TB, α_1= -2.1940 β_1= 0.3945
For ALL OTHERS, α_2= -0.5329 β_2= 0.6892

Estimated age-cause-specific death rates (Mx)

AGE	Mx (TB)	Mx (ALL OTHERS)
15	0.0000058	0.0001142
16	0.0000058	0.0001314
17	0.0000113	0.0001447
18	0.0000109	0.0001621
19	0.0000107	0.0001795
20	0.0000155	0.0002013
21	0.0000149	0.0002230
22	0.0000190	0.0002359
23	0.0000228	0.0002445
24	0.0000257	0.0002485
25	0.0000282	0.0002528
26	0.0000305	0.0002612
27	0.0000319	0.0002696
28	0.0000333	0.0002911
29	0.0000344	0.0003168
30	0.0000353	0.0003425
31	0.0000359	0.0003682
32	0.0000365	0.0003936
33	0.0000368	0.0004147
34	0.0000370	0.0004352
35	0.0000351	0.0004562
36	0.0000354	0.0004768
37	0.0000338	0.0005019
38	0.0000359	0.0005354
39	0.0000362	0.0005686
40	0.0000380	0.0006060
41	0.0000394	0.0006475
42	0.0000392	0.0006844
43	0.0000375	0.0007168
44	0.0000361	0.0007488
45	0.0000332	0.0007808
46	0.0000308	0.0008170
47	0.0000311	0.0008652
48	0.0000314	0.0009218
49	0.0000341	0.0009901
50	0.0000363	0.0010705
51	0.0000386	0.0011500
52	0.0000392	0.0012251
53	0.0000380	0.0012867
54	0.0000357	0.0013482
55	0.0000337	0.0014126
56	0.0000318	0.0014889
57	0.0000309	0.0015846
58	0.0000319	0.0017080
59	0.0000338	0.0018536
60	0.0000356	0.0020218
61	0.0000370	0.0021919
62	0.0000374	0.0023522
63	0.0000373	0.0024860
64	0.0000364	0.0026108

65	0.0000346	0.0027380
66	0.0000331	0.0028945
67	0.0000324	0.0031302
68	0.0000323	0.0034591
69	0.0000317	0.0038548
70	0.0000318	0.0042833
71	0.0000316	0.0047008
72	0.0000311	0.0050935
73	0.0000304	0.0054587
74	0.0000292	0.0057976
75	0.0000282	0.0061293
76	0.0000269	0.0064661
77	0.0000261	0.0068260
78	0.0000250	0.0072208

Source: Output from author's program SURVCH.FOR

Table 3.18 Estimated age-cause specific death rates for tuberculosis (TB) and for 'all other causes,' Botswana, females, 1983.

Cause-specific logit model parameters

For TB, α_1= -1.9735 $ß_1$= 0.3990
For ALL OTHERS, α_2= -0.4327 $ß_2$= 0.6916

Estimated age-cause-specific death rates (Mx)

AGE	Mx (TB)	Mx (ALL OTHERS)
15	0.0000089	0.0001366
16	0.0000086	0.0001574
17	0.0000173	0.0001732
18	0.0000165	0.0001940
19	0.0000162	0.0002149
20	0.0000236	0.0002409
21	0.0000227	0.0002670
22	0.0000291	0.0002825
23	0.0000344	0.0002925
24	0.0000392	0.0002975
25	0.0000430	0.0003025
26	0.0000463	0.0003127
27	0.0000487	0.0003227
28	0.0000508	0.0003484
29	0.0000524	0.0003792
30	0.0000537	0.0004098
31	0.0000548	0.0004406
32	0.0000555	0.0004709
33	0.0000562	0.0004960
34	0.0000565	0.0005208
35	0.0000535	0.0005456
36	0.0000541	0.0005705
37	0.0000516	0.0006002
38	0.0000551	0.0006403
39	0.0000553	0.0006801
40	0.0000580	0.0007246
41	0.0000604	0.0007742
42	0.0000600	0.0008182
43	0.0000575	0.0008570
44	0.0000552	0.0008949
45	0.0000510	0.0009332
46	0.0000472	0.0009762
47	0.0000477	0.0010338
48	0.0000482	0.0011013
49	0.0000522	0.0011824
50	0.0000557	0.0012785
51	0.0000592	0.0013732
52	0.0000602	0.0014626
53	0.0000583	0.0015357
54	0.0000549	0.0016087
55	0.0000517	0.0016852
56	0.0000488	0.0017759
57	0.0000475	0.0018896
58	0.0000491	0.0020359
59	0.0000519	0.0022091
60	0.0000546	0.0024088
61	0.0000569	0.0026107
62	0.0000578	0.0028003
63	0.0000575	0.0029586
64	0.0000558	0.0031058

65	0.0000533	0.0032558
66	0.0000509	0.0034402
67	0.0000498	0.0037185
68	0.0000498	0.0041070
69	0.0000487	0.0045742
70	0.0000488	0.0050790
71	0.0000487	0.0055703
72	0.0000479	0.0060309
73	0.0000468	0.0064577
74	0.0000450	0.0068523
75	0.0000434	0.0072376
76	0.0000417	0.0076276
77	0.0000400	0.0080436
78	0.0000386	0.0084993

Source: Output from author's program SURVCH.FOR

CHAPTER IV

MORBIDITY IN AFRICA AND ITS INDIRECT ESTIMATION

4.1 Levels, Patterns and Trends in Morbidity

As noted in the last chapter, data on causes of death in Africa are scarce. Even more scarce are morbidity data. Many examining morbidity, therefore, use causes of death as proxy measures for unavailable morbidity data.

Malaria is one of the most important diseases in Africa. It is estimated that about a quarter of all adults suffer malarial fever at one time or another but the majority are infected. In Zaire, it is reported that roughly one in ten people suffer from malaria (United Nations, 1982). Generally, highland areas are less affected by malaria than low-lying areas. Hence in places like Kenya with coastal areas as well as high mountainous regions, the country can be distinctly divided into malarial and non-malarial zones. Such a dichotomy was also used by Palloni and Wyrick (1981) in their Latin American study.

Another disease which is strongly environmentally related is onchocerciasis or 'river blindness.' This is prevalent in river basins. In Ghana, largely because of the Volta dam, the number of people infected by onchocerciasis was estimated at 1 million by 1974 with about 70,000 being blind (United Nations, 1982).

Most of the estimates available for morbidity in Africa are obtained from hospital admissions. Based on hospital admission data for Kenya, 1978, Malawi, 1978, Tanzania, 1978 and Zambia, 1974, it was found that infectious, parasitic and respiratory diseases account for between one-fifth and one third of all hospital admissions (UNECA, 1985). In a study on infant and child morbidity in Lagos in 1978, the leading causes of morbidity were found to be infectious, parasitic, digestive, respiratory diseases and blood diseases (Chojnacka and Adegbola, 1984). In another study on infant and early childhood mortality in Sierra Leone, the leading causes of morbidity were acute respiratory infections, malaria and diarrhoeal diseases (Ministry of Health of Sierra Leone and WHO, 1980).

The Demographic and Health Surveys conducted in the late 1980's in several African countries provide comparable information on period prevalence of some common diseases among children of less than five years old. Prevalence here is used to mean the total number of children known to have had the disease at any time during a span of seven days to four weeks prior to the survey. Table 4.1 gives some prevalence rates for diarrhoea, fever and coughs as estimated from the Demographic and Health Surveys data. From the table, the lowest prevalence rates for diarrhoea and fever are found for Botswana while the highest rates are found for Liberia. This is consistent with what is known about their levels of mortality. However, for coughs, which has strong seasonal effects, no pattern is evident. Botswana, with one of the lowest levels of mortality in Africa, has prevalence rates far higher than that of Mali which has one of the highest mortality rates in Africa.

Table 4.1 Prevalence Rates for Certain Infectious and Parasitic Diseases for Selected African Countries.

Country	Survey Year	Prevalence rates[a] (%) for children less than five years old		
		Diarrhoea	Fever	Coughs
Egypt[1]	1988	16.0	N.A	43.0
Togo[2]	1988	29.4	43.2	10.5
Ghana[3]	1988	26.3	35.0	20.0
Kenya[4]	1988	13.0	42.0	18.0
Uganda[5]	1988	24.0	41.0	22.0
Botswana[6]	1988	10.0	3.9	28.7
Zimbabwe[7]	1988	N.A.	19.7	46.4
Liberia[8]	1986	39.1	51.2	37.1
Mali[9]	1987	34.4	33.1	6.6
Senegal[10]	1986	38.0	N.A.	N.A.

Footnotes

a- The period considered is two weeks for the case of diarrhoea (except for Egypt where it was 7 days and Liberia where it was 4 weeks) and 4 weeks in the case of Coughs and Fever (except for Zimbabwe, where it was 2 weeks for coughs)

N.A.- Not available

Sources:

1. Sayed *et al.* (1989)
2. Agounké *et al.* (1989)
3. Ghana Statistical Service (1989)
4. National Council for Population and Development (1989)
5. Kaijuka *et al.* (1989)
6. Lesetedi *et al.* (1989)
7. Central Statistics Office (1989)
8. Chieh-Johnson *et al.* (1988)
9. Traoré *et al.* (1989)
10. Diaye *et al.* (1988)

In a recent study, Diamé *et al.* (1990) published the percentage distribution of diarrhoea morbidity by age for Senegalese children of less than 60 months. This is shown in Table 4.2. The table shows that the peak ages for diarrhoea among children are between 6 months and 2 years, after which the percentages start decreasing.

The study on adult morbidity in Lagos, Nigeria done by Adegbola and Chojnacka (1990) is quite revealing. Using hospital and survey data, they found marked changes in the adult morbidity patterns over the period 1968-1978. For 1968, the leading causes of morbidity were infectious, respiratory and digestive diseases for both genders. By 1978, the leading causes of morbidity among females were infectious, respiratory, and circulatory diseases, and among males, accidents, respiratory diseases and nervous disorders. The difference in the morbidity pattern between 1968 and 1978 was attributed to the side effects of industrial development in Lagos.

For all age groups combined, Table 4.3 shows the percentage distribution of causes of morbidity among the in-patients and out-patients in Kenyan government hospitals in 1960. The leading causes of morbidity among the in-patients were infectious and parasitic diseases, respiratory diseases, injuries, pregnancy related causes and alimentary causes. With the exception of pregnancy related causes and injuries, the above-mentioned diseases are also among the leading causes of morbidity among out-patients. In a study on utilization of health services done in Tunisia, it was found that over the period 1964-1968, three major groups of diseases, namely, respiratory diseases, gastro-intestinal

Table 4.2 Age Distribution of Children with Diarrhoea in Senegal, 1986.

Age (months)	Percentage
0-5	38.5
6-11	55.5
11-17	51.2
15-23	53.9
24-29	48.2
30-35	36.9
36-41	38.7
42-47	27.4
48-53	21.3
54-59	19.1

Source: Diamé *et al.* (1990)

Table 4.3 Causes of Morbidity among in-patients and out-patients in government hospitals, Kenya, 1960.

Cause of morbidity	Percentage among in-patients	Percentage among out-patients
General infectious and parasitic	25.7	17.0
Respiratory	16.6	20.8
Injuries	12.2	9.6
Pregnancy and Puerperium	11.2	1.8'
Alimentary	8.6	15.2
Ill-defined	5.9	14.5
Skin and musculo-skeletal	5.6	10.3
Nervous system and sense organs	4.0	6.4
Genito-urinary	3.6	1.6
Allergic-metabolic, blood	3.3	
Circulatory and new growths	1.2	.31
(Diseases of) New born	.33	.01

' Includes the cause- allergic-metabolic and blood

Source: Fendall (1972)

disorders and skin diseases accounted for almost half of the illnesses reported in the dispensaries (Benyoussef and Wessen, 1974).

4.2 Indirect Estimation of Morbidity

The paucity of data on morbidity makes it even more pressing to resort to indirect estimation techniques. Whilst there is rich volume of work in this area, one finds that diverse approaches have been employed in attempting indirect estimation of morbidity. This is partly due to the many different disciplines with interests in various aspects of morbidity and/or mortality. Researchers who have worked in this direction come from various disciplines such as demography, actuarial science, population biology, mathematics, statistics, epidemiology, biostatistics and medical statistics. Invariably, their writings borrow heavily from their disciplinary methodologies. As a result, there is a lack of coherence on the works done; rather, one often sees pockets of research done where only selected works are referenced.

Based on the type of mortality or health information available, its completeness and reliability, Shigan (1977) suggested five alternatives for constructing general morbidity models and discussed their limitations. These models cover possible cases from very poor routine statistics on health (as in developing countries) to very advanced data gathering systems in some developed countries (where data on population morbidity rates exist, for example Austria and Japan). Bailey *et al.* (1975) distinguish between 'structural'

and 'functional' models. Structural models are those which attempt to portray underlying mechanism of the morbid processes, using biomedical or epidemiological approaches, while functional models are those which are based on analysis of proxy measures of morbidity, like causes of death data or hospital utilization data, in purely statistical ways, making no reference to biological processes. Kitsul (1980) classified morbidity estimation models into four types:

(a) Aggregate morbidity models

(b) Group morbidity models

(c) Specific morbidity models

(d) Stage of disease models.

The classifications used by Bailey *et al.* and by Kitsul may overlap. For example, there are specific disease models which are also functional models, just as there are specific disease models which are structural models.

One example of a specific disease model which is of a functional type was proposed by Damiani (1977). He suggested a relationship between central rates of morbidity and mortality at age x for a particular disease. The morbidity rates were defined in terms of first visits to a general practitioner for a disease. The formula included a parameter ß taken as a measure of seriousness of the disease. Pollard (1980) showed that though the formula fitted the malignant disease data well, ß could not be taken as a measure of seriousness of the disease.

The specific disease models proposed by the Italian group of researchers (Verdecchia *et al.*, 1989; Egidi *et al.*, 1991) are stochastic models which make no assumption about population structure nor about past mortality (and migration) but rather rely on the differential effects of these two phenomena on the sick and the general population. These models use mortality time series data and assume that the incidence of a disease is a logistic function of age and cohort, or age, period and cohort. They mainly use data on survivorship and mortality.

The morbidity models of degenerative diseases proposed at International Institute for Applied Systems Analysis (IIASA) include in various degrees the first three types of models as classified by Kitsul (1980) above. The models are demographic in approach and some are applicable only to lethal diseases *with no possible recovery*. In a degenerative model proposed by Kaihara *et al.* (1977), several assumptions were made: The morbidity rate being only age dependent, the duration of the sickness being only disease dependent and the population structure being stable for the duration of the given degenerative disease. If for such a population the number of sick people and the duration of sickness are estimable, then the morbidity rates can be estimated from a given set of age specific death rates and the population structure.

In the degenerative disease model of Klementiev (1977), the population was also assumed to be stable and the process irreversible. Klementiev's model was originally developed to estimate the prevalence of degenerative diseases for which direct data are

lacking. The model does not consider recovery; it assumes that once a disease has been contracted, it is just a matter of time before the victim dies of either the contracted disease or another. Thus, a number of healthy individuals may either stay healthy or contract a given disease, of which they may or may not die later (but from which they never recover). Those not contracting the given disease eventually die of other causes.

The model is stated mathematically in terms of two sets of balancing equations. One set describes the number of individuals in a given age group (or stratum) as the sum of healthy and sick individuals; the other set describes the number of deaths in each age group as the sum of individuals dying of the contracted disease and of other causes. The equations are then solved for the unknown variables, which are the incidence rates and the number of healthy individuals in a given age group.

Kitsul (1980) proposed a dynamic morbidity model for degenerative disease for a population that was unstable and unstationary. He assumed that the survival probabilities of the given disease as well as the mortality rates were known. Using a cohort approach, he then derived equations to obtain the morbidity rates and the number of sick individuals.

In the morbidity model of infectious diseases of Fujimasa *et al.* (1978), the aim was to measure prevalence rates in developed countries from three 'standard' rates; morbidity rate, recovery rate and disease-specific death rate per patient. These rates were

estimated from conventional health statistics and were assumed to be constant across developed countries. The conventional health data used for this purpose were: the disease specific death rate per population, the prevalence rate and the mean length of stay in the sick state of infectious disease. Using these standard rates, the only input required to calculate the prevalence rates is the population structure. This is an indirect estimation technique suitable designed for developed countries where, even though prevalence rates cannot be easily obtained, conventional health statistics are more readily available.

Examples of specific disease models which are also structural models are those for cancer where it is assumed that a carcinogenic agent initiates the process of changing a somatic cell into a cancer cell. While most of these models use sophisticated biological theories in their explanations, they often end up simplifying by assuming known mathematical functions or distributions. For example, the Gompertz and Weibull distributions have been assumed in modelling the initiation of cancer (Elandt-Johnson and Johnson, 1980). Of special interest is the model proposed by Burch (1966) to obtain age-specific initiation rates for lung cancer, at different initiation ages. While the model is based on a complicated biological process, it ends up with an elegant mathematical formula. The formula includes a parameter S, the proportion of the population at genetic risk to the disease in question, and yields a flexible set of curves. Upon discussing the properties of these curves, Pollard (1980) recommended their use for those interested in using mathematical formulae to fit morbidity rates.

A set of morbidity models which also deal with specific disease but are quite different from the models described above are the epidemic models. These models deal with the spread of communicable diseases through a community or household, and thus provide insight into the biological and sociological mechanisms underlying the disease-spreading process. A major function of an epidemic model is:

> 'to provide a means by which we may go from a description of the role of an infectious individual to a description of the spread of the disease through the community.' (Becker, 1979).

These epidemic models are concerned about determining the spatial spread, finding the determinants of inter-epidemic periods and evaluating vaccination campaigns. Good reviews of epidemic models have been given by Bailey (1975) and Wickwire (1977) among several others. An overview of trends in epidemic model research has been given by Becker (1979).

Recently, after the outbreak of the AIDS epidemic, researchers have constructed several models for AIDS incidence. These epidemic models form a group by themselves. Many widely different approaches, ranging from statistical to epidemio-demographic, have been used. Palloni and Glicklich (1991) classify the models into four types:

-Those focusing on the size of the epidemic and attempting to fit a suitable *a priori* function to the observed new (or cumulated) reported AIDS cases.

-Those distinguishing between new reported AIDS cases and unrecognized HIV infections.

-Those making assumptions about the incubation period and about the non-behavioural modes of transmission.

-Those macro and micro models that incorporate explicit behavioral assumptions about modes of transmission.

To cite a few examples of these different approaches, Zeger *et al.* (1989) propose a log-linear model to estimate the number of AIDS cases and trends in incidence of AIDS while correcting for reporting delays. Taylor (1989) uses the distribution of waiting times from HIV infection to AIDS incidence in a stochastic framework in order to estimate the growth of the HIV infection epidemic. Bongaarts (1989) integrates, within a demographic framework, a set of epidemiological submodels for various routes of HIV transmission in different behaviour groups. Even though this enables one to project the annual incidence and prevalence of AIDS, it does not make much use of mortality data.

Of all these models reviewed above, the ones that mostly make use of data on mortality and survivorship are those developed at IIASA in the late 1970's and by the Italian team in the late 1980's. While Damiani's model does not make use of mortality data, it uses other demographic data namely, hospital statistics data. As discussed under the review of morbidity levels, the predominant causes of morbidity in Africa are infectious and parasitic diseases, especially those of the lethal type. Therefore, the main aim of this chapter is to modify an appropriate morbidity model for estimating lethal infectious diseases for Africa. The models using long time series of mortality data such

as those by the Italian group cannot be gainfully employed. Further, we do not want to make any assumptions about the underlying physiological processes; this rules out the use of biologically-based models. The infectious disease model of Fujimasa *et al.* (1978) is not appropriate for a developing society because it was constructed for developed countries where almost all patients visit hospitals and where the countries are fairly homogeneous with respect to health levels. In Africa, hospitals only treat a fraction of the sick individuals and heterogeneity in health is very high even among population subgroups within a given country. Thus, the only useful model one is left with is that of Klementiev (1977) which will be modified below for its application to African data.

4.2.1 Klementiev's model modified

Klementiev's original model was for irreversible degenerative disease. Generally, such models have been found to increase in accuracy when the survivorship for the specific disease is low. For example, the survival following lung cancer diagnosis is so low that incidence and mortality patterns are in such a close relationship that mortality trend can reasonably be used as a proxy for incidence (Myers *et al.*, 1982). For certain lethal infectious diseases, survival may be very low when there is no vaccination or adequate medical care or proper diagnosis. In such a situation, patients may either die or remain terminally ill. If proper health measures are available, the patients may be cured; in which context, a model of infectious diseases cannot be described by a degenerative disease model such as Klementiev's. Rather, it may have to incorporate certain elements

of the degenerative disease model. This proposition has been outlined by Rajulton and Bah (1991) and is shown below.

Following the formulation of Klementiev, the relevant variables for a population divided into N age groups are defined below (with some modifications):

Variables	Description	Status
p_i	the number of individuals in the *i-th* age group	Known
h_i	the number of healthy individuals	Unknown
μ_i	the incidence rate (at which people contract the infectious disease), per 100,000 healthy individuals from the *i-th.* age group	Unknown
σ_i	the rate of recovery and is defined as the number of individuals who recovered per 100,000 sick individuals who contracted the disease in the *i-th* age group during the year (this assumes that recovery, if any, should occur *within a year*)	Assumed
α_i	the death rate from *all causes*, per 100,000 individuals	Available
τ_i	the death rate according to the infectious cause (given disease), per 100,000	Available
Θ_i	the death rate for other causes per 100,000; that is, $(\Theta_i = \alpha_i - \tau_i)$	Available

d_{ij}	the disease-specific death rate, defined as the number of deaths per 100,000 sick individuals who contracted the disease in the *i-th* age group *j* years ago.	Assumed

We have assumed in the above formulations that once individuals contract a given disease, it becomes their cause of death. They may, however, recover from the disease but the recovery should be possible within a year. Hence within the period of one year ($j = 1$) the rate of elimination β_{ij} of sick individuals is either through death or through recovery; this is given by $\beta_{i1} = \sigma_i + d_{i1}$. Those who recover will eventually die of some other cause sooner or later. For period beyond one year ($j > 1$), the rate of elimination β_{ij} of sick individuals is only through death from the given disease, $\beta_{ij} = d_{ij}$. This assumption is justified because it closely approximates the case of several lethal infectious diseases like cholera, malaria and to some extent tuberculosis, for which modern medicine has found a cure. This is partly validated in an observation made by the United Nations (1982:111):

> 'In Africa, about a quarter of all adults suffer malarial fever at one time or another but virtually all are infected. The majority develop a relative immunity. After infancy almost every child in tropical Africa has malaria and at least 1 million children die of the disease.'

The flow diagram for the disease prevalence is presented in Figure 4.1. The diagram shows that the number of healthy persons in the first age group is h_1. During one year, $\mu_1 h_1$ healthy people contract the disease. Of these people, $\mu_1 h_1 d_{11}$ die of the disease, $\mu_1 h_1 \sigma_1$ recover, and the rest $\mu_1 h_1 (1-\beta_{11})$ remain alive with the disease. For each age group,

the balance equations describing the number of individuals equals the sum of the healthy and sick individuals and those who recover from the illness. These equations can written as follows:

$$p_1 = h_1 \tag{14}$$

$$p_2 = (h_2 + \mu_1 h_1 \sigma_1) + \mu_1 h_1 (1 - \beta_{11}) \tag{15}$$

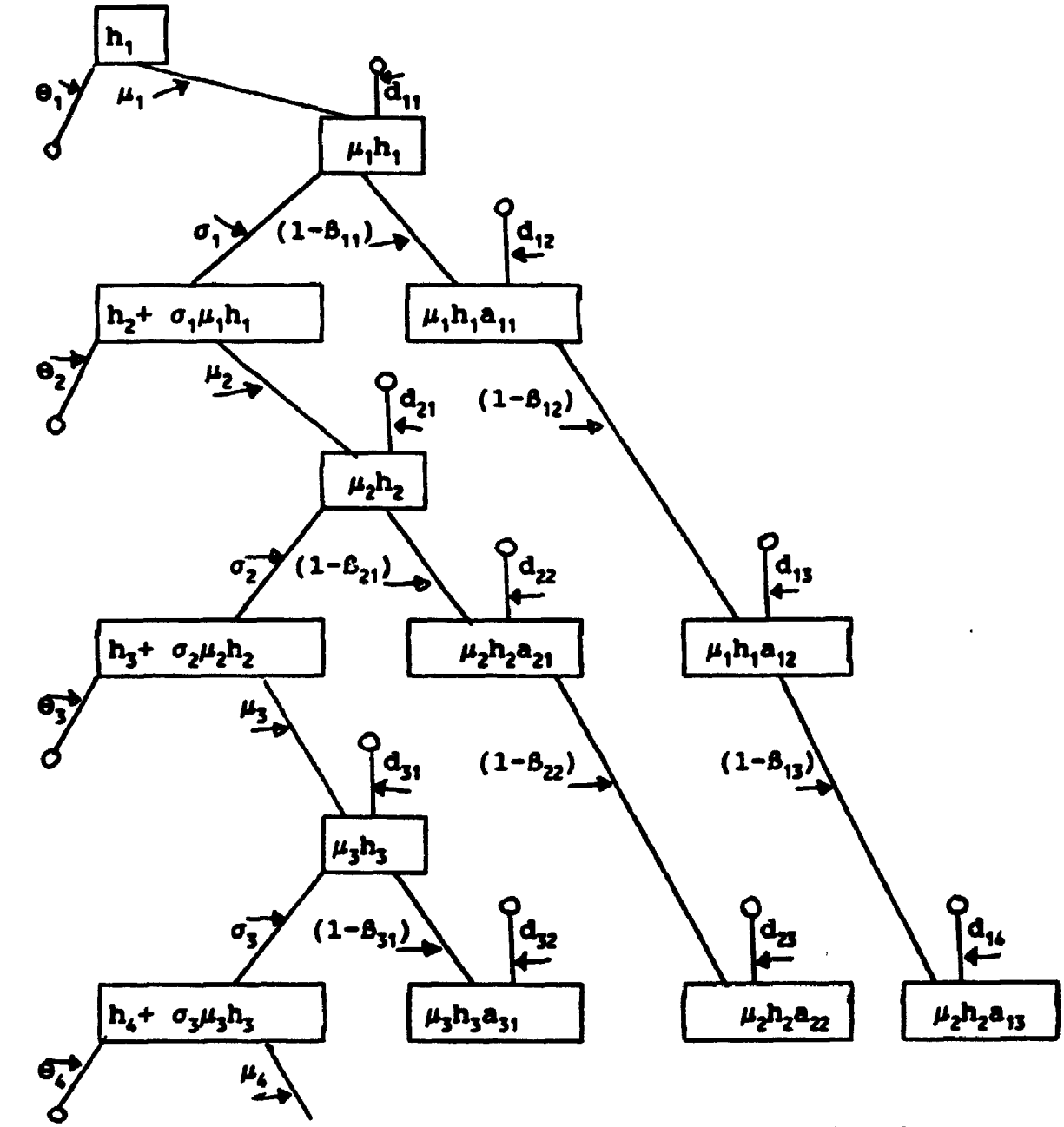
$$p_3 = (h_3 + \mu_2 h_2 \sigma_2) + \mu_2 h_2 (1 - \beta_{21}) + \mu_1 h_1 (1 - \beta_{11})(1 - \beta_{12}) \tag{16}$$

$$p_4 = (h_4 + \mu_3 h_3 \sigma_3) + \mu_3 h_3 (1 - \beta_{31}) + \mu_2 h_2 (1 - \beta_{21})(1 - \beta_{22}) + \mu_1 h_1 (1 - \beta_{11})(1 - \beta_{12})(1 - \beta_{13}) \tag{17}$$

. . . . . .

$$p_i = (h_i + \mu_{i-1} h_{i-1} \sigma_{i-1}) + \sum_{j=1}^{i-1} \mu_{i-j} h_{i-j} a_{i-j,j} , \quad i = 2, \ldots N \tag{18}$$

Figure 4.1 **Flow diagram for the estimation of prevalence for lethal infectious diseases.**



(After Klementiev (1977)).

$$p_i = (h_i + \mu_{i-1}h_{i-1}\sigma_{i-1}) + \mu_{i-1}h_{i-1}a_{i-1,1} + \sum_{j=2}^{i-1} \mu_{i-j}h_{i-j}a_{i-j,j} \quad (19)$$

$$=(h_i + \mu_{i-1}h_{i-1}\sigma_{i-1}) + \mu_{i-1}h_{i-1}a_{i-1,1} + F_i \quad (20)$$

where

$$a_{ij} = \prod_{k=1}^{j} (1 - \beta_{ik}) , \quad i = 1,...N-1 ; j = 1,...N-1 \quad (21)$$

The balance equations for the number of deaths in each age group can be written as follows:

$$p_1\alpha_1 = h_1\theta_1 \quad (22)$$

$$p_2\alpha_2 = (h_2 + \mu_1h_1\sigma_1)\theta_2 + \mu_1h_1d_{11} \quad (23)$$

$$p_3\alpha_3 = (h_3 + \mu_2h_2\sigma_2)\theta_3 + \mu_2h_2d_{21} + \mu_1h_1(1 - \beta_{11})d_{12} \quad (24)$$

$$p_4\alpha_4 = (h_4 + \mu_3h_3\sigma_3)\theta_4 + \mu_3h_3d_{31} + \mu_2h_2(1 - \beta_{21})d_{22} + \mu_1h_1(1 - \beta_{11})(1 - \beta_{12})d_{13} \quad (25)$$

. . . . . .

$$p_i\alpha_i = (h_i + \mu_{i-1}h_{i-1}\sigma_{i-1})\theta_i + \mu_{i-1}h_{i-1}d_{i-1,1} + \sum_{j=2}^{i-1} \mu_{i-j}h_{i-j}a_{i-j,j-1}d_{i-j,j} \quad (26)$$
$$i = 3,...N$$

$$= (h_i + \mu_{i-1}h_{i-1}\sigma_{i-1})\theta_i + \mu_{i-1}h_{i-1}d_{i-1,1} + G_i \quad (27)$$

The equations (14) to (27) can be solved for the unknown variables μ_i and h_i. For example, for the first age group, substituting Equation (14) in equations (15) and (21) and eliminating the expression $(h_2 + \mu_1 h_1 \sigma_1)$, one obtains the following:

$$\mu_1 = \frac{p_2 \tau_2}{p_1(d_{11} - a_{11}\theta_2)} \tag{28}$$

and

$$h_2 = p_2 \cdot \frac{(d_{11} - \sigma_1 \tau_2 - \alpha_2 a_{11})}{(d_{11} - a_{11}\theta_2)} \tag{29}$$

In order to solve for the unknown variables of higher age groups, in a manner similar to that of Klementiev, we introduce the variable U_i defined as:

$$U_i = G_i - F_i \theta_i \tag{30}$$

Where F_i, as in Equation (20), refers to the last term in Equation (19) and G_i, as in Equation (27), refers to the last term in Equation (26). From equations (18), (27) and (30) we obtain that:

$$\mu_{i-1} = \frac{p_i \tau_i - U_i}{h_{i-1}(d_{i-1,1} - a_{i-1,1}\theta_i)}, \quad i = 3,...N \tag{31}$$

and

$$h_i = \frac{p_i(d_{i-1,1} - a_{i-1,1}\alpha_i - \tau_i \sigma_{i-1}) + (\sigma_{i-1} - a_{i-1,1})G_i - (\theta_i \sigma_{i-1} + d_{i-1})F_i}{d_{i-1,1} - a_{i-1,1}\theta_i}, \tag{32}$$

$$i = 3,...N$$

The full derivation of the unknown variables is given in Rajulton and Bah (1991) and

reproduced in the Appendix VII The equations for the unknowns contain three sets of variables; the population variable p_i; the mortality variables, τ_i, α_i and Θ_i and the mortality-morbidity variables, a_{ij} and d_{ij}. The first two sets of variables are easily obtainable. The last set of variables should be obtainable from epidemiologic sources.

4.2.2 Application of the model

A computer program has been prepared to apply the model outlined above. A restriction in the model is that τ_i must be large if $d_{i-1,1}$ is large and must be small if τ_i is small, otherwise improbable results will be obtained. On the application of the model, it has also been necessary to make some assumptions to obtain the distribution of d_{ij}. First, the duration of the illness has been restricted to 5 years ($j \leq 5$). Given initial value. of d_{ij} (j=1), they are multiplied by a constant c_1 to obtain values for $d_{i-1,j+1}$. These values in turn are multiplied by another constant c_2 to obtain values for $d_{i-2,j+2}$. Viewed at one angle, these values of d_{ij} depict the mortality experience of a cohort with respect to a particular disease. For example, $d_{1,3}$, $d_{2,2}$, and $d_{3,1}$ are disease-specific death rates for the population subgroup in age group one, three years (or periods) ago, followed by those in the next age group one year later and finally those in age group three, one year ago. These values form cohort disease-specific death rates. If the values of c_j lie between zero and one, the values of d_{ij} obtained show increase with age of the cohort members. In other words, the highest disease specific death rate for a cohort occur for those who have been ill since a year ago. The disease specific death rate for those ill since two years ago would be less than for

those ill since a year ago. The longer the duration, the less the chance of dying of the disease. If c_j is greater than one, the longer the duration, the higher the chance of dying of the disease.

In this subsection, the behaviour of the model is studied with regard to changes in its various parameters. The age groups are initially restricted to 10. The data in the input data base consist of p_i, τ_i, α_i,σ_i,d_{i1} and c_j. While effort was made to make the values close to different published results, they do not all reflect data from a given country. In the first run of the program, the values of τ_i and d_{i1} were all fixed at .00016 and .0610 respectively. To obtain the values of d_{ij}, the constant c_j was fixed at 0.11. This low value of c_j means that the chance of dying for duration of illness greater than one year is very low. Using these input values, the incidence rates obtained are given in Table 4.4. These values range from .004 to .011. As these values are low, the healthy population h_i has correspondingly been very high.

In the second run of the model, all the previous input values were maintained except the cause specific death rates τ_i which were increased by a factor of 10. The results obtained are shown in Table 4.5 from which it can be seen that the incidence rates showed almost a ten fold increase. With this increases in incidence rates, the healthy population h_i was reduced in varying proportions.

In run 3, the data used in run 2 were retained except that the constant c_j was now increased to 0.2. An increase in c_j means that the duration of the illness is becoming significant. The result of this change on the incidence rates is a slight reduction (to the third decimal place). These results are shown in Table 4.6.

In run 4, the values of c_j used in run 3 were again increased to 0.4 while keeping all other values constant. The effect of this was a further decline in the incidence rates. These decline were greater than the previous declines observed when changing from run 2 to run 3. These results are presented in Table 4.7.

In run 5, the disease-specific death rates d_{ij} were increased from .016 to .2, keeping all other values unchanged. The results shown in Table 4.8 show marked reduction in the incidence rates of more than 50%.

The next part of the investigation is to study the diseases for which the chances of dying increase with the duration of the disease, i.e. those for which $c_j > 1$. In run 6, the same data in run 5 were used except that c_j was increased to 1.02. The results shown in Table 4.9 show slight reduction (to the third decimal) in the incidence rates.

In run 7, with results shown in Table 4.10, the c_j values used in run 6 were increased to 1.4 with all other values retained. The results show that the low limit of the incidence rates might have been reached as the reductions observed were very slight.

In run 8, the d_{11} values were reduced to .18 while the c_j and the other values were kept constant. The results shown in table 4.11 show hardly any change in the incidence rates.

As was done earlier with c_j <1, the cause-specific death rates were again changed to study their effects on the incidence rates. In run 9, the cause-specific death rates τ_i were increased by a factor of 10 while all the other input values were maintained constant. The results shown in Table 4.11 show marked increase in the incidence rates for all age groups. Correspondingly, the healthy population show marked decrease in size. This shows the importance in the cause-specific death rates in determining incidence.

With the cause-specific death rates in run 9 still maintained, the recovery rates were altered to study their effects on the incidence rates. In run 10, the recovery rates were increased by a factor of 10 and the results are shown in Table 4.12. This change had an increasing effect on the incidence rates but the amount is slight.

While the different runs above did not exhaust all the possible combinations of parameter changes, they brought out certain important features. First is the importance of the cause-specific death rate τ_i in determining the incidence rates. This was demonstrated in runs 2 and again in run 9. Closely related to τ_i is the disease-specific death rate d_{ij}. These rates are also influential in determining incidence rates as was demonstrated in run 5. Recovery rates were shown to have little effect on the incidence rates. Also there is

little effect on incidence rates whether or not the duration of illness affects the severity of the illness (i.e. affecting the death rate).

The task in the next chapter is to try to link together the submodels developed in chapters 3 and 4 in order to study the dynamics of morbidity and mortality.

Table 4.4 Output of MORBID.FOR for Prevalence Using Input Data from MORBID4.DAT and DKS4.DAT.

INPUT DATA FOR AGE (I), CAUSE SPECIFIC DEATH RATE (τ_i)
DEATH RATE FOR ALL CAUSES (α_i), AND RECOVERY RATE (σ_i)

i	p_i	τ_i	α_i	σ_i
1	136402.	0.00016	0.07600	0.00100
2	526237.	0.00016	0.00654	0.00450
3	616775.	0.00016	0.00020	0.00160
4	516626.	0.00016	0.00183	0.00027
5	410929.	0.00016	0.00022	0.00520
6	362716.	0.00016	0.00028	0.01150
7	279915.	0.00016	0.00355	0.01700
8	205918.	0.00016	0.00453	0.01800
9	169477.	0.00016	0.00590	0.01900
10	138961.	0.00016	0.00758	0.00194

INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT
D(I,J)=C(J)*D(I+1,J-1)

C(1)	C(2)	C(3)	C(4)
0.11000	0.11000	0.11000	0.11000

D(I,1)	D(I,2)	D(I,3)	D(I,4)	D(I,5)
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000081
.061000	.006710	.000738	.000738	.000738
.061000	.006710	.006710	.006710	.006710

OUTPUT FROM MORBID.FOR USING DATA FROM MORBID4.DAT

AGE	POPULATION	HEALHY POPULATION	INCIDENCE RATE
I	p_i	h_i	μ_i
1	136402.000	136402.000	0.011
2	526237.000	524799.900	0.003
3	616775.000	613682.300	0.002
4	516626.000	512304.000	0.002
5	410929.000	405779.700	0.002
6	362716.000	356849.700	0.003
7	279915.000	274639.000	0.003
8	205918.000	201298.600	0.004
9	169477.000	165436.600	0.004

Table 4.5 Output of MORBID.FOR Prevalence Using Input Data from MORBID5.DAT and DKS5.DAT.

INPUT DATA FOR AGE (i), CAUSE SPECIFIC DEATH RATE (τ_i)
DEATH RATE FOR ALL CAUSES (α_i), AND RECOVERY RATE (σ_i)

i	p_i	τ_i	α_i	σ_i
1	136402.	0.00160	0.07600	0.00100
2	526237.	0.00160	0.00654	0.00450
3	616775.	0.00160	0.00020	0.00160
4	516626.	0.00160	0.00183	0.00027
5	410929.	0.00160	0.00022	0.00520
6	362716.	0.00160	0.00028	0.01150
7	279915.	0.00160	0.00355	0.01700
8	205918.	0.00160	0.00453	0.01800
9	169477.	0.00160	0.00590	0.01900
10	138961.	0.00160	0.00758	0.00194

INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT
D(I,J)=C(J)*D(I+1,J-1)

C(1)	C(2)	C(3)	C(4)
0.11000	0.11000	0.11000	0.11000

D(I,1)	D(I,2)	D(I,3)	D(I,4)	D(I,5)
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000009
.061000	.006710	.000738	.000081	.000081
.061000	.006710	.000738	.000738	.000738
.061000	.006710	.006710	.006710	.006710

OUTPUT FROM MORBID.FOR USING DATA FROM MORBID5.DAT (τ_i MULTIPLIED BY 10)

AGE	POPULATION	HEALHY POPULATION	INCIDENCE RATE
I	p_i	h_i	μ_i
1	136402.000	136402.000	0.110
2	526237.000	512210.600	0.027
3	616775.000	586884.600	0.021
4	516626.000	475330.000	0.018
5	410929.000	362330.500	0.020
6	362716.000	308021.200	0.026
7	279915.000	231945.200	0.027
8	205918.000	165027.600	0.037
9	169477.000	134587.300	0.045

Table 4.6 Output of MORBID.FOR for Prevalence Using Input Data from MORBID6.DAT and DRS6.DAT.

INPUT DATA FOR AGE (i), CAUSE SPECIFIC DEATH RATE (τ_i)
DEATH RATE FOR ALL CAUSES (α_i), AND RECOVERY RATE (σ_i)

i	p_i	τ_i	α_i	σ_i
1	136402.	0.00160	0.07600	0.00100
2	526237.	0.00160	0.00654	0.00450
3	616775.	0.00160	0.00020	0.00160
4	516626.	0.00160	0.00183	0.00027
5	410929.	0.00160	0.00022	0.00520
6	362716.	0.00160	0.00028	0.01150
7	279915.	0.00160	0.00355	0.01700
8	205918.	0.00160	0.00453	0.01800
9	169477.	0.00160	0.00590	0.01900
10	138961.	0.00160	0.00758	0.00194

INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT
D(I,J)=C(J)*D(I+1,J-1)

C(1)	C(2)	C(3)	C(4)
0.20000	0.20000	0.20000	0.20000

D(I,1)	D(I,2)	D(I,3)	D(I,4)	D(I,5)
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000488
.061000	.012200	.002440	.002440	.002440
.061000	.012200	.012200	.012200	.012200

OUTPUT FROM MORBID.FOR USING DATA FROM MORBID6.DAT
(C(J) VALUES INCREASED FROM .11 TO .2)

AGE	POPULATION	HEALHY POPULATION	INCIDENCE RATE
I	p_i	h_i	μ_i
1	136402.000	136402.000	0.110
2	526237.000	512210.600	0.025
3	616775.000	585833.400	0.018
4	516626.000	475385.600	0.015
5	410929.000	363814.300	0.018
6	362716.000	310847.600	0.023
7	279915.000	235709.000	0.023
8	205918.000	168512.700	0.031
9	169477.000	137847.800	0.037

Table 4.7 Output of MORBID.FOR for Prevalence Using Input Data from MORBID7.DAT and DKS7.DAT.

INPUT DATA FOR AGE (i), CAUSE SPECIFIC DEATH RATE (τ_i)
DEATH RATE FOR ALL CAUSES (α_i), AND RECOVERY RATE (σ_i)

i	p_i	τ_i	α_i	σ_i
1	136402.	0.00160	0.07600	0.00100
2	526237.	0.00160	0.00654	0.00450
3	616775.	0.00160	0.00020	0.00160
4	516626.	0.00160	0.00183	0.00027
5	410929.	0.00160	0.00022	0.00520
6	362716.	0.00160	0.00028	0.01150
7	279915.	0.00160	0.00355	0.01700
8	205918.	0.00160	0.00453	0.01800
9	169477.	0.00160	0.00590	0.01900
10	138961.	0.00160	0.00758	0.00194

INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT
D(I,J)=C(J)*D(I+1,J-1)

C(1)	C(2)	C(3)	C(4)
0.40000	0.40000	0.40000	0.40000

D(I,1)	D(I,2)	D(I,3)	D(I,4)	D(I,5)
.061000	.024400	.009760	.003904	.001562
.061000	.024400	.009760	.003904	.001562
.061000	.024400	.009760	.003904	.001562
.061000	.024400	.009760	.003904	.001562
.061000	.024400	.009760	.003904	.001562
.061000	.024400	.009760	.003904	.001562
.061000	.024400	.009760	.003904	.003904
.061000	.024400	.009760	.009760	.009760
.061000	.024400	.024400	.024400	.024400

OUTPUT FROM MORBID.FOR USING DATA FROM MORBID7.DAT
(C(J) VALUES CHANGED FROM .2 TO .4)

AGE	POPULATION	HEALHY POPULATION	INCIDENCE RATE
I	p_i	h_i	μ_i
1	136402.000	136402.000	0.110
2	526237.000	512210.600	0.020
3	616775.000	583542.800	0.013
4	516626.000	475439.700	0.010
5	410929.000	367079.600	0.013
6	362716.000	317071.100	0.016
7	279915.000	244104.100	0.014
8	205918.000	176389.700	0.019
9	169477.000	145054.400	0.023

Table 4.8 Output of MORBID.FOR for Prevalence Using Input Data from MORBID8.DAT and DKS8.DAT.

INPUT DATA FOR AGE (i), CAUSE SPECIFIC DEATH RATE (τ_i)
DEATH RATE FOR ALL CAUSES (α_i), AND RECOVERY RATE (σ_i)

i	p_i	τ_i	α_i	σ_i
1	136402.	0.00160	0.07600	0.00100
2	526237.	0.00160	0.00654	0.00450
3	616775.	0.00160	0.00020	0.00160
4	516626.	0.00160	0.00183	0.00027
5	410929.	0.00160	0.00022	0.00520
6	362716.	0.00160	0.00028	0.01150
7	279915.	0.00160	0.00355	0.01700
8	205918.	0.00160	0.00453	0.01800
9	169477.	0.00160	0.00590	0.01900
10	138961.	0.00160	0.00758	0.00194

INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT
D(I,J)=C(J)*D(I+1,J-1)

C(1)	C(2)	C(3)	C(4)
1.02000	1.02000	1.02000	1.02000

D(I,1)	D(I,2)	D(I,3)	D(I,4)	D(I,5)
.200000	.204000	.208080	.212242	.216486
.200000	.204000	.208080	.212242	.216486
.200000	.204000	.208080	.212242	.216486
.200000	.204000	.208080	.212242	.216486
.200000	.204000	.208080	.212242	.216486
.200000	.204000	.208080	.212242	.216486
.200000	.204000	.208080	.212242	.212242
.200000	.204000	.208080	.208080	.208080
.200000	.204000	.204000	.204000	.204000

OUTPUT FROM MORBID.FOR USING DATA FROM MORBID8.DAT
(C(J) VALUES INCREASED AND ALSO D(I,1) VALUES)

AGE	POPULATION	HEALHY POPULATION	INCIDENCE RATE
I	p_i	h_i	μ_i
1	136402.000	136402.000	0.031
2	526237.000	522801.300	0.004
3	616775.000	607941.800	0.001
4	516626.000	506924.200	0.000
5	410929.000	402650.800	0.001
6	362716.000	355921.800	0.003
7	279915.000	276192.800	0.002
8	205918.000	202730.500	0.001
9	169477.000	166435.700	0.000

Table 4.9 Output of MORBID.FOR for Prevalence Using Input Data from MORBID9.DAT and DKS9.DAT.

INPUT DATA FOR AGE (i), CAUSE SPECIFIC DEATH RATE (τ_i)
DEATH RATE FOR ALL CAUSES (α_i), AND RECOVERY RATE (σ_i)

i	p_i	τ_i	α_i	σ_i
1	136402.	0.00160	0.07600	0.00100
2	526237.	0.00160	0.00654	0.00450
3	616775.	0.00160	0.00020	0.00160
4	516626.	0.00160	0.00183	0.00027
5	410929.	0.00160	0.00022	0.00520
6	362716.	0.00160	0.00028	0.01150
7	279915.	0.00160	0.00355	0.01700
8	205918.	0.00160	0.00453	0.01800
9	169477.	0.00160	0.00590	0.01900
10	138961.	0.00160	0.00758	0.00194

INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT
D(I,J)=C(J)*D(I+1,J-1)

D(I,1)	D(I,2)	D(I,3)	D(I,4)	D(I,5)
1.40000	1.40000	1.40000	1.40000	
.200000	.280000	.392000	.548800	.768320
.200000	.280000	.392000	.548800	.768320
.200000	.280000	.392000	.548800	.768320
.200000	280000	.392000	.548800	.768320
.200000	.280000	.392000	.548800	.768320
.200000	.280000	.392000	.548800	.768320
.200000	.280000	.392000	.548800	.548800
.200000	.280000	.392000	.392000	.392000
.200000	.280000	.280000	.280000	.280000

OUTPUT FROM MORBID.FOR USING DATA FROM MORBID9.DAT
(C(J) INCREASED TO 1.4)

AGE	POPULATION	HEALHY POPULATION	INCIDENCE RATE
I	p_i	h_i	μ_i
1	136402.000	136402.000	0.031
2	526237.000	522801.300	0.003
3	616775.000	607671.800	0.000
4	516626.000	507706.500	0.001
5	410929.000	404836.800	0.003
6	362716.000	358546.000	0.002
7	279915.000	275938.200	0.000
8	205918.000	202379.900	0.001
9	169477.000	167017.400	0.003

Table 4.10 Output of MORBID.FOR for Prevalence Using Input Data from MORBID10.DAT and DKS10.DAT.

INPUT DATA FOR AGE (i), CAUSE SPECIFIC DEATH RATE (τ_i)
DEATH RATE FOR ALL CAUSES (α_i), AND RECOVERY RATE (σ_i)

i	p_i	τ_i	α_i	σ_i
1	136402.	0.00160	0.07600	0.00100
2	526237.	0.00160	0.00654	0.00450
3	616775.	0.00160	0.00020	0.00160
4	516626.	0.00160	0.00183	0.00027
5	410929.	0.00160	0.00022	0.00520
6	362716.	0.00160	0.00028	0.01150
7	279915.	0.00160	0.00355	0.01700
8	205918.	0.00160	0.00453	0.01800
9	169477.	0.00160	0.00590	0.01900
10	138961.	0.00160	0.00758	0.00194

INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT
D(I,J)=C(J)*D(I+1,J-1)

C(1)	C(2)	C(3)	C(4)
1.40000	1.40000	1.40000	1.40000

D(I,1)	D(I,2)	D(I,3)	D(I,4)	D(I,5)
.180000	.252000	.352800	.493920	.691488
.180000	.252000	.352800	.493920	.691488
.180000	.252000	.352800	.493920	.691488
.180000	.252000	.352800	.493920	.691488
.180000	.252000	.352800	.493920	.691488
.180000	.252000	.352800	.493920	.691488
.180000	.252000	.352800	.493920	.493920
.180000	.252000	.352800	.352800	.352800
.180000	.252000	.252000	.252000	.252000

OUTPUT FROM MORBID.FOR USING DATA FROM MORBID10.DAT
(C(J) VALUES INCREASED TO 1.4 AND D(I,1) VALUES REDUCED)

AGE	POPULATION	HEALHY POPULATION	INCIDENCE RATE
I	p_i	h_i	μ_i
1	136402.000	136402.000	0.035
2	526237.000	522313.200	0.003
3	616775.000	606087.400	0.000
4	516626.000	506153.500	0.000
5	410929.000	403832.800	0.003
6	362716.000	358036.000	0.003
7	279915.000	275697.500	0.000
8	205918.000	201641.000	0.000
9	169477.000	166410.700	0.003

Table 4.11 Output of MORBID.FOR for Prevalence Using Input Data from MORBID11.DAT and DKS11.DAT.

INPUT DATA FOR AGE (i), CAUSE SPECIFIC DEATH RATE (τ_i)
DEATH RATE FOR ALL CAUSES (α_i), AND RECOVERY RATE (σ_i)

i	p_i	τ_i	α_i	σ_i
1	136402.	0.01600	0.07600	0.00100
2	526237.	0.01600	0.00654	0.00450
3	616775.	0.01600	0.00020	0.00160
4	516626.	0.01600	0.00183	0.00027
5	410929.	0.01600	0.00022	0.00520
6	362716.	0.01600	0.00028	0.01150
7	279915.	0.01600	0.00355	0.01700
8	205918.	0.01600	0.00453	0.01800
9	169477.	0.01600	0.00590	0.01900
10	138961.	0.01600	0.00758	0.00194

INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT
D(I,J)=C(J)*D(I+1,J-1)

C(1)	C(2)	C(3)	C(4)
0.20000	0.20000	0.20000	0.20000

D(I,1)	D(I,2)	D(I,3)	D(I,4)	D(I,5)
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000488
.061000	.012200	.002440	.002440	.002440
.061000	.012200	.012200	.012200	.012200

OUTPUT FROM MORBID.FOR USING DATA FROM MORBID11.DAT
(tau i increased by a factor of 10)

AGE	POPULATION	HEALHY POPULATION	INCIDENCE RATE
I	p_i	h_i	μ_i
1	136402.000	136402.000	0.883
2	526237.000	413087.000	0.215
3	616775.000	387957.400	0.147
4	516626.000	237224.400	0.100
5	410929.000	120209.600	0.126
6	362716.000	69527.990	0.416
7	279915.000	75665.840	0.296
8	205918.000	60230.770	0.367
9	169477.000	57703.260	0.311

Table 4.12 Output of MORBID.FOR for Prevalence Using Input Data from MORBID12.DAT and DKS12.DAT.

INPUT DATA FOR AGE (i), CAUSE SPECIFIC DEATH RATE (τ_i)
DEATH RATE FOR ALL CAUSES (α_i), AND RECOVERY RATE (σ_i)

i	p_i	τ_i	α_i	σ_i
1	136402.	0.01600	0.07600	0.01000
2	526237.	0.01600	0.00654	0.04500
3	616775.	0.01600	0.00020	0.01600
4	516626.	0.01600	0.00183	0.00270
5	410929.	0.01600	0.00022	0.05200
6	362716.	0.01600	0.00028	0.11500
7	279915.	0.01600	0.00355	0.17000
8	205918.	0.01600	0.00453	0.18000
9	169477.	0.01600	0.00590	0.19000
10	138961.	0.01600	0.00758	0.01940

INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT
D(I,J)=C(J)*D(I+1,J-1)

C(1)	C(2)	C(3)	C(4)
0.20000	0.20000	0.20000	0.20000

D(I,1)	D(I,2)	D(I,3)	D(I,4)	D(I,5)
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000098
.061000	.012200	.002440	.000488	.000488
.061000	.012200	.002440	.002440	.002440
.061000	.012200	.012200	.012200	.012200

OUTPUT FROM MORBID.FOR USING DATA FROM MORBID12.DAT
(σ_i INCREASED BY A FACTOR OF 10)

AGE	POPULATION	HEALHY POPULATION	INCIDENCE RATE
I	p_i	h_i	μ_i
1	136402.000	136402.000	0.884
2	526237.000	412949.000	0.218
3	616775.000	389308.800	0.150
4	516626.000	240781.900	0.101
5	410929.000	122809.000	0.128
6	362716.000	71819.790	0.415
7	279915.000	77320.270	0.305
8	205918.000	62599.590	0.380
9	169477.000	62546.370	0.316

CHAPTER V

THE DYNAMICS OF MORBIDITY AND MORTALITY

5.1 Introduction

In the previous chapter, the primary focus was on the estimation of morbidity. In some of the models reviewed in that chapter, mortality and cause of death data were used in the estimation process. The concern in this chapter is not the estimation of morbidity but rather to study the effect of morbidity on mortality or vice-versa or their joint effect on certain demographic parameters. The actual study is preceded by review of models which relate to the aims of the chapter.

5.2 Review of Models

Without attempting at any rigid classification, some of the different types of research done on morbidity and mortality dynamics over the last three or four decades will be briefly reviewed.

Fix and Neyman (1951) proposed a stochastic model of morbidity and mortality. They proposed a Markov model for follow-up study comprising of four states:

S_0 - under treatment for a disease

S_1 - died after treatment

S_2 - apparently recovered from the disease, not under treatment but still under observation

S_3 - lost from S_2, either by death or difficulty of being traced.

Using this model they derived formulae for measures such as net risk of transfer from one state to another with the risk of a specified interstate transfer eliminated and also the expected length of normal life within a specified period following a given treatment.

Chiang and Hsu (1976) later reexamined the Fix-Neyman model for a stochastic system with two transient states, the state of health and the state of illness, and a number of absorbing states (different causes of death). In their work, they considered the number of transitions between the two transient states and the length of time interval required for these transitions. They then proceeded to derive formulae for the density functions and the distribution functions of the multiple transition times.

Lagakos (1976) used a stochastic model to relate disease progression to mortality. He considered three states; 'alive without progressive disease,' 'alive but having previously experienced progressive disease' and 'dead.' Using this model, he was able to study the effect of disease progression on survival.

Further work on disease progression (or stages of disease) and mortality was done by Chiang (1979, 1980, 1984). He first proposed a stochastic model to describe survival and the stage of a disease. In that model, the stage of a disease plays a dominating role in the survival or death of a patient. The density function, distribution function and the maximum likelihood estimators of the parameters involved were derived. In a later work, the same problem was reworked using life table methodology. From the life table

constructed, (life table for survival and stages of disease) he derived several biometric functions such as the 'probability of advancement from stage i to stage i+1,' 'waiting time in stage i,' 'life time in stage i' etc. A serious shortcoming of this model is the failure to take into account the age variable; this limits its demographic application.

Another line of research was on the dynamics of, for want of better expression, the 'physical manifestation of disease progression' and mortality. This aspect of disease progression is generally described as 'health status', and work in this area is often categorized as 'health status indices' research. To this research area belong the indices of health expectancy. The pioneering article of health expectancy indices, and perhaps the most widely quoted, is that of Sullivan (1971). He proposed a single index of morbidity and mortality based on a life table model. His technique employed a relatively simple modification of the conventional life table model in which the age specific disability rates were applied to the person-years of life lived between two ages. From this, he subsequently obtained the expectation of disability and the expectation of life free of disability. The technique is similar to the methods used to compute expected values for conditions such as labour force participation and school enrolment.

A similar technique has been employed by Katz *et al.* (1983), Wilkins and Adams (1983a, 1983b), Crimmins *et al.* (1989). They differ mainly in the disability states they employ. Sullivan (1971) restricted his definition to bed-disability which covers both institutional and noninstitutional populations. Wilkins and Adams (1983b) used six states

of disability ranging from 'activities not restricted' to 'major activity impossible' and 'long-term institutionalization.' Crimmins *et al.* (1989) employed a classification closely similar to that of Wilkins and Adams in one part of their analysis and one similar to Sullivan's in another part.

Although using a similar methodology, Katz *et al.* (1983) actually restricted themselves to one aspect of health status namely 'active life expectancy,' which was defined as the expected duration of functional wellbeing and which signified a person's independence in the activities of daily living (ADL). Their methodology was commended for its simplicity and advantage of being related directly to available data. Moreover, it paved the way for using life table techniques to link mortality to morbidity in a manner different from Chiang's. However, if one wants to measure the health status of a population rather than some aspects of health, then the methodology suggested by Katz *et al.* can be criticised on two grounds: First, the different authors consider mainly disability. It is evident that disability does not represent the whole spectrum of health. Having a chronic disease or condition does not necessarily mean that a person's daily life is limited or that a person is disabled by that condition (American Council of Life Insurance, 1986). It has also been noted by Manton (1986a, 1986b) that the chronic conditions that produce the largest number of deaths generate only small amounts of the total chronic disability. Wilkins and Adams (1983a, 1983b) observe that the leading causes of long-term disability differ from the leading causes of death. Thus, disability is just one dimension of health and hence the measures based solely on it cannot capture the

health status of a population. Morbidity statuses have to be explicitly included for measurement of health status.

Second, the authors do not consider re-entries into any of the non-absorbing states considered. In the measurement of health status, this has to be taken into account as people are always making transitions across different health states:- they fall ill, get hospitalized and recover. This dynamic change is experienced even in advanced ages. The failure to take reentries into account treats someone who had long term disability and from which the person had since recovered, the same way as one with long term disability (Wilkins and Adams, 1983a, 1983b)

Some of the works outlined above have been further developed by other authors. Chen, Bush and Patrick (1975) and Newman (1988) criticized and subsequently added other dimensions to Sullivan's work. Chen, Bush and Patrick (1975) used two operational definitions for health; function level or the level of wellbeing and prognosis or an individual's expected transitions to other levels of health. To quantify function level, they combined different steps of the norms for social activity, mobility and physical activity, and created thirty-one levels of function activity to which they assigned different values W_j ranging from 0, representing death and 1 representing being well. They proposed the function status index W given as:

$$W = \sum_{j=0}^{30} \frac{N_j W_j}{N} \qquad (33)$$

Where

N is the total number of persons in the population

N_j is the number of persons in function level j

W_j is the measured weight or social preference for function level j

j is the index for function levels 0, 1, 2, ..,30.

They cite ten advantages of this function status index. However by their operational definition, the function status index was an incomplete indicator since it did not include prognoses. To determine prognoses, they used transition probabilities in which they assumed a Markovian process under which transitions between function levels can only occur from one age to another and not within the same age. Using these transition probabilities, they constructed a grand transition matrix which they raised to higher powers to obtain an equilibrium distribution. From the equilibrium distribution, they aggregated the proportions for the different function levels over age intervals to obtain an equilibrium vector, from which in turn they computed the equilibrium function level expectancies Y^*. These values for Y^* give the expected duration in the different function levels.

Finally, they combined the function level expectancies Y^* and the function status

index W to obtain the value adjusted life expectancy Q^* :

$$Q^* = \sum_j W_j Y_j^* \qquad (34)$$

The quality-adjusted life expectancy of Wilkins and Adams (1983) is very similar to this life expectancy and goes by the same name. While it has several merits, the quality adjusted life expectancy has some arbitrariness to it as it cannot easily be replicated.

The Chen, Bush and Patrick model has several merits. First, the inclusion of transitions between states adds a dimension to ordinary life table techniques. Second, they used different states of health which tries to capture the multidimensional nature of health. A few points of criticism are as follows: i) they considered only disability states and failed to explicitly include states that are morbid but not disabled as is the case for the other models. ii) A model with thirty-one possible states for each age group is very large and can hardly be described as being parsimonious. They recommended that these be consolidated into a shorter list on the basis of further studies. iii) The type of transitions suggested by them are preferred transitions or transitions expected to be made rather than actual transitions already made. iv) Both the function level expectancy and value-adjusted life expectancy are single values from which the age component has been eliminated. This is a serious shortcoming as it cannot reveal age differences in health expectancy. v) In seeking a health model that could provide the framework for the Canada Health Survey, Collishaw (1974) reviewed several models including the model of Chen, Bush and Patrick. His comments on the scaling technique used in determining W, are as

follows:

> " This technique has proved to be valid and reliable with reference to small groups where attributes and values can be expected to be relatively homogeneous, but it may be of limited utility in a national survey that cuts across many cultural groups where such homogeneity of values is unlikely." (Collishaw, 1974)

In his work, Newman (1988) sought to put Sullivan's framework in a multistate perspective. As will be seen later, what Newman did had actually been done by Pollard (1980) and generalized by Haberman (1983). Nevertheless, Newman considered a three state Markov process namely well, ill and dead. Theoretically, he considered reentries from the ill state to the well state. He however did not develop this further; he made the assumption of a large recovery rate thereby eliminating chronic diseases. Using this assumption of large recovery, he obtained an expression for the person-years spent in the ill state which is very similar to Sullivan's result.

Another weakness in Newman's analysis was in making use of hospital separation data to estimate transition rates $T_{12}(k)$, between the well and sick states and the days-of-stay data ($\div$ 365) to estimate person years spent in the ill state. Using hospital morbidity data for estimating transition rates has several disadvantages. First, the term 'separation' is used to cover death or discharge of a patient. Even if deaths were discounted from the separation data to obtain discharges, still no distinction is made as to whether the discharged patient has become well or is still in the morbid state. This creates a problem in the estimation of reentries. Second, separations are based on events and hence cannot distinguish between multiple hospitalization for one patient from single

hospitalizations for multiple patients (Bah, 1989). For these reasons, Newman's model has to be modified if it is to be used to measure the health status of a population.

Rogers, Rogers and Branch (1989) also improved on the work of Katz *et al.* Theoretically, the methodology of Rogers, Rogers and Branch is very sound as they used reentries in a multistate perspective. They however restricted themselves to active life expectancy and considered only dependence and independence in the population.

In the light of the shortcomings of the various models Bah (1990) proposed a multistate model for health status which incorporated some of the merits in the different models above and added a new dimension to them. The model suggests that there exist morbid states which are different from disability states and that there are transitions between these morbid states and the disability states. As in other multistate models, the assumption was made that the transitions were governed by a Markov process. Using these assumptions, he proposed a more comprehensive model for the measurement of health status of low mortality populations.

A. H. Pollard (1980) made a detailed attempt to assess the relationship between morbidity (without disease progression) and mortality using a deterministic model. The author used data on the prevalence rates from certain chronic degenerative diseases and constructed morbidity-mortality tables to analyze the effect of morbidity on mortality. The

method consists of three decrements, one due to incurring the illness, the other due to death from the specific cause and the last due to deaths from other causes. From the morbidity-mortality table, Pollard obtained several indices related to morbidity and its interaction with mortality.

Pollard's (1980) paper, however, considered only decrements from the healthy population without considering recoveries or increments into healthy state. In a follow-up paper, Haberman (1983) took account of recoveries by applying the theory of multistate life table and Markov chains to Pollard's morbidity-mortality model. He applied the general multistate model to study a three-state situation involving a disease Z, the states are: 'free of Z', 'ill with Z' and 'death'. He made the assumption that persons dying of the disease were already affected by the disease just before death. Following the theory of multistate life tables, Haberman derived the multistate life table functions including the expectation of life in the two non-absorbing states. Beside the contribution that Haberman's paper made to morbidity-mortality analysis, it also made a vital link between two disciplines; multistate demography and actuarial statistics.

The empirical morbidity-mortality models of Golini and Egidi (1984) incorporate both the duration aspect of illness as well as the age of onset of the illness. The morbidity-mortality tables constructed by them were analogous to duration dependence life tables for marital status. The models include variables like probabilities of remaining healthy, recovery, remaining sick and probability of dying for the sick. The models were

simplified by assuming that these probabilities were invariant with respect to age and sex. At each age, the states considered were the healthy state, and various durations of sick states. A transition matrix was then constructed which governs the passage from age x to x+1. The elements of this matrix express the probabilities of an individual's remaining in a given state, or of changing state from age x to age x+1. A specific transition matrix was applied at each age. Two variants of the model were considered; one in which only a single illness-death process is acting and the other in which there are two competing causes of death. In the latter model, illness-death process is assumed to govern the first cause of death, but for the second cause, only death is considered. Using the models, they were able to evaluate the effect of changes in morbidity on the level and structure of mortality on the one hand and the growth and composition of population on the other.

Building on the works of A. H. Pollard's (1980) as well as Golini and Egidi (1984) and Kitagawa (1955), J. H. Pollard (1990) made some theoretical contributions to the morbidity-mortality modelling. His work was on 'physical manifestation of disease progression' (or health status) and mortality. In this model, the population is categorised by age *i* and health status *j* and a transition matrix is then constructed which governs moves between various healthy and unhealthy states. The differentials in life expectancy can then be partitioned into the various health statuses according to contributions, by age, of the various transition rate differentials.

One can summarize that in the modelling of morbidity and mortality, even though the methodologies employed differ as well as the terminologies used, work has proceeded in four broad directions. In the first, morbidity is considered as one entity without any subdivisions. In this case, the conditions included in the analysis are illness, recovery and death. In the second, the duration of the illness is taken into account. In the third, morbidity is subdivided into ordinal states ranging from mild to severe conditions or from short to long duration. In the third case, morbidity is subdivided into discrete health statuses which include various aspects of disability and dependency.

Since this work is written at an aggregate level, the last approach cannot be gainfully employed. Instead the second approach is favoured. This is partly because the morbidity model outlined in the previous chapter involved some element of duration. The model adopted is reviewed in the following section.

5.3 Dynamic Model of Morbidity and Mortality

From the different models reviewed above, it could be seen that any study on the dynamics of morbidity and mortality necessitates the inclusion of several states from the health space spanning 'health' to 'death'. The methodologies employed range from deterministic to stochastic. Furthermore, the aspect of ill-health studied takes various forms. In some cases the duration of illness is studied and in others, the health status (including the degree of disability or the degree of severity) is studied without paying

much attention to kind of illness.

The method opted for is a multistate demographic approach. The difference between the method to be proposed here and the other related works cited above will be in the state space employed. As the diseases being focused on are the lethal infectious diseases and also because of data constraints, the 'health status' approach cannot be employed. The state space proposed are as follows:

1) healthy state (H) (origin stage)

2) ill from disease 1 (I1)

3) ill from all other diseases (I2)

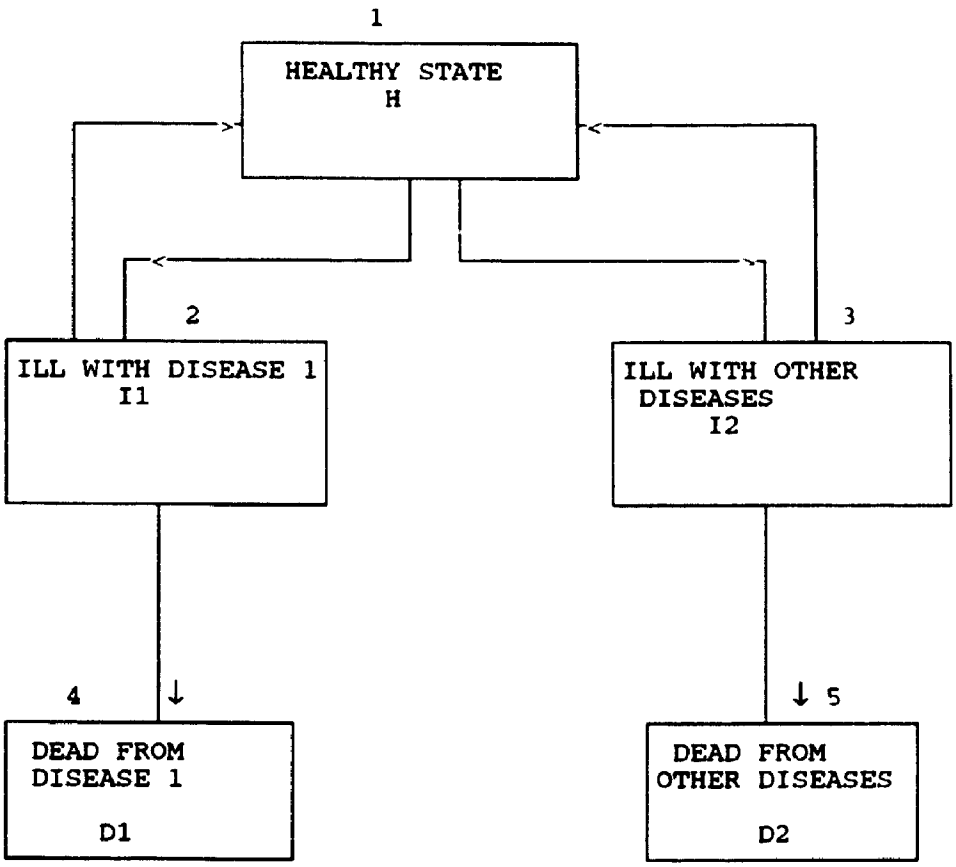
4) dead from disease 1 (D1)

5) dead from all other diseases (D2)

In this state space, there are 3 transient states H,,I1 and I2, all of which experience both increments and decrements. The rest of the states are absorbing. If one assumes that the diseases act independently (the normal practice in mortality analysis), the possible transitions from one state to another (i-j) are 1-2, and 1-3, and their reverse, 2-1, 3-1 and the transition to the absorbing states, 2-4 and 3-5. These states and the possible transitions are illustrated in Figure 5.1.

The core of analysis in multistate demography is the derivation of the transition probability matrix **p**(x). The elements $p_{ij}(x)$ of **p**(x) represent the probability that an individual of exact age x in state i will be in state j at age x+1 (one year later).

Figure 5.1 State Space for Morbidity-Mortality Analysis Showing Possible Transitions.



This transition probability matrix $p(x)$ is given by:

$$p(x) = [I + \tfrac{1}{2}\, m(x)]^{-1}[I - \tfrac{1}{2}\, m(x)] \qquad (35)$$

where $\mathbf{m}(x)$ is the matrix of occurrence exposure rates given as[1]:

$$\begin{bmatrix} m_{12}(x) + m_{13}(x) & -m_{21}(x) & -m_{31}(x) \\ -m_{12}(x) & m_{21}(x) + m_{28_1}(x) & 0 \\ -m_{13}(x) & 0 & m_{31}(x)+m_{38_2}(x) \end{bmatrix} \qquad (36)$$

The derivation of the matrix $\mathbf{p}(x)$ has been given in several articles on multistate demography (Rogers (1975);Rogers and Ledent (1976); Ledent (1980)). The assumptions made for its derivation include the following:

a) Markovian assumption- the probability of transition during a certain interval depend only on the state in which an individual is at the beginning of the interval and is independent of previous states.

b) Uniformity- transitions are assumed to be distributed uniformly over the interval x to x+1.

c) Equality of rates- the age-specific life table rates are equal to annual age-specific rates of observed population (Willekens, 1980).

In this mortality-morbidity multistate model, the transition from the healthy state to the illness states are the incidence rates which are represented in the matrix $\mathbf{m}(x)$ by the mobility rates $m_{12}(x)$ and $m_{13}(x)$. The transition from the illness states to the healthy

[1] The assistance of Dr. Ledent is gratefully acknowledged in conceptualizing this matrix.

state are the recovery rates which are represented by the mobility rates $m_{21}(x)$ and $m_{11}(x)$. Finally, the transitions from the illness states to the dead states are the case fatality rates (closely related to the cause of death rates) and are represented by the mobility rates m_{281} (x) and $m_{382}(x)$.

If one is considering a 'status-based' multistate life table, the other life table measures are given below:

$$ {}_{x}L(x) = .5*[I+p(X)] \tag{37} $$

where ${}_{x}L(x)$ is the matrix of the person years lived. Its elements are ${}_{kx}L_{i}(x)$ which is the average time in years spent in state i between ages x and x+1 by a person in state k at the exact age x. And,

$$ {}_{x}e(x)={}_{x}T(x)=\sum_{t=x}^{z} {}_{x}L(t) \tag{38} $$

where ${}_{x}e(x)$ and ${}_{x}T(x)$ are, respectively, the matrices of life expectancy and person years expected to be lived by status at age x. The ${}_{x}e(x)$ matrix is perhaps the most informative of the multistate life table functions. Its elements $e_{ij}(x)$ denote the number of years that a person in state i at age x can expect to spend in state j beyond that age.

While the dynamic model uses a multistate approach it incorporates the submodels described in the previous chapters (the morbidity and the cause of death submodels) in an attempt to obtain the relevant transition rates. The incidence rates are obtained directly from the morbidity submodel and the case fatality rates (m_{281} (x) and $m_{382}(x)$) are derived

from the cause of death submodel. All of these rates served as inputs for the dynamic model.

5.4 Application of the Dynamic Model

In order to apply the model, two programs are used; a modified version of MSTATE.FOR[2] and the author's program DYNAMIC.FOR. Both of them together are referred to here as the Dynamic model. The program MSTATE.FOR is used for the main multistate demographic analysis. The program DYNAMIC.FOR consists of subprograms which deal with the estimation of the relevant transition rates needed for the main multistate model. As such, all the assumptions of the cause of death submodel and the morbidity submodel outlined in the previous chapters were incorporated in the dynamic model. Since some of the data needed could not be easily obtained, it has been found necessary to make further simplifying assumptions. Specifically, it has been assumed that the case fatality rates were related to the corresponding cause of death rates by a constant multiple.

The outputs of the different runs of the Dynamic model are given in Tables 5.1 through 5.6. As the multistate life tables constructed are 'status based', for each output of the program, three panels are provided, each representing the initial cohort in a specific non-absorbing state i.e Well State, TB state and Others State. The input data for the

[2] Provided by Dr. R. Fernando.

model are the predicted rates for Guinea males, 1965 when applied to a standard population. Two multistate matrix functions are shown in the tables; the elements of the matrix of survivorship functions l(x) and those of the matrix of life expectancy e(x). It was necessary to start the radix at age 15 as there was no occurrence of TB below those ages.

In the first run of the model, the results obtained are shown in Table 5.1. Focusing on the e(x) matrix, panel a shows that for a cohort whose initial status at age 15 is the well state, the total expectation of life at age 15 is 42.74. Of this amount, 35.64 years is expected to be spent in the well state, 6.89 in the others state and 0.2 in the TB state. With increase in age, the total expectation of life in each of these states decreases. The highest decrease occurs for those in the well state followed by those in the others state. For the cohort whose initial status at age 15 is the TB state, panel b shows that the total expectation of life at age 15 is 6.30 years. Of this amount, 5.21 years is expected to be spent in the TB state, 0.93 year in the well state and .16 year in the others state. With increase in age, the time expected to be spent in both the well and the others state show increase. Lastly, for the cohort whose initial status at age 15 is the Others state, panel c shows that the total expectation of life at age 15 is 17.67 years. Of this amount, 17.61 years is expected to be spent in the others state, none in the TB state and a mere 0.06 year in the well state. The expected time to be spent in the well state remains almost constant with increase in age while that in the others state show slight decrease.

Generally, the life expectancies are slightly high for those who are in the same state as they were at age 15. Part of the reason for this is that the predicted death rates and incidence might be low. Secondly, the only deaths included in the model are those that are preceded by illnesses, violent and sudden deaths have been excluded. This exclusion probably contributes to the upward bias in the life expectancy. In spite of these shortcomings, the dynamic model could still be useful in studying changes in the morbidity and mortality parameters. In the following sections, this exercise is demonstrated.

In the second run of the model, the cause-specific and general death rates obtained from the cause-of-death submodel are inflated to see their effect on the multistate life expectancies. The rates were increased by a factor of three and the output results are shown in Table 5.2. When compared to the results in run 1, panel a shows more than 50% decline in the total life expectancies. For age 15, out of a total life expectancy of 20.26, 13.10 is expected to be spent in the others state while 0.21 year is expected to be spent in the TB state and 6.95 in the well state. With increase in age, the distribution pattern is similar. This suggests that with increase in general death rates, the incidence rates for other diseases increase so that the population would expect to spend more time in ill health than in health. At the same time, as cause-specific mortality increases, less time is expected to be spent in the TB state since those contracting the disease are now subjected to higher risk of dying of the disease. This is confirmed again from the results in panel b which show decrease in life expectancy of those whose initial status is TB.

Since only mortality rates for TB were increased and not for other causes, panel c does not show much change from those in run 1.

In run 3, the default data in run 1 were used with the recovery rate for TB reduced to from .005 to .001. The results are shown in Table 5.3. This reduction applies only to TB so the panels a and c show that there is hardly any effect on the life expectancies in the states other than TB. However for panel b, this reduction in recovery rate caused reduction in the expectation of life in the well state. As those ill with TB are now recovering at a lower rate, the life expectancies in TB state show slight increase.

In run 4, with results shown in Table 5.4, the case fatality rates for TB are increased slightly by a factor of about 1.06. As in the case with run 3, the results in panel c are not affected, those in panel showed little change. The main changes occur for those in panel b. The result show that, compared to the results in run 1, the life expectancy for those in the TB state show decline. For age 15, for example, the decline is from 5.21 years to 5.04. Unlike the case in run 3 where lower recovery rates mean longer life with TB, in this case, the higher case fatality rates imply lower life expectancy for those with TB.

In a similar manner, the case fatality rates for other illnesses were increased in run 5 and the results are shown in Table 5.5. Panel c shows that the expectation of life in the others state show a decline of about a year for all the ages shown.

In run 6, the five-year distribution of the disease-specific death rate is changed by increasing c(i) from 0.2 to 0.35. The results are shown in Table 5.6. Comparing with the results in run 1, all the total life expectancies show increase in all the panels. The increase is most for those whose initial status at age 15 was the well state followed by those in the TB state.

This chapter has been using a multistate demographic model to investigate the dynamics of morbidity and mortality. The investigation has shown that increasing the case fatality rates of TB would not only reduce the life expectancy of those ill with the disease but also cause reduction in the expected time to be spent in the healthy state by those with TB. The results also show that if the recovery rate is reduced, the more years will be spent in ill health and less in the healthy state. The next chapter hopes to interpret these findings in the light of health care planning in Africa. Before that, the situation of health care planning in Africa is reviewed.

Table 5.1 Multistate life table of morbidity and mortality-rural

	Expected number of survivors at age x in each state (l_x)				Expectation of life by status at age 15 (e_x)				
Panel a)	Initial status of cohort- Well State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	100000	0	0	15	42.74	35.64	0.20	6.89
16	99960	96984	254	2722	16	41.76	34.67	0.20	6.88
17	99830	94378	682	4769	17	40.81	33.76	0.20	6.85
18	99554	91984	712	6858	18	39.92	32.92	0.19	6.81
19	99196	89572	780	8844	19	39.06	32.12	0.18	6.76
20	98732	86904	945	10884	20	38.24	31.38	0.18	6.69
21	98081	84494	884	12712	21	37.50	30.71	0.17	6.62
22	97326	82115	922	14288	22	36.78	30.09	0.16	6.53
Panel b)	Initial status of cohort- TB State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	100000	0	15	6.60	0.93	5.31	0.16
16	91166	470	90690	6	16	5.86	1.02	4.67	0.17
17	83262	884	82357	21	17	5.37	1.10	4.08	0.19
18	69689	1237	68410	46	18	5.32	1.30	3.79	0.23
19	58688	1511	57099	78	19	5.22	1.53	3.43	0.27
20	49716	1724	47874	118	20	5.07	1.77	2.99	0.31
21	39124	1885	37079	160	21	5.31	2.20	2.71	0.39
22	31162	1995	28964	202	22	5.54	2.70	2.35	0.49
Panel c)	Initial status of cohort- OTHERS State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	0	100000	15	17.67	0.06	0.00	17.61
16	97981	10	0	97971	16	17.02	0.06	0.00	16.96
17	95703	19	0	95684	17	16.42	0.07	0.00	16.35
18	93261	28	0	93233	18	15.83	0.07	0.00	15.77
19	90600	36	0	90564	19	15.29	0.07	0.00	15.22
20	87743	44	0	87698	20	14.77	0.07	0.00	14.70
21	84648	51	0	84597	21	14.29	0.07	0.00	14.22
22	81349	59	0	81290	22	13.85	0.08	0.00	13.77

Source: Extract from the output of the Dynamic model

Table 5.2 Multistate life table of morbidity and mortality-rural

	Expected number of survivors at age x in each state (l_x)					Expectation of life by status at age 15 (e_x)			
Panel a)	Initial status of cohort- Well State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	100000	0	0	15	20.26	6.95	0.21	13.10
16	99882	91010	749	8123	16	19.29	6.00	0.21	13.08
17	99491	82864	1941	14686	17	18.36	5.15	0.19	13.01
18	98666	74966	1998	21702	18	17.51	4.39	0.18	12.94
19	97577	66886	2131	28560	19	16.70	3.72	0.16	12.83
20	96145	57313	2442	36390	20	15.94	3.13	0.14	12.68
21	94135	48605	2196	43334	21	15.27	2.63	0.11	12.53
22	91784	40898	2110	48776	22	14.65	2.21	0.09	12.35
Panel b)	Initial status of cohort- TB State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	100000	0	15	5.68	0.15	5.21	0.32
16	91166	455	90691	19	16	5.18	0.16	4.67	0.35
17	83261	828	82364	69	17	4.62	0.17	4.07	0.38
18	69684	1108	68419	158	18	4.43	0.19	3.79	0.45
19	58676	1285	57112	279	19	4.16	0.21	3.43	0.53
20	49693	1345	47893	455	20	3.83	0.22	2.99	0.62
21	39082	1336	37098	648	21	3.73	0.24	2.71	0.78
22	31093	1276	28986	831	22	3.56	0.26	2.35	0.95
Panel c)	Initial status of cohort- OTHERS State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	0	100000	15	17.63	0.01	0.00	17.62
16	97981	9	0	97971	16	16.98	0.01	0.00	16.97
17	95703	18	0	95685	17	16.37	0.01	0.00	16.36
18	93261	25	0	93235	18	15.79	0.01	0.00	15.78
19	90600	31	0	90568	19	15.24	0.01	0.00	15.23
20	87742	35	1	87706	20	14.72	0.01	0.00	14.71
21	84647	38	1	84609	21	14.24	0.01	0.00	14.23
22	81347	39	1	81307	22	13.79	0.01	0.00	13.78

Source: Extract from the output of the Dynamic model

Table 5.3 Multistate life table of morbidity and mortality-rural

	Expected number of survivors at age x in each state (l_x)					Expectation of life by status at age 15 (e_x)			
Panel a)	Initial status of cohort- Well State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	100000	0	0	15	42.71	35.62	0.20	6.89
16	99960	96983	255	2722	16	41.73	34.64	0.20	6.88
17	99829	94376	684	4769	17	40.78	33.73	0.20	6.85
18	99553	91980	715	6858	18	39.89	32.89	0.19	6.81
19	99194	89565	785	8844	19	39.04	32.09	0.19	6.76
20	98730	86894	953	10883	20	38.22	31.35	0.18	6.69
21	98076	84472	893	12711	21	37.47	30.69	0.17	6.61
22	97319	82099	932	14287	22	36.76	30.07	0.16	6.53
Panel b)	Initial status of cohort- TB State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	100000	0	15	5.51	0.19	5.29	0.03
16	91149	94	91054	1	16	4.99	0.21	4.75	0.04
17	83199	177	83017	4	17	4.42	0.22	4.16	0.04
18	69493	248	69235	9	18	4.20	0.27	3.88	0.05
19	58340	304	58020	16	19	3.90	0.31	3.54	0.05
20	49210	348	48838	24	20	3.53	0.36	3.11	0.06
21	38390	381	37977	32	21	3.39	0.46	2.85	0.08
22	30227	404	29782	41	22	3.17	0.57	2.50	0.10
Panel c)	Initial status of cohort- OTHERS State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	0	100000	15	17.67	0.06	0.00	17.61
16	97981	10	0	97971	16	17.02	0.06	0.00	16.96
17	95703	19	0	95684	17	16.42	0.07	0.00	16.35
18	93261	28	0	93233	18	15.83	0.07	0.00	15.77
19	90600	36	0	90564	19	15.29	0.07	0.00	15.22
20	87743	44	0	87698	20	14.77	0.07	0.00	14.70
21	84648	51	0	84597	21	14.29	0.07	0.00	14.22
22	81349	58	0	81290	22	13.85	0.07	0.00	13.77

Source: Extract from the output of the Dynamic model

Table 5.4 Multistate life table of morbidity and mortality-rural

	Expected number of survivors at age x in each state (l_x)					Expectation of life by status at age 15 (e_x)			
Panel A)	Initial status of cohort- Well State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	100000	0	0	15	42.91	35.82	0.18	6.91
16	99960	96998	240	2722	16	41.93	34.85	0.18	6.90
17	99830	94417	643	4770	17	40.98	33.94	0.18	6.87
18	99555	92030	665	6860	18	40.10	33.09	0.17	6.83
19	99199	89627	725	8847	19	39.24	32.30	0.17	6.77
20	98738	86973	876	10888	20	38.42	31.55	0.16	6.71
21	98091	84561	813	12718	21	37.67	30.89	0.15	6.63
22	97343	82203	843	14296	22	36.95	30.27	0.14	6.54
Panel B)	Initial status of cohort- TB State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	100000	0	15	9.09	0.90	5.04	0.15
16	90685	468	90210	6	16	5.67	0.99	4.51	0.17
17	82394	879	81494	21	17	5.19	1.08	3.92	0.19
18	68263	1224	66993	46	18	5.16	1.29	3.64	0.22
19	56925	1494	55355	77	19	5.09	1.53	3.29	0.27
20	47772	1699	45957	116	20	4.97	1.79	2.86	0.32
21	37091	1851	35082	158	21	5.25	2.25	2.60	0.40
22	29176	1953	27024	199	22	5.54	2.80	2.24	0.51
Panel C)	Initial status of cohort- OTHERS State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	0	100000	15	17.67	0.06	0.00	17.61
16	97981	10	0	97971	16	17.03	0.06	0.00	16.96
17	95703	19	0	95684	17	16.42	0.07	0.00	16.35
18	93261	28	0	93233	18	15.84	0.07	0.00	15.77
19	90600	36	0	90564	19	15.29	0.07	0.00	15.22
20	87743	44	0	87698	20	14.77	0.07	0.00	14.70
21	84648	51	0	84597	21	14.29	0.07	0.00	14.22
22	81349	58	0	81290	22	13.85	0.08	0.00	13.77

Source: Extract from the output of the Dynamic model

Table 5.5 Multistate life table of morbidity and mortality-rural

	Expected number of survivors at age x in each state (l_x)					Expectation of life by status at age 15 (e_x)			
Panel a)	Initial status of cohort- Well State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	100000	0	0	15	46.25	40.20	0.22	5.82
16	99960	97289	255	2417	16	45.27	39.23	0.22	5.81
17	99830	94926	684	4220	17	44.33	38.32	0.22	5.79
18	99554	92791	715	6048	18	43.45	37.48	0.21	5.75
19	99198	90639	784	7775	19	42.60	36.69	0.21	5.70
20	98738	88256	952	9529	20	41.80	35.96	0.20	5.64
21	98091	86106	893	11092	21	41.07	35.31	0.19	5.57
22	97345	83986	934	12425	22	40.38	34.70	0.18	5.50
Panel b)	Initial status of cohort- TB State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	100000	0	15	6.39	1.05	5.21	0.13
16	91166	470	90690	6	16	5.96	1.15	4.67	0.15
17	83262	886	82357	19	17	5.48	1.25	4.08	0.16
18	69689	1239	68410	40	18	5.45	1.47	3.79	0.19
19	58688	1520	57100	68	19	5.38	1.73	3.43	0.22
20	49716	1739	47874	103	20	5.26	2.01	2.99	0.26
21	39124	1906	37079	139	21	5.55	2.50	2.72	0.33
22	31162	2023	28965	175	22	5.84	3.08	2.35	0.41
Panel c)	Initial status of cohort- OTHERS State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	0	100000	15	16.53	0.07	0.00	16.46
16	97731	10	0	97721	16	15.90	0.07	0.00	15.84
17	95179	19	0	95160	17	15.32	0.07	0.00	15.25
18	92451	28	0	92423	18	14.75	0.07	0.00	14.68
19	89489	36	0	89453	19	14.23	0.07	0.00	14.15
20	86320	44	0	86276	20	13.73	0.08	0.00	13.65
21	82902	51	0	82851	21	13.27	0.08	0.00	13.20
22	79276	58	0	79218	22	12.86	0.08	0.00	12.78

Source: Extract from the output of the dynamic model

Table 5.6 Multistate life table of morbidity and mortality-rural

	Expected number of survivors at age x in each state (l_x)					Expectation of life by status at age 15 (e_x)			
Panel a)	Initial status of cohort- Well State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	100000	0	0	15	47.85	41.57	0.21	6.07
16	99960	96984	254	2722	16	46.87	40.60	0.21	6.06
17	99836	94834	650	4351	17	45.93	39.69	0.20	6.04
18	99587	92918	623	6046	18	45.04	38.85	0.20	6.00
19	99275	90968	673	7635	19	44.18	38.04	0.19	5.95
20	98876	88768	829	9279	20	43.36	37.29	0.18	5.89
21	98319	86836	759	10724	21	42.60	36.60	0.18	5.82
22	97679	84922	805	11952	22	41.87	35.96	0.17	5.74
Panel b)	Initial status of cohort- TB State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	100000	0	15	6.43	1.08	5.21	0.14
16	91166	470	90690	6	16	6.01	1.18	4.67	0.15
17	83262	887	82357	18	17	5.53	1.29	4.08	0.17
18	69689	1242	68409	38	18	5.51	1.52	3.79	0.20
19	58688	1526	57098	64	19	5.45	1.79	3.43	0.23
20	49718	1749	47873	96	20	5.34	2.08	2.99	0.27
21	39127	1921	37077	130	21	5.65	2.59	2.72	0.34
22	31167	2041	28962	163	22	5.97	3.19	2.35	0.43
Panel c)	Initial status of cohort- OTHERS State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	0	100000	15	17.68	0.07	0.00	17.61
16	97981	10	0	97971	16	17.03	0.07	0.00	16.96
17	95703	19	0	95684	17	16.43	0.08	0.00	16.35
18	93261	28	0	93232	18	15.84	0.08	0.00	15.77
19	90600	37	0	90563	19	15.29	0.08	0.00	15.21
20	87743	44	0	87698	20	14.78	0.08	0.00	14.69
21	84648	52	0	84596	21	14.30	0.08	0.00	14.21
22	81349	59	0	81289	22	13.86	0.09	0.00	13.77

Source: Extract from the output of the Dynamic model

CHAPTER VI

HEALTH CARE PLANNING IN AFRICA AND THE DYNAMICS OF MORBIDITY AND MORTALITY

6.1 Health Care Planning in Africa

As mentioned in Chapter 2, health care planning takes account of health needs and wants, utilization of health care, allocation of health care resources and health care delivery. Literature on health care delivery in Africa reveals two distinct dichotomies: the Western versus the traditional medical system and urban versus rural.

The first dichotomy is actually a simplification used by several authors for the purpose of general discussion. While some authors use 'traditional' to refer to indigenous African medicine (Good *et al.*, 1979), others use it synonymously with non-Western medicine including those with 'empirico-rational' as well as 'magico-rational' elements (Buschkens and Slikkerveer, 1982). There is however evidence of other health care systems (HCS) in Africa (Gesler, 1984). For example, before the arrival of modern medicine, there were two health care systems in many Islamic societies in Africa; in Morocco, there was the *galenic* medicine (traced back to the Greek physician Galen) and the *Prophetic* (also known as *tibbi* or Islamic) medicine (Greenwood, 1981). In a study of health care in Eastern Oromo in Ethiopia, Buschkens and Slikkerveer (1982) identify three types of health care systems: the traditional, the modern and the transitional. The traditional health care system is described as a fusion of pre-Islamic (Oromo health care), Islamic and Amharic (Ethiopian) medicine. The transitional health care system refers to

a system which includes elements of both the modern and the traditional system, but which is virtually beyond the control of either of them.

Traditional medicine in Africa is very significant. According to Dunlop (1975:582):

> 'Evidence suggests ...that a traditional health care system exists, legally or illegally in virtually all African countries if for no other reason than historical precedent and cultural acceptance.'

Dunlop has outlined the costs and benefits of the traditional health care system and Good (1977:705-706) has defended it as follows:

> '*Traditional medicine*, although nonscientific in the Western biomedical view, is logical (internally consistent), valid and relevant in its own behavioral settings.'

Despite the prevalence of the traditional health care system, it barely gets formal recognition in many African countries. Rather, most countries emphasize Western medicine and the Western health care delivery model. Therein lies the contradiction: the Western medicine reaches only a fraction of the population (Good, 1977). In Ethiopia, for example, a study showed that 85% of the population were beyond the reach of modern medicine (Workneh and Giel, 1975). Furthermore, what has not been realized in Africa is that the Western health care model is being questioned by scientists and non-scientists alike even in the developed world (Gesler, 1984). In an attempt to resolve the problems inherent in the Western-traditional system dichotomy, a call for integration between the two systems has been made by the WHO as early as the 1970's (Mahler, 1977). After

reviewing health care delivery systems in Africa. Good *et al.* (1979:152) conclude as follows:

> 'In summary, it is argued that countries of Africa and the developing world in general do themselves a disservice by officially ignoring traditional medicine, by excluding it from the central planning process, and by not considering its partial or fuller incorporation as a public policy option in health care planning.'

And quite recently, Alubo (1990:165) has reiterated the point again:

> 'It is here proposed that a saving option for health services in Africa is a return to the indigenous healing system which being part of the cultural heritage, would be self reliant.'

This idea of integration of Non-Western and Western health care systems has received further support through the advocates of 'medical pluralism' - the recognition of different health care systems as being valid and making the best out of them. According to Gesler (1984:75):

> 'A generalization can be made that medical pluralism is part of a solution to health care delivery problems because it offers a wide choice of alternatives to those who are sick.'

On the second dichotomy of urban versus rural, the prevailing pattern in the health care system in most African countries is a skewed focus on health in the urban areas. One of the outspoken critics of this imbalance is Mburu (1979, 1981). He argues strongly against urban based health care systems in Africa when less than 25% of the population is urbanized. Furthermore,

> 'The emphasis on hospitals, complete with highly technical equipment, is fundamentally an emphasis in the care of urban communities, less needy than their rural counterparts.' (Mburu, 1981:18).

Mburu contends that the prevention of infectious and communicable diseases (the most important causes of morbidity and mortality) do not need highly specialized medical staff.

In Africa, the root source of both of these dichotomies is the same - colonialism. The colonialists assumed that the Western medicine was the only valid medical system, which resulted in the rejection of non-Western medical systems (Gesler, 1984). Furthermore, as the colonialists were mostly settled in the cities, the health care system they developed was to cater to their own needs rather than those of the population in the hinterlands. At the end of colonialism, the urban elites took over the same system and further perpetrated the 'core-periphery' model of health care delivery.

Turning now to the other aspect of health care planning, namely health care utilization, one sees the actual behaviour of the population with respect to ill health. One can see if the pattern of health care utilization matches the expectations of governments. In a study done by Sussman (1982) in Mauritius, it was found that despite the free medical services provided to the population by the government and the abundance of biomedical practitioners, there were ten other types of healers reflecting the ethnic, religious and cultural diversity existing on the island. In another study, Swantz (1979) speaks of the Zaramo ethnic group in and around Dar-es Salaam in Tanzania who make use of both traditional and modern medicine. In Nigeria, Ojanuga and Lefcowitz (1982) found that there were four types of health care users, those who:

1) Used either Western or traditional medicine exclusively (most of the population)

2) Used traditional medicine first and then modern medicine

3) Switched from modern to traditional medicine; and

4) Used both simultaneously.

A similar pattern was found in other developing countries such as India (Bannerji, 1981) and Malaysia (Chen, 1981) among several others.

Thus in Africa, evidence from both health care delivery systems and health care utilization suggests that health care planning can best be approached from the standpoint of medical pluralism on the one hand and the focus on the majority rural population on the other hand. The concept of medical pluralism fits in well with the health transition theory elaborated on in chapter 2. As the health transition theory puts emphasis on non-medical determinants of health, in one respect, this suggests the need for a non-Western health care system to respond to the diversities in determinants of health. Both Western and non-Western health care systems are catered for in medical pluralism.

6.2 Review of Health Care Planning Models

The aspect of health care planning to which demographers have contributed the most is in the estimation of needs and demands for health care. This involves measuring various aspects of health status through the use of health indicators and health indices. A health indicator is defined as:

> 'A variable, susceptible to direct measurement that reflects the state of health of persons in a community. Examples include infant mortality rates, incidence rates based on notified cases of disease, disability days etc.' (Last, 1988:57)

and a health index is defined as:

> 'A numerical indication of the health of a given population derived from a specified composite formula. The components of the formula my be infant mortality rates, incidence rates for particular disease, or other health indicators.' (Last, 1988:57).

In general, indicators represent only one class of data (e.g. mortality or morbidity but not the two together) and indexes refer to more complex, multi-dimensional measures or scales composed of a number of indicators (Murnaghan, 1981). Recent work on indicators would include the indirect estimation of morbidity and the conversion of ordinary health statistics into the form required by health planners. Work done on health indices would include the rich volume of research done in developing various health status indices (some of which have already been discussed in the previous chapter).

The next significant aspect of health care planning (HCP) to which demographers have contributed is in resource allocation. In reviewing resource allocation models, Gibbs (1977) has classified the existing models into three types: 'macro-economic, behaviour simulation and system optimization.' These have been defined as follows:

> '*macro-economic:* models consisting of linear equations (or transforms of linear equations) relating aggregate variables such as consumption, supply and price of health services, and population attributes, whose parameters are estimated by multiple regression analysis of current or historic aggregate data;
> *behavior simulation:* models based on hypotheses concerning the behaviour of physicians, patients, and other decentralized decision makers in the HCS: and,

> *system optimization:* models designed to identify the set of resource allocations that optimize a defined objective function of the HCS.'(Gibbs, 1977:3)

Among the shortcomings of these different approaches, Gibbs (1977) notes the following. In macro-economic models, there is a high level of aggregation; the hypotheses proposed are limited to linear relations (or their transformations) between aggregate quantities. The models are not valid outside the range of variables in the data used for the estimation of the models' coefficients. In system optimization models, it is impossible to define satisfactory objective function for the HCS as a whole. They are also unrealistic since real behaviour in the HCS does not correspond to 'system optimum' allocations. In behaviour simulation models, data may be difficult to obtain; standard techniques and terminology are not available and the models may not be universally applicable. After comparing all the advantages and disadvantages of the different approaches, Gibbs (1977) recommended that the behaviour simulation models be used in the HCS modelling task at the International Institute for Applied Systems Analysis (IIASA).

For overall HCP, several approaches are possible. Some of the most widely used methods, in addition to optimization models, are as follows:

> '*input-output techniques:* under some restrictive assumptions for the production (generation) of certain amounts of things (outputs, commodities, illness-types, etc.) the amounts of necessary inputs at a certain level of technology are given. These techniques are often used in combination with constraints and an objective function for optimization.
>
> *stochastic processes:* a matrix shows the probability of transition from state i to j over time (Markovian model).

queuing: one can express how many units of things or persons arrive within an interval of time. and how long they have to wait to be served (or treated).

gravity model: This model is used mostly by urban or regional planners. It implies that people living at a greater distance from an institution are less likely to use it, and that people use a larger institution more often than a smaller one.

DYNAMO-model: to create a dynamic model, main variables are split into levels (stocks) and rates (flows, changes per time units). A computer language enables the user to circumvent the explicit mathematical formulation by means of direct programming statements. Only recursive structures can be generated. (No influence from A to B and from B to A at the same moment is allowed.) DYNAMO's numerical abilities approximate a set of canonical differential equations by means of (nonlinear) difference equations.

econometric models: originally these methods were developed for application in the economic sector. Later on these techniques of model formulation, parameter estimation and forecasting were used in other sectors as well. An econometric model consists of behavioural and "quasi laws" of the sector under investigation, definitional equations represent identities assured by theoretical considerations.

logical model: for describing decisions the logical structure can be reflected by 0/1 variables (yes/no, true/false) and their relationships.' (Fleissner and Klementiev, 1977:6)

Of the different approaches, those that incorporate health indicators and indices and other demographic variables in the overall modelling are of more interest. This was attempted in a system dynamics (DYNAMO) model proposed by Kaihara (1976) and further developed in Kaihara *et al.* (1977). The population is divided into four health groups: healthy, unaware sick (i.e., sick without being aware of it), sick without medical care and patients. Each group is categorised into these health groups. It is then assumed

that flows of people exist between health groups within an age group and between age groups within a health group. The analytic approach adopted is to transform health statistical data pertaining to these groups into essential or 'primary factors.' The concept of 'primary factors' was proposed by Kaihara *et al.* in order to distinguish them from 'secondary factors' such as socio-economic factors which are also related to the health care system. The primary factors identified for the health care system were: population structure, morbidity rate, recovery rate, death rate, patient registration rate and awareness rate. The mechanism of change of medical demand is broken down into four factors: demographic change, rate at which the unaware sick become aware of their illnesses, the rate of recovery and accessibility to physicians (Fleissner and Klementiev, 1977). The model was applied to Japanese data by Kaihara *et al.* (1978) where the trends of the primary factors were calculated and used to obtain future trends in medical demand in Japan.

Klementiev (1976) proposed a mathematical approach to developing a simulation model of a health care system. The model includes some aspects of morbidity, resource supply and resource allocation. The morbidity aspect of the model considers the processes by which individuals transfer between the states of healthy, latent sick, revealed sick, treated sick, dead and between the different phases of disease. The resource allocation aspect of the model is of the behaviour-simulation type, more specifically, it is based on queuing theory. This was only a first step in the model development.

Another health care system model that uses demographic inputs for health care planning is the 'Aggregate Model for Estimating Health Care System Resource Requirements (AMER)' by Klementiev and Shigan (1978). The approach adopted is quite different from that of Klementiev (1976). This model is based on the linkage of sub-models dealing with population, disease prevalence, resource need, resource supply and resource allocation. In addition to the demographic inputs, the model relies heavily on 'aggregate standards' to replace data that are usually obtained from health surveys. These standards are control variables such as: percent of patients hospitalized, average length of stay in hospital, number of consultations per episode, bed turnover interval, bed occupancy rate, 'beds per inpatient doctor equivalent' and workload in the form of number of consultations per year. Using these data inputs, the model could forecast aggregate resource for health care planning.

At the University of Michigan, a health care model was developed whose essential elements were:

> 'estimates of morbidity and mortality of each age group from the main disease problem for typical populations; of the effects on these rates of providing particular patterns of health care and other social inputs such as sanitation and nutrition and of the cost services provided.'
> (Cumper, 1983:38)

Using the model it was possible to calculate the effect of different patterns of primary health care upon population coverage, and mortality and morbidity reduction by age group. From the model the important conclusion reached is that

> 'approaches which concentrate resources in relatively intensive forms of care (hospitals, health centres) may produce the greatest reduction in mortality and morbidity, though at the expense of limited population coverage. It therefore identifies a possible conflict between two policy objectives- mortality/morbidity reduction as against coverage- which has often been assumed away in the literature on primary health care.' (Cumper, 1983:38-39).

Further development of the Michigan health model led to the DYNPLAN model used for health planning in the context of rapid population growth (Simmons *et al.*, 1986). This model takes account of changes over time of population size, density and age distribution flowing from the changes in mortality and fertility behaviour and their interactions. The DYNPLAN model consist of four sub-systems:

> 'a) the family planning/fertility subsystem
> b) the disease incidence/prevention subsystem
> c) the treatment/curative intervention/mortality sub-system and
> d) the population dynamics built into the model.'
> (Simmons *et al.*, 1986:20).

While this model incorporates rapid population growth in health planning, it has several weaknesses. First, the model requires detailed information about health and population. Second, the model does not specify the characteristics of health problems and interventions of more than one geographic area at a time. Thirdly, the model does not elaborate the full set of interrelationships among its constituent parts, for example, the dynamics between morbidity and mortality or the relationship between different diseases (Simmons *et al.*, 1986).

The health care system models summarized above are those making paramount use of demographic as opposed to economic data. A comprehensive review of other health

care system models is provided in Fleissner and Klementiev (1977).

6.3 Towards Health Care Planning Model for Africa

Having reviewed the literature covering health care systems in Africa and the different models for health care planning, it can be seen that the different models cannot be appropriate for the African setting unless substantially modified. An appropriate health care planning model for Africa has to take account of several factors including the reality of medical pluralism, the high illiteracy and the wide disparity between urban and rural areas in terms of accessibility to and utilization of modern health care services. The reality of medical pluralism means that the health researcher is faced with a situation where individuals switch between two contrastingly different health care systems even in the presence of free medical services. None of the health care planning models reviewed above come even close to doing this task. Only the Primary Health Care system gives some recognition to the use of non-Western medical health care. To model health care in Africa, there is need for a different conceptualization grounded in strong socio-medical theory and developed within the framework of Primary Health Care.

At this stage no attempt is made to develop any health care planning model for Africa. Rather, the next section discusses what the implications for health care planning would be when the dynamics of morbidity and mortality are taken into account.

6.4 Dynamics of Morbidity and Mortality and Health Care Planning

In developing health care programs, a choice is often made on whether the program should be focusing on incidence of and mortality due to specific diseases (disease-specific) or the general improvement in level of health. In some cases where a specific disease is endemic and is a major cause of death, attention is immediately focused on such a disease. That was the case in several countries where vertical intervention programs were implemented especially in the 1950's and 1960's. Such programs were successful partly because these diseases were in the first place responsive to such intervention efforts.

In the 1990's when African countries are undergoing health transitions at different paces, the stage of vertical intervention program of the type described above is mostly over. Even though a few diseases could still be major killers, their reduction need more than just vertical intervention campaigns. At such stages, decision making plays a role. Health care planners have to decide on whether to focus on specific diseases or whether to try to improve the general level of health. For a such decision making, the dynamic model of morbidity and mortality could be useful. This would require comparison of the changes in the diagonal elements in the life expectancy matrix. These elements give the life expectancies that individuals in either the healthy or illness states expect to live.

Another concern of health care planning is on the quality of life. While this concern is taken very seriously in the more developed countries, African countries also pay some attention to it. If the concern is on by how much the quality of life will improve, the dynamic model could also be used to assess this. In such a case, the off-diagonal elements of the $\mathbf{e}_x$ matrix provide the necessary information on the number of years that an individual in the ill state expects to spend in the healthy state. The off-diagonal elements could be compared for different illness states or if hypothetical changes are made to the model, their changes could be observed. In the section following, two scenarios are given to show the application of the dynamic model to health care planning.

6.4.1 Scenario 1

Let us consider an African country that is already in the second stage of the epidemiologic transition- with no pandemics but with death rates still high and some infectious diseases still posing health problems. If the government decides to spend money on sanitation and nutrition, the death rates may decline with overall improvement in health. On the other hand, the death rates due to specific infectious diseases may be affected only to a little extent. For major reduction in deaths due to that disease, special treatment facilities may have to be introduced. Which option should the government adopt ?

Based on experiences from 'rapid mortality decline countries' the African country can estimate the realistic maximum gain in life expectancy achievable if such a country were to invest heavily into sanitation and nutrition programs. Suppose the health planners assess that with such programs in the given country, the realistic gain in life expectancy achievable within 5 years is 4 years. The next information needed is on the efficacy of the special treatment facility. Suppose it is known that the facilities can help reduce death rates by 50%, the dynamic model can then be used to assess the expected gain in life expectancy with the 50% reduction in cause specific death rates. These are then compared with the gain of 4 years expected using the alternative health promoting program.

In order to use the dynamic model to assist in decision making, the default values in Table 5.1 were used as the initial base values. This table is reproduced in Table 6.1. The rates for TB were then reduced by 50% and the results from the model are shown in Table 6.2. Comparison of panel a in the two tables show the following: The 50% reduction in cause-specific death rate would succeed in improving the life expectancy in the healthy population by about 3 years. The expected number of years that the healthy population would expect to spend in the ill health would reduce. The other life expectancies would not show much change. In such a hypothetical situation, the alternative program would be preferable.

6.4.2 Scenario 2

Suppose a given African country is faced with a prevalent lethal infectious disease which could be treated fairly successfully in its early stages but not quite so at the later stages. At the later stage of the disease, health authorities could only hope to improve the quality of life in ill health and reduce case fatality rates. Which option should the health planners take in trying to tackle this problem ?

In order to use the dynamic model to assist in decision-making one needs to know about the efficacy of different curative measures. Suppose it is known that the intensive preventive measures could only improve recovery rates by 30% and that the intensive curative measures could reduce case fatality rates by 20%. With this information, one can run the model for the existing recovery rates and run it again with the increased recovery rates. Similarly, one can run the model with the existing case fatality rates and run it again with the reduced rates. With these sets of results, one can compare the gains in life expectancy 'within' a given health measure as well as 'between' the two health measures.

As was done above, the data in Table 6.1 was used as the base data. The recovery rates were then improved by 30% and the results obtained from the dynamic model are shown in Table 6.3. Comparing of panel b in the two sets of results, one observes that there is reduction in the life expectancy expected to be spent in the TB state. Correspondingly, there is slight increase in expected time that the ill population expects

to spend in the well state.

From the base data, the case fatality rates were reduced by 20% and the results are shown in Table 6.4. On comparing Tables 6.1 and 6.4, one observes from panel b that there is gain in life expected to be lived in the TB and the well states. The expected time that the ill population expects to spend in the well state showed only minimal increase. However comparing panel b in Table 6.1 to each of the Tables 6.3 and 6.4 brings out an interesting situation. While the expected life to be spent in the well state is higher in Table 6.3 than in Table 6.4, the expected time to be spent with TB is higher in Table 6.4. In other words, 30% improvement in TB recovery rate would succeed in improving the expected amount of years in the well state but for those already ill with TB. their life expectancy will not be much altered. From this hypothetical situation, it would seem that the improvement in the case fatality rates would earn the ill population added years of life hence it could be the chosen option.

This chapter started by outlining the problems facing health care planning in Africa. These unique set of problems pose challenges to the formulation of models for health care planning for Africa. However, without resorting to modelling, the dynamic model could still be useful in decision making for health program implementation.

Table 6.1 Multistate life table of morbidity and mortality-with base values

	Expected number of survivors at age x in each state (l_x)				Expectation of life by status at age 15 (e_x)				
Panel a)	Initial status of cohort- Well State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	100000	0	0	15	42.74	35.64	0.20	6.89
16	99960	96984	254	2722	16	41.76	34.67	0.20	6.88
17	99830	94378	682	4769	17	40.81	33.76	0.20	6.85
18	99554	91984	712	6858	18	39.92	32.92	0.19	6.81
19	99196	89572	780	8844	19	39.06	32.12	0.18	6.76
20	98732	86904	945	10884	20	38.24	31.38	0.18	6.69
21	98081	84484	884	12712	21	37.50	30.71	0.17	6.62
22	97326	82115	922	14288	22	36.78	30.09	0.16	6.53
Panel b)	Initial status of cohort- TB State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	100000	0	15	6.30	0.93	5.21	0.16
16	91166	470	90690	6	16	5.86	1.02	4.67	0.17
17	83262	884	82357	21	17	5.37	1.10	4.08	0.19
18	69689	1233	68410	46	18	4.52	1.30	3.79	0.23
19	58688	1511	57099	78	19	3.22	1.53	2.43	0.27
20	49716	1724	47874	118	20	3.07	1.77	2.09	0.31
21	39124	1885	37079	160	21	3.31	2.20	2.71	0.39
22	31162	1995	28964	202	22	3.54	2.70	2.35	0.49
Panel c)	Initial status of cohort- OTHERS State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	0	100000	15	17.67	0.06	0.00	17.61
16	97981	10	0	97971	16	17.02	0.06	0.00	16.96
17	95703	19	0	95684	17	16.42	0.07	0.00	16.35
18	93261	28	0	93233	18	15.83	0.07	0.00	15.77
19	90600	36	0	90564	19	15.29	0.07	0.00	15.22
20	87743	44	0	87698	20	14.77	0.07	0.00	14.70
21	84648	51	0	84597	21	14.29	0.07	0.00	14.22
22	81349	58	0	81290	22	13.85	0.08	0.00	13.77

Source: Extract from the output of the Dynamic Model

Table 6.2 Multistate life table of morbidity and mortality-with 50% reduction in TB rates

	Expected number of survivors at age x in each state (l_x)					Expectation of life by status at age 15 (e_x)			
Panel a)	Initial status of cohort- Well State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	100000	0	0	15	45.61	38.67	0.11	6.83
16	99966	97192	128	2706	16	44.62	37.69	0.11	6.82
17	99858	94806	342	4709	17	43.67	36.77	0.11	6.79
18	99647	92542	556	6749	18	42.76	35.91	0.10	6.75
19	99358	90288	790	8679	19	41.88	35.09	0.10	6.69
20	98975	87887	474	10614	20	41.04	34.33	0.09	6.62
21	98448	85124	444	12879	21	40.26	33.63	0.09	6.54
22	97816	82476	463	13876	22	39.52	32.99	0.09	6.45
Panel b)	Initial status of cohort- TB State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	100000	0	15	6.37	1.01	5.21	0.16
16	91166	470	90690	6	16	5.94	1.10	4.67	0.17
17	83262	886	82355	21	17	5.46	1.20	4.07	0.19
18	69690	1237	68407	45	18	5.42	1.42	3.78	0.22
19	58689	1517	57096	76	19	5.35	1.66	3.42	0.26
20	49718	1736	47869	113	20	5.22	1.93	2.99	0.31
21	39128	1900	37073	154	21	5.50	2.40	2.71	0.39
22	31167	2016	28957	194	22	5.78	2.95	2.34	0.48
Panel c)	Initial status of cohort- OTHERS State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	0	100000	15	17.68	0.07	0.00	17.61
16	97981	10	0	97971	16	17.03	0.07	0.00	16.96
17	95703	19	0	95684	17	16.42	0.07	0.00	16.35
18	93261	28	0	93233	18	15.84	0.07	0.00	15.77
19	90600	36	0	90564	19	15.29	0.07	0.00	15.22
20	87743	44	0	87698	20	14.77	0.08	0.00	14.70
21	84648	52	0	84596	21	14.29	0.08	0.00	14.22
22	81349	59	0	81290	22	13.85	0.08	0.00	13.77

Sources: Extract from the output of the Dynamic model

Table 6.3 Multistate life table of morbidity and mortality-with 30% improvement in TB recovery rates.

	Expected number of survivors at age x in each state (l_x)					Expectation of life by status at age 15 (e_x)			
Panel a)	Initial status of cohort- Well State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	100000	0	0	15	42.75	35.65	0.20	6.89
16	99960	96984	254	2722	16	41.77	34.68	0.20	6.88
17	99830	94379	681	4769	17	40.82	33.77	0.20	6.85
18	99554	91986	710	6858	18	39.93	32.93	0.19	6.82
19	99196	89575	777	8844	19	39.07	32.13	0.18	6.76
20	98733	86908	942	10884	20	38.26	31.39	0.18	6.69
21	98082	84490	881	12712	21	37.51	30.72	0.17	6.62
22	97329	82122	918	14289	22	36.79	30.10	0.16	6.53
Panel b)	Initial status of cohort- TB State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	100000	0	15	6.59	1.20	5.18	0.20
16	91173	610	90554	8	16	6.18	1.31	4.64	0.22
17	83286	1147	82111	28	17	5.72	1.43	4.04	0.24
18	69762	1600	68103	60	18	5.73	1.68	3.75	0.29
19	58817	1958	56758	101	19	5.70	1.97	3.39	0.34
20	49904	2234	47518	152	20	5.63	2.28	2.95	0.40
21	39397	2442	36748	207	21	6.00	2.83	2.67	0.51
22	31508	2583	28664	262	22	6.37	3.45	2.30	0.63
Panel c)	Initial status of cohort- OTHERS State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	0	100000	15	17.67	0.06	0.00	17.61
16	97981	10	0	97971	16	17.02	0.06	0.00	16.96
17	95703	19	0	95684	17	16.42	0.07	0.00	16.35
18	93261	28	0	93233	18	15.83	0.07	0.00	15.77
19	90600	36	0	90564	19	15.29	0.07	0.00	15.22
20	87743	44	0	87698	20	14.77	0.07	0.00	14.70
21	84648	51	0	84597	21	14.29	0.07	0.00	14.22
22	81349	58	0	81290	22	13.85	0.08	0.00	13.77

Source: Extract from the output of the Dynamic model

Table 6.4 Multistate life table of morbidity and mortality-with 20% reduction in TB case fatality rates.

	Expected number of survivors at age x in each state (l_x)					Expectation of life by status at age 15 (e_x)			
Panel a)	Initial status of cohort- Well State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	100000	0	0	15	41.96	34.83	0.30	6.82
16	99960	96918	321	2721	16	40.97	33.86	0.30	6.81
17	99829	94198	865	4766	17	40.03	32.95	0.29	6.79
18	99650	91773	927	6850	18	39.14	32.11	0.29	6.75
19	99185	89317	1037	8831	19	38.28	31.31	0.28	6.69
20	98712	86578	1270	10863	20	37.46	30.57	0.27	6.62
21	98040	84128	1228	12683	21	36.71	29.91	0.26	6.55
22	97260	81707	1302	14251	22	36.01	29.30	0.25	6.46
Panel b)	Initial status of cohort- TB State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	100000	0	15	7.17	1.04	5.96	0.18
16	92870	474	92390	7	16	6.68	1.11	5.38	0.19
17	86371	899	85451	22	17	6.15	1.19	4.75	0.20
18	74916	1268	73602	47	18	6.01	1.36	4.42	0.23
19	65292	1572	63640	79	19	5.82	1.54	4.02	0.27
20	57165	1817	55226	122	20	5.58	1.72	3.55	0.30
21	47168	2012	44990	167	21	5.65	2.05	3.24	0.36
22	39270	2156	36902	212	22	5.69	2.41	2.85	0.43
Panel c)	Initial status of cohort- OTHERS State								
Age	Total	Well	TB	Oth.	Age	Total	Well	TB	Oth.
15	100000	0	0	100000	15	17.67	0.06	0.00	17.61
16	97981	10	0	97971	16	17.02	0.06	0.00	16.96
17	95703	19	0	95684	17	16.42	0.06	0.00	16.35
18	93261	28	0	93233	18	15.83	0.07	0.00	15.77
19	90600	36	0	90564	19	15.28	0.07	0.00	15.22
20	87743	44	0	87698	20	14.77	0.07	0.00	14.70
21	84648	51	0	84597	21	14.29	0.07	0.00	14.22
22	81348	58	1	81290	22	13.85	0.07	0.00	13.77

Source: Extract from the output of the Dynamic model

CHAPTER VII

SUMMARY AND CONCLUSION

7.1 Summary

Health in Africa is generally characterized as one of very high mortality. Often such a description is taken at face value and this has methodological and as well as policy implications. The methodological implication is that analysis should be focused on determining the levels of mortality, especially infant and child mortality and to some extent some of the major differentials in mortality. The policy implication is that vertical intervention programs would be best suited to reduce these high mortality levels. Theoretically, this description fits the epidemiologic transition theory. To a large extent, this perspective worked well in the 1950's and 1960's. However, since mortality decline started to stagnate in parts of Africa, in the 1980's and late 1970's, this perspective has been criticized on several grounds, from various quarters.

From one quarter, there is the advocation of primary health care. From this perspective, the approach of vertical intervention programs are criticized and in their place, a bottom-to-top approach to health care delivery is advocated. In addition, the primary health care perspective gives recognition to indigenous health care systems to complement the Western allopathic health care system. From another quarter, theorists are arguing that the epidemiologic transition theory which provided the framework for understanding African mortality in the past, is actually part of a broader health transition theory which also includes a health care transition. In this theory, the overemphasis on

biomedical interventions is down-played. To understand African mortality, it is argued, we have to consider the social and cultural determinants. Also, we have to consider morbidity and its relationship to mortality. This thesis is only considering two aspects of these concerns of health transition theory, namely health care planning and the dynamic relationship between morbidity and mortality and . This is important because as Findlay (1991) pointed out, the transformations during the health transition do not uniformly facilitate both lower mortality and lower morbidity. Also as Frenk *et al.* (1991) pointed out, some heterogenous populations might be experiencing counter-transitions from high mortality to high morbidity. Hence, we need further insights on these counter-transitions.

In order to study the dynamics of morbidity and mortality, a multistate demographic model, called the dynamic model, was used. This model is however very demanding in its data requirements. One way out was to assume mathematical functions to obtain the various transition rates as was done by Pollard (1990). A better option however was to attempt to obtain the required rates by indirect means. This idea of indirectly obtaining the relevant rates for the multistate model has been applied before by Willekens (1982) and also by Doeve (1984) in different contexts.

In trying to obtain the relevant age-cause-specific death rates, one has to take two considerations into account. Firstly, African mortality is heterogenous so that very broad regionalization will be meaningless. Secondly, African mortality cannot be adequately described by a one-parameter model. Review of the different models for estimating causes

of death show that none can easily do the task at hand. There is need then to improvise. This was done by synthesizing Preston's cause of death model and an extension of the Brass logit model. Preston's model yields cause of death structure and the life table survivorship function implied by that cause of death structure. Brass general logit model was fitted to these implied life table values to obtain the α and β values. Using a separate regression model, these α and β values were used to obtain cause specific parameters from which age-cause specific death rates were finally derived.

In trying to obtain morbidity rates, one also has to take several considerations into account. Firstly, one has to deal with infectious diseases rather than degenerative diseases. Secondly, one wants to use a functional model rather than a structural one. Thirdly, one wants to make primary use of mortality, survivorship and cause of death data. Lastly since data in Africa is hard to get, one wants to make use of period data instead of longitudinal data. The only models which come close to doing this task are the morbidity models proposed at IIASA in the late 1970's. With one or two exceptions, these models deal mostly with degenerative diseases. Hence there is the need here again to improvise. This was done by using Klementiev's degenerative disease model as the starting point and this had to be reworked in order to convert it to a lethal infectious disease model. The main assumption that was relaxed for the sake of application to African data was that of no recovery. Recovery was allowed within one year after which the disease becomes the cause of death for the individual. Upon application of the model, some of the main findings were first, the importance of cause-specific death rate in determining incidence

rates and second, the little effect that recovery rates have on incidence rates.

With the data obtained from these submodels, the multisate model was used to investigate the mechanisms of the health transition; that is, the effect of changes in risk factors and that of health care technology. Changes in risk factors affect the incidence rates while changes in health care technology affect the case fatality rates. With changes in the incidence rates, case fatality rates or recovery rates are changed, the model was used to obtain estimates of life expectancies in either the illness states or the well state.

One application of the dynamic model is to use it as a tool for studying aspects of the health transition dealing with changes in mortality and morbidity. Another is in health care planning. In African countries, five-year development plans are outlined and thereafter pursued. At that stage it is crucial to have criteria to guide in decision making. In the two hypothetical examples given in the thesis, scenarios were constructed and the application of the dynamic model in aiding in decision making was outlined. In the first scenario, the option the government is faced with is the choice between spending money on nutrition and sanitation programs and that of spending money on special treatment facilities for a specific disesase. Not surprisingly, the use of the dynamic model leads one to opt for the first choice. In the second scenario, a prevalent lethal infectious disease was considered which could be treated fairly successfully in the early stage but in the later stage, only case fatality rates could be reduced. In such a situation, the dynamic model would favour more attention on reducing the case fatality rates.

7.2 Conclusion

Each aspect (theoretical, empirical or policy) addressed in this thesis has been shown to deserve research in its own right. At the theoretical level, it has been argued that research in the dynamics of morbidity and mortality fits within the framework of the health transition theory. According to Frenk *et al.* (1991), two of the mechanisms in the health transition theory are the changes in risk factors, which affect the incidence of disease, and improvement in health care technology and organization, which modify the case fatality rates. This study is an attempt to explore the relationship between these two mechanisms. At the empirical level, the thesis has shown that indirect estimation of mortality can be extended to yield age-cause-specific death rates. It has also shown that morbidity models need not stop at chronic degenerative diseases but can be successfully extended to chronic infectious diseases. It has demonstrated further that multistate demography can be used to explore the dynamics of morbidity and mortality. However, with such a web of indirect estimation methods, the rates used in the dynamic model can be subject to cumulation of errors.

At the policy level, the thesis has shown that for Africa, one need not have (or wait for) a health care planning model before simulating scenarios for health care planning. The core of health care planning is decision-making. As an alternative to modelling the health care system, one can use a model that helps in decision making. This is one practical use of the dynamic model. One must end however on a note of caution.

As often in health care planning, there are ethical issues that cannot be resolved by modelling. For example, the value of life. On what grounds does one choose between a short life of good health versus an extended life of ill health. Perhaps there is no need for decision-making in such situations, at least not on a national level. Also can the policy makers maintain neutrality in the face of vested interests of various groups, for example, politicians, urban elites and health care professionals. As health care planning is a multi-disciplinary field, the modelling can be considered as the demographers contribution, to be complemented by contributions from the medical sociologists, economists, anthropologists and religious scholars.

This has been a humble but pioneering work on the dynamics of morbidity and mortality in Africa. There is still room for further research in the following areas:

1) The age-cause-specific death rates can be refined by incorporating submodels that estimate rates for a particular set of causes of death. For example, a model that can give fairly good estimates of maternal mortality can be first used and its rates subtracted from age-specific death rates to give age-cause-specific death rates for 'other causes'. These can serve as upper limits for the rates of any other causes of death included in the 'other causes'.

2) The assumptions in the modified Klementiev's model can be further relaxed, especially those dealing with the recovery and with the disease-specific death rates. If the recovery

is to be extended beyond a year after infection, the data requirements would increase. Also the algebraic method for the derivation of the unknowns in the model would become more complicated so that the alternative matrix method should be preferred. Also, with the inclusion of recovery, the Klementiev's modified model could be treated as a multistate system. Hence the model could be developed into a multistate model instead of using it as a submodel for a main multistate model.

3) The multistate demographic model can be extended to include transitions from one disease to another and to consider mortality that is not preceded by morbidity, for example, accident mortality.

4) Instead of using a multistate model to investigate the dynamics of morbidity and mortality, a systems dynamics model (the DYNAMO software) could be used.

5) A health care model for Africa could be constructed and built into the dynamic model.

6) A specific country could be used as a case study and its health care planning policies evaluated in the light of the predictions of the dynamic model.

APPENDIX I.

Preston's regression coefficients for estimating cause of death structure (standard population used 'West' female stable population with e^0_0 =65,r=.01).

Cause of death, i	Coefficient of Correlation with death rate, all causes combined		Parameters of the simple linear regression of the form, $M_i = a_i + b_i.M$ (Standard errors of b_i in parentheses)	
	Females	Males	Females a_i b_i	Males a_i b_i
Respiratory tuberculosis	.860	.866	-.0007 .1059 (.0049)	-.0011 .1188 (.0054)
Other infectious and parasitic	.905	.880	-.0011 .1398 (.0052)	-.0015 .1458 (.0062)
Neoplasms	-.477	-.664	.0016 -.0254 (.0035)	.0024 -.0569 (.0050)
Influenza/ pneumonia/ bronchitis	.926	.938	-.0016 .2434 (.0078)	-.0026 .2831 (.0082)
Diarrhoeal	.804	.782	-.0007 .1041 (.0060)	-.0010 .1050 (.0066)
Certain chronic	.291	.308	.0003 .0165 (.0043)	.0003 .0206 (.0050)
Maternal	.890	-	-.0001 .0197	- -
Certain diseases of infancy	.765	.733	.0000 .0422 (.0028)	-.0001 .0447 (.0033)
Violence	.224	.404	.0003 .0041 (.0014)	.0006 .0232 (.0041)
Other and unknown (residual)	.892	.872	-.0013 .3307 (.0131)	-.0020 .3475 (.0153)
Sum			.0000 .9998	.0002 1.0002

Source: Preston and Nelson (1974) Table 3.

APPENDIX II

Regression equations relating life table functions to level of mortality

Dep Var.	ax	bx	r^2
Females			
log(l1/l0)	0.05356	-9.8722	0.874
		(0.2947)	
log(l5/l1)	0.05997	-7.7134	0.88
		(0.2243)	
log(l20/l5)	0.02982	-4.5864	0.906
		(0.1162)	
log(l40/l20)	0.05411	-9.9101	0.897
		(0.2642)	
log(l60/l40)	0.00036	-13.5567	0.848
		(0.4520)	
log(l80/l60)	-0.44342	-40.0032	0.867
		(1.2358)	
Males			
log(l1/l0)	0.09451	-11.5953	0.872
		(0.3482)	
log(l5/l1)	0.07836	-7.5193	0.826
		(0.2717)	
log(l20/l5)	0.03007	-3.8824	0.814
		(0.1467)	
log(l40/l20)	0.06562	-9.5712	0.856
		(0.3104)	
log(l60/l40)	0.00675	-15.8733	0.872
		(0.4794)	
log(l80/l60)	-0.6716	-34.2633	0.676
		(1.8687)	

Source: Preston (1976) Table 5.3.

APPENDIX III

Coefficients of regression equations relating log of survivorship proportions to age standardized death rates from particular causes

Dep var	Coefficients of							
	constant	TB	infec	respi	diarrhoeal	violence	others	maternal
Females								
log (l1/l0)	0.08866	-9.9285	10.8098	-14.9013	-30.5294	8.4661	-2.5468	-58.7727
log (l5/l0)	0.02004	-6.1873	-20.3757	-4.2076	-20.0184	4.3736	-2.0869	31.3303
log (l20/l5)	0.01265	-14.0136	-13.8533	0.5256	-2.3515	2.9396	-2.5863	0
log (l40/l20)	0.02142	-34.1307	-10.0233	0	-3.0013	-8.2513	-4.9133	-132.159
log (l60/l40)	0.00902	-15.1575	-15.0771	-11.0574	7.1795	-79.2064	-11.5012	-217.849
log (l80/l60)	-0.2101	25.388	-47.3659	-81.5535	10.0419	-113.396	-74.597	331.71
Males								
log (l1/l0)	0.02454	-10.7699	-1.6411	-13.5127	-31.9127	-17.4076	-1.8465	
log (l5/l0)	0.0261	-0.9512	-22.376	-2.6001	-16.8333	6.0128	-2.6772	
log (l20/l5)	0.00579	-5.3421	-16.608	-0.3039	-1.8474	-4.9403	-1.1097	
log (l40/l20)	0.025424	-31.633	-18.0716	-2.5905	-4.8679	-37.5044	-2.0634	
log (l60/l40)	0.03922	-36.1242	-10.8434	-15.5975	0	-56.8239	-15.8312	
log (l80/l60)	-0.18828	37.1778	19.9587	-65.3533	8.9527	20.0802	-98.5621	

Source: Preston (1976) Table 5.5.

APPENDIX IV.

Lopez and Hull's regression coefficients for estimating cause of death structure (standard population used 'West' female stable population with e^{o}_{0} =45, r=.02).

Cause of death, i	Parameters of the simple linear regression of the form, $M_i = a_i + b_i.M$ (Standard error of b_i in parentheses)					
	Males		Females		Both sexes	
	a_i	b_i	a_i	b_i	a_i	b_i
Respiratory tuberculosis	-.00040	.09350 (.00426)	-.00029	.09264 (.00452)	-.00034	.09254 (.00307)
Other infectious and parasitic	-.00094	.16661 (.00610)	-.00065	.16272 (.00583)	-.00076	.16153 (.00432)
Neoplasms	.00104	-.02633 (.00230)	.00079	-.01039 (.00167)	.00091	-.01770 (.00147)
Cardiovascular diseases						
Influenza/ pneumonia/ bronchitis	-.00126	.25028 (.00768)	-.00080	.2284 (.00760)	-.00100	.23696 (.00546)
Diarrhoeal	-.00062	.12598 (.00723)	-.00042	.12538 (.00707)	-.00050	.12361 (.00507)
Certain degenerative diseases	.00022	.01269 (.00245)	.00017	.01164 (.00224)	.00019	.01276 (.00167)
Complications of pregnancy	-	-	.00004	.01854 (.00071)	-.00001	.00734 (.00086)
Certain diseases of infancy	.00022	.07457 (.00486)	.00020	.06796 (.00429)	.00020	.07323 (.00317)
Violence	.00058	.02370 (.00452)	.00021	.00826 (.00134)	.00034	.02242 (.00335)
All other and unknown causes (residual)	-.00072	.27315 (.00879)	-.00041	.25952 (.00813)	-.00055	.26442 (.00598)
Total	-.00001	1.00037	.00002	1.00032	.00000	1.00042

Source: Lopez and Hull (1983) Table 2

APPENDIX V.

Lopez and Hull's regression coefficients for estimating cause of death structure during infancy and early childhood

Cause of death, i	Parameters of the simple linear regression of the form, $M_i = a_i + b_i.M$ (both sexes combined)			
	Age 0-1		Age 1-4	
	a_i	b_i	a_i	b_i
Respiratory tuberculosis	-.00016	.00572	-.00002	.02113
Other infectious and parasitic	-.00274	.11703	-.00022	.27130
Neoplasms	.00010	-.00010	.00010	-.00181
Cardiovascular diseases	-.00084	.01948	.00002	.01096
Influenza/ pneumonia/ bronchitis	-.00217	.20369	-.00007	.24409
Diarrhoeal	-.00255	.18998	-.00017	.19509
Certain degenerative diseases	.00002	.00151	.00003	.00695
Certain diseases of infancy	.01029	.20779	-	-
Violence	.00032	.00800	.00039	.01050
All other and unknown causes (residual)	-.00226	.24690	-.00006	.24128
Total	.00001	1.00000	.00000	.99949

Source: Lopez and Hull (1983) Table 4

APPENDIX VI

Multiple regression equations for relating Brass' α_i and β_i of TB and 'All Other Causes ' (OTH) to α and β for 'All Causes'

$$Y_i = a_i + c_{1i}*\beta_i + c_{2i}*\alpha_i$$

i	Y_i	a_i	c_{1i}	c_{2i}	R^2
1	β_1 (TB)	-0.6509	1.5330	-0.1352	0.843
2	α_1 (TB)	-2.6129	2.2352	1.6796	0.912
3	β_2 (OTH)	-.0010	1.0075	-.0942	0.999
4	α_2 (OTH)	-.0724	.0824	.8700	0.999

Source: The α and β values were obtained from Chilean mortality data over the period 1940-1964 as recorded in Preston *et al.* (1976).

Technical note: As the data obtained was in five year age groups, the single year data was obtained using Beers' interpolation coefficients. This task was accomplished with the BEERS program of the MCPDA package; interpolation attempt using the Heligman-Pollard formula did not prove successful. Having obtained the data in single years, Brass' logit model was then fitted to the data using the program BRASSCH.FOR (with source listing given in the Appendix VIII). Three standards were used, all taken from the 1964 Chilean male data; general mortality rates, cause specific death rate for TB and cause specific death rate for 'all others.' Finally because of the limitation of the logit model in handling zero mortality rates, fitting was done for ages greater than 20 year where cause specific mortality rates were all greater than zero.

APPENDIX VII

Derivation of Unknown Variables for Klementiev's Model Modified

Substituting (14) in equations (15) and (22), one obtains

$$p_2 = (h_2 + \mu_1 p_1 \sigma_1) + \mu_1 p_1 (1 - \beta_{11}) \tag{40}$$

$$p_2 \alpha_2 = (h_2 + \mu_1 p_1 \sigma_1)\theta_2 + \mu_1 p_1 d_{11} \tag{41}$$

Multiplying (40) by Θ_2 and subtracting from (41), one obtains that

$$p_2(\alpha_2 - \theta_2) = \mu_1 p_1 (d_{11} - a_{11}\theta_2) \tag{42}$$

hence,

$$\mu_1 = \frac{p_2 \tau_2}{p_1 (d_{11} - a_{11}\theta_2)} \tag{43}$$

Substituting (43) in (40), one obtains that

$$p_2 = h_2 + (\sigma_1 + a_{11}).\frac{p_2 \tau_2}{d_{11} - a_{11}\theta_2} \tag{44}$$

Hence

$$h_2 = p_2.\{1 - \frac{(\sigma_1 + a_{11})\tau_2}{d_{11} - a_{11}\theta_2}\} \tag{45}$$

$$= p_2.\{\frac{d_{11} - \sigma_1 \tau_2 - a_{11}\alpha_2}{d_{11} - a_{11}\theta_2}\} \tag{46}$$

In a similar manner, to obtain μ_{i-1} one uses equations (20) and (27). Rewriting (20) and (27),

$$p_i = (h_i + \mu_{i-1} h_{i-1} \sigma_{i-1}) + \mu_{i-1} h_{i-1} a_{i-1,1} + F_i \,, \; i = 3,...N \tag{47}$$

Multiplying (47) by Θ_i and subtracting from (48) one obtains that.

Multiplying (47) by Θ_i and subtracting from (48) one obtains that.

$$p_i(\alpha_i - \theta_i) = \mu_{i-1}h_{i-1}d_{i-1,1} - (\mu_{i-1}h_{i-1}a_{i-1,1})\theta_i + G_i - \theta_i F_i \tag{49}$$

$$p_i\tau_i = \mu_{i-1}h_{i-1}(d_{i-1,1} - a_{i-1,1}\theta_i) + U_i \tag{50}$$

Hence.

$$\mu_{i-1} = \frac{p_i\tau_i - U_i}{h_{i-1}(d_{i-1,1} - a_{i-1,1}\theta_i)} \tag{51}$$

Substituting for (51) in (47) gives.

$$p_i = h_i + \frac{(p_i\tau_i - U_i)(\sigma_{i-1} + a_{i-1,1})}{(d_{i-1,1} - a_{i-1,1}\theta_i)} + F_i \tag{52}$$

Hence.

$$h_i = p_i - \frac{(p_i\tau_i - U_i)(\sigma_{i-1} + a_{i-1,1})}{(d_{i-1,1} - a_{i-1,1}\theta_i)} - F_i \tag{53}$$

$$h_i = \frac{\{p_i(d_{i-1,1} - a_{i-1,1}(\theta_i + \tau_i) - \tau_i\sigma_{i-1}) + U_i(\sigma_{i-1} + a_{i-1,1}) - F_i(d_{i-1,1} - a_{i-1,1}\theta_i)\}}{(d_{i-1,1} - a_{i-1,1}\theta_i)} \tag{54}$$

Substituting for U_i in (54) and simplifying, one obtains

$$h_i = \frac{p_i(d_{i-1,1} - a_{i-1,1}\alpha_i - \tau_i\sigma_{i-1}) + (\sigma_{i-1} - a_{i-1,1})G_i - (\theta_i\sigma_{i-1} + d_{i-1})F_i}{d_{i-1,1} - a_{i-1,1}\theta_i} \tag{55}$$

Source: Rajulton and Bah (1991)

APPENDIX VIII

Source listing of FORTRAN programs used in the thesis

```
C ************************************************************
C PROGRAM NAME- CAUSE.FOR
C
C DESCRIPTION- PROGRAM TO COMBINES THE VARIOUS PROGRAMS WRITTEN
C    SO AS SOLVE THE PROBLEMS SET IN CHAPTER 3
C
C PROGRAMMER- SULAIMAN BAH
C
C DATE STARTED- DECEMBER 16 1991
C
C+++++++++++++++ VARIABLE DEFINITIONS AND DECLARATIONS++++++++
C DEFN  AA(I,J)- REGRESSION COEFF;I=GENDER, J= ai COEFFICIENTS
C     AB(I,J)- REGRESSION COEFF;I=GENDER, J= bi COEFFICIENTS
C     RMORT(I)-CRUDE MORTALITY RATE
C     BA(I,J)- REGRESSION COEFF;I=AGE (1-AGE 0-1, 2- AGE 1-4,
C           3- 5+)  J= ai COEFFICIENTS
C     BB(I,J)- REGRESSION COEFF;I=AGE (1-AGE 0-1, 2- AGE 1-4,
C           3- 5+)  J= bi COEFFICIENTS,
C     CA(I,J)- CONSTANT REGRESSION COEFF:I=GENDER, J= BROAD AGE
C           GROUPS (6)
C     CB(I,J,K)- REGRESSION COEFF:I=GENDER, J= BROAD AGE
C          GROUPS (6),K=REDUCED CAUSES OF DEATH (7)(MALES, 6)
C     LOGAGE(I,J) -LOG OF AGE GROUPS,I= GENDER (MALE=1);
C           J=BROAD AGE GROUPS (6)
C     AGEXN(I,J)- LIFE TABLE SURVIVORSHIP PROPORTIONS
C           OBTAINED AS EXP(LOGAGE(I,J))
C     CAUSE(I,J)-I=GENDER,(1-MALE,2 FEMALE,3 BOTH) J=CAUSES (11)
C     CCSR(I,K)-CRUDE CAUSE OF DEATH RATE; I= GENDER
C           K= REDUCED CAUSES OF DEATH (7) (6 FOR MALES)
C     ACAUSE(I,J) CAUSE STRUCTURE BY AGE FOR BOTH GENDERS, I=
C                AGE GROUP (1: 0-1, 2:1-4), J- CAUSES
C     RLXS(I)- lx FROM NEW STANDARD LIFE TABLE
C     RSLX(I) npx FROM STANDARD FOR SPECIAL AGE GROUPINGS
C           1: O-1, 2:1-4, 3:5-20, 4:20-40, 5:40-60,6:60-80
C     AQCAUSE(I,J) COMPUTED  nqx VALUES FROM CENTRAL DEATH
C           RATES
C     APCAUSE(I,J) COMPUTED  npx VALUES FROM nqx
C           POPULATION
C     RLOGTPX(I,J) LOGIT VALUES OF npx OF OBSERVED POPULATION
C           I GENDER : J- AGE  (1:0-1, 2:1-4, 3:5-20,
C           4:20-40, 5:40-60,  6:60-80)
C     RSLOGTPX(J) LOGIT VALUES OF npx OF STANDARD POPULATION
C           FOR SPECIAL AGE GROUPING (6) AS ABOVE
C     ALPHA(I)- BRASS'S ALPHA VALUES
C     BETA(I)-BRASS'S BETA VALUES
C     AGEG(I)- CHARACTER VARIABLE FOR LOG npx
C     AGEG2(I)- CHARACTER VARIABLE FOR npx
C     CNAMES(I)- CHARACTER VARIABLE NAMES OF CAUSES
```

```
C       RECT(I)-MEASURE OF RECTANGULARITY FROM ANSON
C    RMORT(I)- INPUTED MORTALITY RATES 1- CRUDE DEATH RATE/000
C          2 IMR /000 LIVE BIRTHS 3- CHILD MORTALITY RATE
C          DEATHS IN AGE 1-4 /000 POPULATION
C INPUT DATA FORMAT FOR COEFS - MALE AI FOLLOWED BY MALE BI FOR
C
C CAUSE I THEN FOR THE CAUSES AFTER WHICH FEMALE VALUES FOLLOW
C FOR AGECOEFS. AI FOR AGE 0-1 FOLLOWED BY BI FOR AGE 0-1 AFTER
C WHICH VALUES FOR 1-4 FOLLOW.
CINPUT DATA FORMAT FOR AGECAUSE COEFFICIENTS-MALE A(I) FOLLOWED
C BY MALE CB(I,J,K) FOR CAUSE THEN FOR THE DIFFERENT AGE GROUPS
C AFTER  WHICH FEMALE VALUES FOLLOW
      PROGRAM COMB5
      DIMENSION CAUSE(3,11), AA(3,11), AB(3,11), BA(3,10),
     1 BB(3,10),RMORT(3), ACAUSE(3,11), PERCAUSE(2,11),
     1 AGE(21), RLXS(21), RLX(2,6),
     1 RSLOGTPX(6),RLOGTPX(2,6), TCAUSE(2),
     1 ALPHA(2), BETA(2), AGELOG(2,6), CA(2,6), CB(2,6,7),
     1 CCSR(2,7), AGEXN(2,6),TOTPER(2), GCAUSE(3),TOTPERG(3),
     1 PERGCAUSE(3,11),RECT(2)
      CHARACTER AGEG(6)*14, AGEG2(6)*10, CNAMES(11)*14
C++++++++END OF VARIABLE DEFINITIONS AND DECLARATIONS++++++++
C ==========READING FROM INPUT FILE AND REWRITING==========
      OPEN (UNIT=12,FILE= 'STRUCT1.OUT')
      OPEN (UNIT=11,FILE= 'AFCAUSE1.OUT')
      OPEN (UNIT=8,FILE= 'LOGITS1.OUT')
      OPEN (UNIT=7,FILE= 'ALLDATA.DAT')
      OPEN (UNIT=6,FILE= 'AFMORT1.DAT')
C READ COEFFS FOR CRUDE CAUSE SPECIFIC RATES
C     WRITE (12, 3)
C   3 FORMAT (2X, 'REPRINTING THE INPUT DATA AND COEFFICIENTS')
      DO 10 J=1,11
      READ (7, *, end=20) AA(1,J), AB(1,J),AA(2,J), AB(2,J),
     1 AA(3,J), AB(3,J)
C     WRITE (12, 9) AA(1,J), AB(1,J),AA(2,J), AB(2,J),
C    1 AA(3,J), AB(3,J)
C  9  FORMAT (6F9.5)
   10 CONTINUE
C READ COEFFS FOR LIMITED AGE CAUSE-SPECIFIC RATES
   20 DO 30 J=1,10
      READ (7, *, end=40) BA(1,J), BB(1,J),BA(2,J), BB(2,J)
C     WRITE (12, 21) BA(1,J), BB(1,J),BA(2,J), BB(2,J)
C  21 FORMAT (4F9.5)
   30 CONTINUE
C READ COEFFS RELATING LOG (lx+n/lx) TO CRUDE CAUSE SPECIFIC
C RATES
   40 DO 60 J=1,6
      READ (7,*, end=61) CA(1,J), CB(1,J,1), CB(1,J,2),
     1 CB(1,J,3), CB(1,J,4), CB(1,J,5), CB(1,J,6)
C     WRITE (12, 45) CA(1,J), CB(1,J,1), CB(1,J,2),
C    1 CB(1,J,3), CB(1,J,4), CB(1,J,5), CB(1,J,6)
C  45 FORMAT (7F10.5)
```

```
   60 CONTINUE
   61 DO 64 J=1,6
      READ (7,*, end=65) CA(2,J), CB(2,J,1), CB(2,J,2),
     1 CB(2,J,3), CB(2,J,4), CB(2,J,5), CB(2,J,6), CB(2,J,7)
C     WRITE (12, 63)  CA(2,J), CB(2,J,1), CB(2,J,2),
C    1 CB(2,J,3), CB(2,J,4), CB(2,J,5), CB(2,J,6), CB(2,J,7)
C  63 FORMAT (8F10.5)
   64 CONTINUE
C READ AGE AND lx OF NEW STANDARD LIFE TABLE OF EWBANK ET AL.
   65 DO 70 I=1,21
      READ (7, *, end=80) AGE(I), RLXS(I)
   70 CONTINUE
C READ CHARACTER STRINGS FOR AGE GROUPING
   80 DO 90 J=1,6
      READ (7, *, end=100) AGEG(J), AGEG2(J)
C     WRITE (12, *) AGEG(J), AGEG2(J)
   90 CONTINUE
  100 DO 110 J=1,11
      READ (7, *, end=120) CNAMES(J)
C     WRITE (12, *) CNAMES(J)
  110 CONTINUE
      READ (6, *) NY
      WRITE (8, 114) NY
  114 FORMAT (I5)
  115 READ (6, *) N, RMORT(1), RMORT(2),RMORT(3)
      IF (N .EQ. 99) GOTO 600 ! CLOSE FILES
      WRITE (12, 118) NY,N
      WRITE (11, 118) NY,N
  118 FORMAT (/,6X,'ESTIMATIONS USING',I5, ' DATA, FOR AFRICAN COUNTRY,
     1#', I3,/)
      WRITE (12, 119) RMORT(1), RMORT(2),RMORT(3)
      WRITE (11, 119) RMORT(1), RMORT(2),RMORT(3)
  119 FORMAT (/,6X, 'CRUDE DEATH RATE/000=', F5.4,/,
     16x, 'INFANT MORTALITY RATE/000= ',F5.4,/,
     16x, 'CHILD MORTALITY RATE /000 (AGE 1-4)=',F5.4,/)
C =====END OF READING FROM INPUT FILE AND REPRINTING==========
C ========== COMPUTATION BLOCK================================
  120 DO 220 I=1,3
      DO 220 J=1,11
      CAUSE(I,J)=AA(I,J)+AB(I,J)*RMORT(1)
      IF (CAUSE(I,J) .GE. 0) GOTO 220
      PRINT *, 'FOR COUNTRY #',N, 'CAUSE',I, J, 'IS NEGATIVE'
  220 CONTINUE
      WRITE (12, 221)
  221 FORMAT (12X, 'CAUSE OF DEATH STRUCTURE (ALL AGES)',/)
      WRITE (12,222)
  222 FORMAT (5X,'CAUSES      MALES         FEMALES         BOTH ',/)
      DO 224 J=1,11
      WRITE (12,223) CNAMES(J), CAUSE(1,J), CAUSE(2,J), CAUSE(3,J)
  223 FORMAT (A14,4X, F8.6,4X, F8.6,4X, F8.6)
  224 CONTINUE
C ESTIMATION OF PERCENTAGE OF TOTAL DEATHS IN AGE GROUP
```

```
      GCAUSE(1)=0 ! TOTAL DEATHS FOR MALES
      GCAUSE(2)=0 ! TOTAL DEATHS FOR FEMALES
      GCAUSE(3)=0 ! TOTAL DEATHS FOR BOTH
      TOTPERG(1)=0
      TOTPERG(2)=0
      TOTPERG(3)=0
      DO 227 I=1,3
      DO 225 J=1,11
      GCAUSE(I)=GCAUSE(I)+CAUSE(I,J)
  225 CONTINUE
      DO 226 J=1,11
      PERGCAUSE(I,J)=CAUSE(I,J)*100/GCAUSE(I)
      TOTPERG(I)=TOTPERG(I)+PERGCAUSE(I,J)
  226 CONTINUE
  227 CONTINUE
      WRITE (12,230)
      WRITE (11,230)
  230 FORMAT (/,12X,'CAUSE OF DEATH STRUCTURE '/,
     1 5X,'(PERCENTAGE OF DEATHS FOR ALL AGE GROUPS)'/)
      WRITE (12,232)
      WRITE (11,232)
  232 FORMAT (6X,'CAUSES              MALES    FEMALES     BOTH'/)
      DO 236 J=1,11
      WRITE (12,234) CNAMES(J), PERGCAUSE(1,J), PERGCAUSE(2,J),
     1 PERGCAUSE(3,J)
      WRITE (11,234) CNAMES(J), PERGCAUSE(1,J), PERGCAUSE(2,J),
     1 PERGCAUSE(3,J)
  234 FORMAT (6X, A14,4X, F6.2,4X,F6.2,4X,F6.2)
  236 CONTINUE
      WRITE (12,238) TOTPERG(1), TOTPERG(2), TOTPERG(3)
      WRITE (11,238) TOTPERG(1), TOTPERG(2), TOTPERG(3)
  238 FORMAT (/,10X,'TOTAL',8X, F7.2,3X,F7.2, 3X,F7.2)
C COMPUTE CAUSE STRUCTURE FOR 3 BROAD AGE GROUPS. TOTAL NUMBER OF
C CAUSES IS 10 (MATERNAL CAUSES IS EXCLUDED FOR YOUNGER AGES. FOR
C OLDER,5+, IT IS INCLUDED UNDER ALL OTHERS)
      DO 240 I=1,2
      DO 240 J=1,10
      ACAUSE(I,J)=BA(I,J)+BB(I,J)*RMORT(I+1)
      IF (ACAUSE(I,J) .GE. 0) GOTO 240
      PRINT *, 'FOR COUNTRY #',N, 'ACAUSE', I, J, 'IS NEGATIVE'
  240 CONTINUE
      WRITE (12,241)
  241 FORMAT (/,12X, 'CAUSE OF DEATH STRUCTURE '/,
     1 5X,'(CENTRAL DEATH RATES BY AGES FOR BOTH)'/)
      WRITE (12,242)
  242 FORMAT (6X,'CAUSES        0-1          1-4 '/)
      DO 244 J=1,10
      WRITE (12,243) CNAMES(J), ACAUSE(1,J), ACAUSE(2,J)
  243 FORMAT (A14,4X, F9.7,4X, F9.7)
  244 CONTINUE
C ESTIMATION OF PERCENTAGE OF TOTAL DEATHS IN AGE GROUP
C GIVE TITLE
```

```
      TCAUSE(1)=0 ! TOTAL DEATHS FOR 0-1
      TCAUSE(2)=0 ! TOTAL DEATHS FOR 1-4
      TOTPER(1)=0
      TOTPER(2)=0
      DO 255 I=1,2
      DO 250 J=1,10
      TCAUSE(I)=TCAUSE(I)+ACAUSE(I,J)
  250 CONTINUE
      DO 253 J=1,10
      PERCAUSE(I,J)=ACAUSE(I,J)*100/TCAUSE(I)
      TOTPER(I)=TOTPER(I)+PERCAUSE(I,J)
  253 CONTINUE
  255 CONTINUE
      WRITE (12,256)
      WRITE (11,256)
  256 FORMAT (/,12X, 'CAUSE OF DEATH STRUCTURE '/,
     1 5X,'(PERCENTAGE OF DEATHS IN EACH AGE GROUP FOR BOTH)',/)
      WRITE (12,257)
      WRITE (11,257)
  257 FORMAT (/,6X,'CAUSES                0-1     -4 ',/)
      DO 260 J=1,10
      WRITE (12,258) CNAMES(J), PERCAUSE(1,J), PERCAUSE(2,J)
      WRITE (11,258) CNAMES(J), PERCAUSE(1,J), PERCAUSE(2,J)
  258 FORMAT (6X, A14,4X, F6.2,4X,F6.2)
  260 CONTINUE
      WRITE (12,270) TOTPER(1), TOTPER(2)
      WRITE (11,270) TOTPER(1), TOTPER(2)
  270 FORMAT (/,10X,'TOTAL',8X, F7.2,3X,F7.2)
C FIRST MAKE THE CRUDE CAUSE SPECIFIC DEATH RATES (CAUSE)
C CORRESPOND TO THE REDUCED CAUSES (CCSR) USED IN PRESTON'S
C (1974) MODEL
      DO 415 I=1,2
      CCSR(I,1)=CAUSE(I,1) !TUBERCULOSIS
      CCSR(I,2)=CAUSE(I,2) !INFECTIOUS AND PASITIC
      CCSR(I,3)=CAUSE(I,5) !RESPIRATORY
      CCSR(I,4)=CAUSE(I,6)+ CAUSE(I,10) !DIA+CERT.INF
      CCSR(I,5)=CAUSE(I,8) !VIOLENCE
      CCSR(I,6)=CAUSE(I,3)+CAUSE(I,4)+CAUSE(I,7)+CAUSE(I,9)!ALL OTHERS
  415 CONTINUE
      CCSR(2,7)=CAUSE(2,11)! MATERNAL CAUSES
C COMPUTATION FOR LOG OF lx+n/lx VALUES USING CRUDE CAUSE
C SPECIFIC DEATH RATES AND REGRESSION COEFFICIENTS.
      DO 430 J=1,6
      CR=0
      DO 420 K=1,6
      CR=CR+CB(1,J,K)*CCSR(1,K)
  420 CONTINUE
      AGELOG(1,J)=CA(1,J)+CR
      AGEXN(1,J)=EXP(AGELOG(1,J))
  430 CONTINUE
      RECT(1)=AGEXN(1,5)/(AGEXN(1,4)* AGEXN(1,4))! 20P40/(20P20^2)
  440 DO 443 J=1,6
```

```
      CR=0
      DO 441 K=1,7
      CR=CR+CB(2,J,K)*CCSR(2,K)
  441 CONTINUE
      AGELOG(2,J)=CA(2,J)+CR
      AGEXN(2,J)=EXP(AGELOG(2,J))
  443 CONTINUE
      RECT(2)=AGEXN(2,5)/(AGEXN(2,4)* AGEXN(2,4))! 20P40/(20P20^2)
C ESTIMATE ALPHA AND BETA (ALL USING CAUSES COMBINED) FROM THE
C PAIRS OF RLOGTPX(I,J) AND RSLOGTPX(J) USING LEAST SQUARES
C
C OBTAIN lx FROM CHAIN SURVIVORSHIP
      WRITE (12, 444)
  444 FORMAT (/, 'LIFE TABLE lx AND lx+n/lx VALUES AT SELECTED AGES'/,
     !'(INCORPORATING INFORMATION ON CAUSE OF DEATH STRUCTURE)'//,
     1 '                    MALES              FEMALES      '//,
     1 'x-x+n        lx+n/lx    lx+n     lx+n/lx      lx+n'/)
      DO 447 I=1,2
      DO 447 J=1,6
      AM=1
      DO 445 M=1,J
      AM=AGEXN(I,M)*AM
  445 CONTINUE
      RLX(I,J)=AM
      RLOGTPX(I,J)=.5*LOG((1-RLX(I,J))/RLX(I,J))
  447 CONTINUE
      DO 450 J=1,6
      WRITE (12,448) AGEG2(J),AGEXN(1,J),RLX(1,J), AGEXN(2,J),RLX(2,J)
  448 FORMAT (A10,3X,F7.5,3X,F7.5,3X, F7.5,3X,F7.5)
  450 CONTINUE
C CALCULATING LOGIT OF STANDARD
C FOR THE STANDARD, MAKE THE SELECTED AGES CORRESPOND
      RSLOGTPX(1)=.5*LOG((1-RLXS(2))/RLXS(2))
      RSLOGTPX(2)=.5*LOG((1-RLXS(6))/RLXS(6))
      RSLOGTPX(3)=.5*LOG((1-RLXS(9))/RLXS(9))
      RSLOGTPX(4)=.5*LOG((1-RLXS(13))/RLXS(13))
      RSLOGTPX(5)=.5*LOG((1-RLXS(17))/RLXS(17))
      RSLOGTPX(6)=.5*LOG((1-RLXS(21))/RLXS(21))
  454 WRITE (12,455)
  455 FORMAT (/,8X,'ESTIMATE RECANGULARITY AND BRASS ALPHA AND BETA'/,
     1'FROM lx VALUES ESTIMATED USING INFORMATION ON CAUSE STRUCTURE'/,
     1'                (1-MALES; 2-FEMALES)')
      WRITE (12, 456)
  456 FORMAT (/,4X,'COUNTRY   GENDER     ALPHA     BETA RECTANGULARITY')
      SX=0
      SXX=0
      DO 457 J=1,6
      SX=SX+RSLOGTPX(J)
      SXX=SXX+(RSLOGTPX(J)*RSLOGTPX(J))
  457 CONTINUE
      DO 480 I=1,2
      SY=0
```

```
      SXY=0
      DO 458 J=1,6
      SY=SY+RLOGTPX(I,J)
      SXY=SXY+(RLOGTPX(I,J)*RSLOGTPX(J))
  458 CONTINUE
      BETA(I)=(6*SXY-(SX*SY))/(6*SXX-(SX*SX))
      ALPHA(I)=(SY-(BETA(I)*SX))/6
      WRITE (12,470) N, I, ALPHA(I), BETA(I), RECT(I)
      WRITE (8,460) N, I, ALPHA(I), BETA(I) , RECT(I)
  460  FORMAT (I3,2X,I2,2X,F6.3,6X,F6.3,6X,F6.3)
  470  FORMAT (8X,I3,2X,I2,8X,F6.3,6X,F6.3,7X,F6.3)
  480  CONTINUE
C ========END OF COMPUTATION BLOCK==========================
      GOTO 115
  600 CLOSE (6)
      CLOSE (7)
      CLOSE (8)
      CLOSE (11)
      CLOSE (12)
      END
```

```
C*********************************************************************
C PROGRAM NAME- MORBID.FOR
C PROGRAMMER- SULAIMAN BAH
C THE PROGRAM INCLUDES FRAGMENTS FROM KLEMENTIEV'S PROGRAM
C DATE STARTED FEB/92; REVISED SEPTEMBER/92
C THIS PROGRAM ESTIMATES MORBIDITY RATES FOR LETHAL INFECTIOUS
C DISEASES- FURTHER ASSUMES THAT DISEASE ACTS FOR ONLY FIVE YEARS
C*********************************************************************
C VARIABLE DEFINITIONS
C*********************************************************************
C P- GENERAL POPULATION
C H- HEALTHY POPULATION
C REC- RECOVERY RATE
C A(I,J)- PI(1-BET)
C AMU- INCIDENCE RATE
C DTIL- ALL CAUSE DEATH RATE
C TAU- INFECTIOUS DISEASE DEATH RATE
C D(I,J)- DISEAS SPECIFIC DEATH RATE- CONTRACTED THE DISESES
C         IN THE ith AGE GROUP j YEARS AGO.
C
C*********************************************************************
C              SUBROUTINE
C*********************************************************************
      SUBROUTINE DKS(C,D,N,I,J)
C     SHORT PROGRAM TO PRODUCE D(I,J) VALUE GIVEN D(I,1) VALUES
      DIMENSION C(10),D(20,20)
      OPEN (UNIT=7,FILE='C:\WATFOR77\PROGRAMS\DKS14.DAT')
      READ (7,*) C(1),C(2),C(3),C(4)
      READ (7,*) D(1,1),D(2,1),D(3,1),D(4,1),D(5,1),D(6,1),D(7,1),
     1 D(8,1),D(9,1),D(10,1)
      N=10
      DO 50 J=1,4
      DO 50 I=1,N-1
      IF (I .EQ. (N-1)) D(N,J)=D(N,1)
      D(I,J+1)=C(J)*D(I+1,J)
   50 CONTINUE
C     DO 53 I=1,N
C     DO 53 J=1,N
C     IF ((I+J) .GT. N) D(I,J)=0
C 53  CONTINUE
      RETURN
      END
C*********************************************************************
C       MAIN PROGRAM
C*********************************************************************
      PROGRAM MORB4
      DIMENSION P(10), H(10),REC(10),AMU(10),TAU(10),
     1 THE(10),BET(10,10), A(10,10),ALPH(10), U(10),
     1 F(10),G(10), AGE(11),D(20,20),C(10)
      OPEN (UNIT=4, FILE='C:\WATFOR77\PROGRAMS\MORBID14.DAT')
      OPEN (UNIT=11, FILE='C:\WATFOR77\PROGRAMS\PREVE14.OUT')
      DO 60 I=1,10
```

```
      READ (4,*) AGE(I), P(I),TAU(I),ALPH(I),REC(I)
   60 CONTINUE
      READ (4,*) C(1),C(2),C(3),C(4)
      CALL DKS(C,D,10,IJ)
C******** COMPUTATION BLOCK************************************
C GIVEN AND DERIVED VALUES FOR MORTALITY AND MORBIDITY
      N=10
      DO 150 I=1,N
         THE(I)=ALPH(I)-TAU(I)
         BET(I,1)=REC(I)+D(I,1)
  150 CONTINUE
      DO 160 I=1,N-1
      DO 160 J=2,5
         BET(I,J)=D(I,J)
  160 CONTINUE
C OBTAIN A VALUES
      DO 200 I=1,N-1
         A(I,1)= 1-BET(I,1)
      DO 200 J=2,5
         A(I,J)=A(I,J-1)*(1-BET(I,J))
  200 CONTINUE
C OBTAINING UNKNOWN VALUES FOR THE FIRST 2 AGE GROUPS
      H(1)=P(1)
      AM1= P(2)*TAU(2)
      AM2= P(1)*(D(1,1)-(A(1,1)*THE(2)))
      AMU(1)= AM1/AM2
      AH1=P(2)*(D(1,1)-(REC(1)*TAU(2))-(A(1,1)*ALPH(2)))
      AH2=(D(1,1)-(A(1,1)*THE(2)))
      H(2)=AH1/AH2
C WITHIN ONE LOOP CALCULATE Fi AND Gi'S THAT CAN BE USED IN
C CALCULATING Hi AND AMU i-1
      DO 230 I=3,N
         FT=0
         GT=0
            DO 220 J=2,I-1
               IF (J .GE. 6) GOTO 220
               FT=FT+(AMU(I-J)*H(I-J)*A(I-J,J))
               GT=GT+(AMU(I-J)*H(I-J)*A(I-J,J)*D(I-J,J))
  220          CONTINUE
         F(I)=FT
         G(I)=GT
         U(I)=G(I)-(F(I)*THE(I))
         H1=P(I)*(D(I-1,1)-(A(I-1,1)*ALPH(I))-(TAU(I)*REC(I-1)))
         H2=G(I)*(REC(I-1)-A(I-1,1))
         H3=F(I)*((THE(I)*REC(I-1))+D(I-1,1))
         H4=(D(I-1,1)-(A(I-1,1)*THE(I)))
         H(I)=(H1+H2-H3)/H4
         AMU1=(P(I)*TAU(I))-U(I)
         AMU2=H(I-1)*(D(I-1,1)-(A(I-1,1)*THE(I)))
         AMU(I-1)=AMU1/AMU2
  230 CONTINUE
C*************************************************************************
```

```
C            WRITE BLOCK
C*************************************************************************
     WRITE (11,231)
  231 FORMAT ('INPUT DATA FOR AGE (I), CAUSE SPECIFIC DEATH RATE (TAUi)'
    1 /,'DEATH RATE FOR ALL CAUSES (ALPH i), AND RECOVERY RATE (RECi)'
    1//,'  i       Pi     Tau i    Alph i       Rec i'/)
     DO 232 I=1,10
          WRITE (11,234) I, P(I),TAU(I),ALPH(I),REC(I)
  232 CONTINUE
  234 FORMAT (I4,3X,F8.0,3X,F7.5,3X,F7.5,3X,F7.5)
     WRITE (11,235)
  235 FORMAT (/,'INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT'/,
    1'                  D(I,J)=C(I)*D(I+1,J-1)'/)
     WRITE (11,240)
  240 FORMAT ('D(I,1)',3X,'D(I,2)',3X,'D(I,3)',3X,'D(I,4)',3X,'D(I,5)',
    1/)
     WRITE (11,245) C(1),C(2),C(3),C(4)
  245 FORMAT (F7.5,2X,F7.5,2X,F7.5,2X,F7.5/)
     DO 255 I=1,N-1
          WRITE (11,260) D(I,1),D(I,2),D(I,3),D(I,4),D(I,5)
  255 CONTINUE
  260 FORMAT (F7.6,2X,F7.6,2X,F7.6,2X,F7.6,2X,F7.6)
     WRITE (11,261)
  261 FORMAT (/,'OUTPUT FOR MORBID4.FOR USING DATA FROM MORBID .DAT')
     WRITE (11,262)
  262 FORMAT (/,'I        Pi        Hi       AMUi'/)
     DO 265 I=1,N-1
          WRITE (11,270) I, P(I), H(I),AMU(I)
  265 CONTINUE
  270 FORMAT (I2,2X,F10.3,2X,F10.3,2X,F9.3)
     END
```

```
C PROGRAM NAME:DYNAMIC.FOR
      PROGRAM MAINP
      DIMENSION P(80), H(80),REC(80),AMU(80),TAU(80),AGE(80),ALPH(80),
     1 D(80,5),C(4),QQ(3,80),THE(80),DD(80,5),HO(80),AMUO(80),
     1 RECO(80)
      CHARACTER*10 CHAR1
      CHARACTER*8 CHAR2
      OPEN (UNIT=6,FILE='FILE6.DAT')
      OPEN (UNIT=7,FILE='CAUSE10.DAT')
      OPEN (UNIT=11,FILE='MORBID.OUT')
      OPEN (UNIT=40,FILE='TABLE40.OUT')
C*******************************************************************************
C                    SUBPROGRAM 2 (MORBID)
C*******************************************************************************
      DO 60 I=1,80
      READ (6,*) AGE(I),P(I)
   60 CONTINUE
   62 READ (7,63) CHAR1,IY,CHAR2
   63 FORMAT (A10,I4,A8)
      DO 65 I=15,78
      READ (7,*) AGE(I),QQ(2,I),QQ(3,I)
      QQ(1,I)=QQ(2,I)+QQ(3,I)
   65 CONTINUE
C TAU- CAUSE, ALPH-ALL CAUES, THE-ALL OTHERS
      DO 70 I=15,78
      ALPH(I)=QQ(1,I)
      TAU(I)=QQ(2,I)
      THE(I)=QQ(3,I)
   70 CONTINUE
      XR1=.005
      XRO1=.0001
      XD1=350.0
      XDD1=40.0
      CALL VALUES(XR1,XRO1,REC,RECO,QQ,XD1,XDD1,D,DD)
C     SUBROUTINE VALUES(XR,XRO,REC,RECO,QQ,XD,XDD,D,DD)
C THE FIRST 2 ARGUMENTS ARE THE CONSTANTS FOR THE RECOVERY RATES, THE
C NEXT 2 ARE THE OUTPUT RECOVERY RATES FOLLOWED BY QQ DEATH RATES.
C THE THIRD PAIR ARE THE MULTIPLIERS FOR THE CASE FATALITY RATE FOR
C TB AND ALL OTHERS FOLLOWDD BY THE OUTPUT CASE FATALITY RATES
      N=78
C C VALUES ARE USED IN THE DKS SUBROUTINE TO OBTAIN DISTRIBUTION
C OF D(I,J) VALUES
      C(1)=.2
      C(2)=.2
      C(3)=.2
      C(4)=.2
      CALL DKS(C,D,78,I,J)
      CALL MORBID(78,P,TAU,THE,ALPH,REC,D,H,AMU)
      WRITE (11,220) CHAR1,CHAR2,IY
  220 FORMAT ('INPUT AND OUTPUT DATA FOR A STANDARD POPULATION
WHICH'/
     1'EXPERIENCES THE MORTALITY OF ',A10,'FOR  ',A8,', ',I4,/)
```

```
      WRITE (11,225) XR1,XRO1,XD1,XDD1
  225 FORMAT ('PARAMETERS ASSUMED:'/,
     1'VALUES('F7.4,2X,F7.4,2X,'REC, RECO, QQ',2X,F6.2,2X,F6.2,2X,'D,
     1 DD)'/)
      WRITE (11,231)
  231 FORMAT ('INPUT DATA FOR AGE (I), CAUSE SPECIFIC DEATH RATE (TAUi)'
     1 /,'DEATH RATE FOR ALL CAUSES (ALPH i), AND RECOVERY RATE (RECi)'
     1//,'  i       Pi     Tau i    Alph i       Rec i'/)
  232 DO 233 I=15,N
      WRITE (11,234) I, P(I),TAU(I),ALPH(I),REC(I)
  233 CONTINUE
  234 FORMAT (I4,3X,F8.0,3X,F7.5,3X,F7.5,3X,F7.5)
      WRITE (11,235)
  235 FORMAT (/,'INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT'/,
     1'                D(I,J)=C(I)*D(I+1,J-1)'/)
      WRITE (11,240)
  240 FORMAT ('I ',3X,'D(I,1)',3X,'D(I,2)',3X,'D(I,3)',3X,'D(I,4)',3X,
     1'D(I,5)'/)
      WRITE (11,245) C(1),C(2),C(3),C(4)
  245 FORMAT (12X,F7.5,3X,F7.5,3X,F7.5,3X,F7.5/)
      DO 255 I=15,N-1
      WRITE (11,260) I, D(I,1),D(I,2),D(I,3),D(I,4),D(I,5)
  255 CONTINUE
  260 FORMAT (I3,2X,F7.6,2X,F7.6,2X,F7.6,2X,F7.6,2X,F7.6)
      WRITE (11,261)
  261 FORMAT (/,'OUTPUT FOR MORBID4.FOR USING DATA FROM MORBID .DAT')
      WRITE (11,262)
  262 FORMAT (/,'I       Pi       Hi       AMUi'/)
      DO 265 I=15,N-1
      WRITE (11,270) I, P(I), H(I),AMU(I)
  265 CONTINUE
  270 FORMAT (I2,2X,F10.3,2X,F10.3,2X,F9.5)
      CALL DKS(C,DD,78,I,J)
      CALL MORBID(78,P,THE,TAU,ALPH,RECO,DD,HO,AMUO)
      WRITE (11,281)
  281 FORMAT (/,'INPUT DATA FOR AGE (I), CAUSE SPECIFIC DEATH RATE FOR
     1OTHERS (OTHERi)',
     1 /,'DEATH RATE FOR ALL CAUSES (ALPH i), AND RECOVERY RATE (RECi)',
     1 //,'  i       Pi     Other i    Alph i       RecO i'/)
      DO 283 I=15,N
      WRITE (11,284) I, P(I),THE(I),ALPH(I),RECO(I)
  283 CONTINUE
  284 FORMAT (I4,3X,F8.0,3X,F7.5,3X,F7.5,3X,F7.5)
      WRITE (11,285)
  285 FORMAT (/,'INPUT DATA FOR D(I,J) USING THE RELATIONSHIP THAT'/,
     1'                DD(I,J)=C(I)*DD(I+1,J-1)'/)
      WRITE (11,290)
  290 FORMAT ('D(I,1)',3X,'D(I,2)',3X,'D(I,3)',3X,'D(I,4)',3X,'D(I,5)',
     1/)
      WRITE (11,245) C(1),C(2),C(3),C(4)
      DO 300 I=15,N-1
      WRITE (11,310) DD(I,1),DD(I,2),DD(I,3),DD(I,4),DD(I,5)
```

```
  300 CONTINUE
  310 FORMAT (F7.6,2X,F7.6,2X,F7.6,2X,F7.6,2X,F7.6)
      WRITE (11,321)
  321 FORMAT (/,'OUTPUT FOR MORBID4.FOR USING DATA FROM MORBID .DAT')
      WRITE (11,322)
  322 FORMAT (/,'I        Pi        HOi       AMUOi',/)
      DO 365 I=15,N-1
      WRITE (11,370) I, P(I), HO(I),AMUO(I)
  365 CONTINUE
  370 FORMAT (I2,2X,F10.3,2X,F10.3,2X,F9.5)
      WRITE (40,372)
  372 FORMAT ('TRANSITION RATES FOR RUN 1',/,10X,
     1    '1-2       1-3       2-1       2-D       3-1       3-D')
      DO 375 I=15,N-1
      WRITE (40,380) I,AMU(I),AMUO(I),REC(I),D(I,1),RECO(I),DD(I,1)
  375 CONTINUE
  380 FORMAT (I2,2X,F9.5,2X,F9.5,2X,F7.5,2X,F7.5,2X,F7.5,2X,F7.5)
      END
C*************************************************************************
C                    SUBROUTINE (VALUES)
C*************************************************************************
      SUBROUTINE VALUES(XR,XRO,REC,RECO,QQ,XD,XDD,D,DD)
C THIS SUBPROGRAM GIVE THE ASSUMED RELATIONSHIPS BETWEEN CASE
FATALITY
C RATES, DEATH RATES AND RECOVERY RATES
      DIMENSION REC(80),D(80,5),DD(80,5),QQ(3,80),RECO(80)
      DO 70 I=15,78
      REC(I)=XR
      RECO(I)=XRO
      D(I,1)=XD*QQ(2,I)
      DD(I,1)=XDD*QQ(3,I)
   70 CONTINUE
      RETURN
      END
C*************************************************************************
C                    SUBROUTINE (DKS)
C*************************************************************************
      SUBROUTINE DKS(C,D,N,IJ)
C     SHORT PROGRAM TO CONSTRUCT D(I,J) VALUES
      DIMENSION C(4),D(80,5)
      N=78
      DO 450 J=1,4
      DO 450 I=15,N-1
      IF (I .EQ. (N-1)) D(N,J)=D(N,1)
      D(I,J+1)=C(J)*D(I+1,J)
  450 CONTINUE
      RETURN
      END
C*************************************************************************
C                    SUBROUTINE (MORBID)
C*************************************************************************
      SUBROUTINE MORBID(N,P,TAU,THE,ALPH,REC,D,H,AMU)
```

```
      DIMENSION P(80), H(80),REC(80),AMU(80),TAU(80),
     1 THE(80),BET(80,80), A(80,80),ALPH(80), U(80),
     1 F(80),G(80),D(80,5)
      N=78
      DO 550 I=15,N
      BET(I,1)=REC(I)+D(I,1)
  550 CONTINUE
      DO 560 I=15,N-1
      DO 560 J=2,5
      BET(I,J)=D(I,J)
  560 CONTINUE
C OBTAIN A VALUES
      DO 600 I=15,N-1
      A(I,1)= 1-BET(I,1)
      DO 600 J=2,5
      A(I,J)=A(I,J-1)*(1-BET(I,J))
  600 CONTINUE
C OBTAINING UNKNOWN VALUES FOR THE FIRST 2 AGE GROUPS
      H(15)=P(15)
      AM1= P(16)*TAU(16)
      AM2= P(15)*(D(15,1)-(A(15,1)*THE(16)))
      AMU(15)= AM1/AM2
      AH1=P(16)*(D(15,1)-(REC(15)*TAU(16))-(A(15,1)*ALPH(16)))
      AH2=(D(15,1)-(A(15,1)*THE(16)))
      H(16)=AH1/AH2
C WITHIN ONE LOOP CALCULATE Fi AND Gi'S THAT CAN BE USED IN
C CALCULATING Hi AND AMU i-1
      DO 630 I=3,N-14
      FT=0
      GT=0
      DO 620 J=2,I-1
      IF (J .GE. 6) GOTO 620
      FT=FT+(AMU(I-J+14)*H(I-J+14)*A(I-J+14,J))
      GT=GT+(AMU(I-J+14)*H(I-J+14)*A(I-J+14,J)*D(I-J+14,J))
  620  CONTINUE
      F(I+14)=FT
      G(I+14)=GT
      U(I+14)=G(I+14)-(F(I+14)*THE(I+14))
      H1=P(I+14)*(D(I-1+14,1)-(A(I-1+14,1)*ALPH(I+14))-
     1 (TAU(I+14)*REC(I-1+14)))
      H2=G(I+14)*(REC(I-1+14)-A(I-1+14,1))
      H3=F(I+14)*((THE(I+14)*REC(I-1+14))+D(I-1+14,1))
      H4=(D(I-1+14,1)-(A(I-1+14,1)*THE(I+14)))
      H(I+14)=(H1+H2-H3)/H4
      AMU1=(P(I+14)*TAU(I+14))-U(I+14)
      AMU2=H(I-1+14)*(D(I-1+14,1)-(A(I-1+14,1)*THE(I+14)))
      AMU(I-1+14)=AMU1/AMU2
  630 CONTINUE
      RETURN
      END
```

```
C PROGRAM NAME: BRASSCH.FOR
C THIS PROGRAM HOPEFULLY FITS BRASS MODEL
      DIMENSION GT(80),GTS(80),AGE(80),AQX1(80),AQX2(80),AQX3(80),
     1 QNX1(80),QNX2(80),QNX3(80)
      CHARACTER*6 CHAR1
      CHARACTER*8 CHAR2
      OPEN (UNIT=7,FILE= 'CHILEREF.DAT')
      OPEN (UNIT=8,FILE= 'CHILEDT4.DAT')
      OPEN (UNIT=9,FILE= 'CHILEDT4.OUT')
C READ AGE AND Nqx OF LIFE TABLE OF CHILE MALE 1964 AS STANDARD
C 3 CORRESPONDING STANDARDS ARE USED FOR ALL CAUSES, TB AND ALL OTHER
   65 DO 70 I=1,75
      READ (7, *) AGE(I), QNX1(I),QNX2(I),QNX3(I)
   70 CONTINUE
      IC=1
   75 READ (8,76) IY,CHAR1, CHAR2
   76 FORMAT (I4,A6,A8)
      DO 80 I=1,75
      READ (8, *) AGE(I), AQX1(I),AQX2(I),AQX3(I)
   80 CONTINUE
C AQX1,AQX2,AQX3 REFER RESPECTIVELY TO CHILE MALES FOR ALL CAUSES
C TB, ALL OTHERS. ONE YEAR'S DATA IS FOLLOWED BY ANOTHER, IN ORDER, THE
C YEARS ARE 1940, 1950,1959
C M IS THE INITIAL YEAR OF ANALYSIS
      M=20
      CALL BRASS(QNX1,GTS,75)
      CALL BRASS(AQX1,GT,75)
      CALL LEAST(GTS,GT,M,75,B1,A1)
      CALL BRASS(QNX2,GTS,75)
      CALL BRASS(AQX2,GT,75)
      CALL LEAST(GTS,GT,M,75,B2,A2)
      CALL BRASS(QNX3,GTS,75)
      CALL BRASS(AQX3,GT,75)
      CALL LEAST(GTS,GT,M,75,B3,A3)
      WRITE (9,100) IY, CHAR1,CHAR2,B1,A1
  100 FORMAT (/,'BRASS ESTIMATES FOR ',I4,2X,A6,1X,A8,' FOR ALL CAUSES',
     1//,10X,'BETA=',F7.4,/,10X,'ALPHA=',F7.4,/)
      WRITE (9,200) IY, CHAR1,CHAR2,B2,A2
  200 FORMAT (/,'BRASS ESTIMATES FOR ',I4,2X,A6,1X,A8,' FOR TB',
     1//,10X,'BETA=',F7.4,/,10X,'ALPHA=',F7.4,/)
      WRITE (9,300) IY, CHAR1,CHAR2,B3,A3
  300 FORMAT (/,'BRASS ESTIMATES FOR ',I4,2X,A6,1X,A8,' FOR ALL OTHERS',
     1//,10X,'BETA=',F7.4,/,10X,'ALPHA=',F7.4,/)
      IC=IC+1
      IF (IC .EQ. 7) GOTO 350
      GOTO 75
  350 END
C*********************************************************************
C     SUBROUTINE
C*********************************************************************
      SUBROUTINE BRASS(AQX,GT,N)
      DIMENSION GT(N),AQX(N)
```

```
      DO 50 I=15,N
      GT(I)=-.5*LOG(AQX(I)/(1.0-AQX(I)))
   50 CONTINUE
      RETURN
      END
C*********************************************************************
C     SUBROUTINE
C*********************************************************************
      SUBROUTINE LEAST(X,Y,M,N,B,A)
      DIMENSION X(N),Y(N)
      SX=0
      SXX=0
      DO 30 J=M,N
      SX=SX+X(J)
      SXX=SXX+(X(J)*X(J))
   30 CONTINUE
      SY=0
      SXY=0
      DO 40 J=M,N
      SY=SY+Y(J)
      SXY=SXY+(Y(J)*X(J))
   40 CONTINUE
      B=((N-M+1)*SXY-(SX*SY))/((N-M+1)*SXX-(SX*SX))
      A=(SY-(B*SX))/(N-M+1)
      RETURN
      END
C*********************************************************************
```

```
C PROGRAM NAME: SURVCH.FOR
      DIMENSION GTS(80),AGE(80),ALX1(80),ALX2(80),C(4),B1(4),
     1 B2(4),QXS1(80),QXS2(80),QXS3(80),AA(2),BB(2),ALXS2(80),ALXS3(80),
     1 AMX1(80),AMX2(80)
      OPEN (UNIT=3,FILE='ALBETCH.DAT')
      OPEN (UNIT=4,FILE='CHILEREF.DAT')
C ALBETCH.DAT ARE REGRESSION COEFFICIENTS- FIRST CONSTANT
C FOLLOWED BY COEFFICIENTS FOR BETA FOR ALL CAUSES THEN ALPHA
C FOR ALL CAUSES. SET 1 IS FOR BETA FOR TB, SET 2, ALPHA FOR TB
C SIMILARLY FOR OTHER CAUSES. CHILEREF.DAT CONTAINS THE STANDARDS.
C IN NQX AND SINGLE YEARS FROM AGE 5 FIRST FOR ALL CAUSES THEN FOR
C TB AND THEN FOR ALL OTHERS
      DO 20 I=1,4
      READ (3,*) C(I),B1(I),B2(I)
   20 CONTINUE
      DO 30 I=5,79
      READ (4,*) AGE(I),QXS1(I),QXS2(I),QXS3(I)
   30 CONTINUE
C I IN THIS CASE IS STANDING FOR THE AGE. THE FIRST
C VALUE IS ACTUALLY AGE 5.
      OPEN (UNIT=7,FILE='LOGITS3.OUT')
      OPEN (UNIT=8,FILE='SURVCH.OUT')
   35 READ (7,*) N,K,A,B
   36 BB(1)=C(1)+(B1(1)*B)+(B2(1)*A)
      AA(1)=C(2)+(B1(2)*B)+(B2(2)*A)
      BB(2)=C(3)+(B1(3)*B)+(B2(3)*A)
      AA(2)=C(4)+(B1(4)*B)+(B2(4)*A)
      WRITE (8,37) N,K
   37 FORMAT (/,'FOR AFRICAN COUNTRY #',I3,' GENDER = ',I2,/)
C BB IS FOR BETA, AA FOR ALPHA. 1 FOR TB AND 2 FOR OTHERS
C AL5 IS .99967 FOR TB, ASSUMING RADIX=0 AT AGE 1
      WRITE (8,38) AA(1),BB(1)
   38 FORMAT(' FOR TB, AA AND BB ARE  ', F7.4,1X,F7.4)
      WRITE (8,39) AA(2),BB(2)
   39 FORMAT(' FOR OTH, AA AND BB ARE  ', F7.4,1X,F7.4)
      CALL QXLX(QXS2,ALXS2,.99967,79)
      CALL BRASS(ALXS2,GTS,79)
      CALL SURV(AA,BB,GTS,79,1,ALX1)
      CALL LXMX(ALX1,AMX1,79)
C WRITE ANOTHER SUB TO CONVERT ALX TO MX (IA)
C AL5 IS .858528 FOR OTHERS, ASSUMING RADIX=0 AT AGE 1
      CALL QXLX(QXS3,ALXS3,.858528,79)
      CALL BRASS(ALXS3,GTS,79)
      CALL SURV(AA,BB,GTS,79,2,ALX2)
      CALL LXMX(ALX2,AMX2,79)
      WRITE (8,42)
   42 FORMAT (/,'AGE     Mx (TB)    Mx (ALL OTHERS)',/)
      DO 47 I=15,78
      WRITE (8,48) I, AMX1(I),AMX2(I)
   47 CONTINUE
   48 FORMAT(I3,1X,F9.7,2X,F9.7)
      IF ((N .EQ. 33) .AND. (K .EQ. 2)) GOTO 50
```

```
      GOTO 35
   50 END
C*********************************************************************
C     SUBROUTINE
C*********************************************************************
      SUBROUTINE QXLX(AQX,ALX,ALX5,N)
      DIMENSION ALX(80),AQX(80)
C THE QXS DATA START FROM AGE 5
      ALX(5)=ALX5
      DO 55 I=5,N-1
      ALX(I+1)=(1.0-AQX(I))*ALX(I)
   55 CONTINUE
      RETURN
      END
C*********************************************************************
C     SUBROUTINE
C*********************************************************************
      SUBROUTINE BRASS(ALX,GT,N)
      DIMENSION GT(80),ALX(80)
      DO 70 I=15,N
      GT(I)=.5*LOG((1.0-ALX(I))/ALX(I))
   70 CONTINUE
      RETURN
      END
C*********************************************************************
C     SUBROUTINE
C*********************************************************************
      SUBROUTINE SURV(AA,BB,GTS,N,NS,ALXN)
      DIMENSION GTT(80),GTS(80),ALXN(80),AA(2),BB(2)
      DO 80 I=15,N
      GTT(I)=AA(NS)+(BB(NS)*GTS(I))
      ALXN(I)=(1+(EXP(2*GTT(I))))**(-1)
   80 CONTINUE
      RETURN
      END
C*********************************************************************
C     SUBROUTINE
C*********************************************************************
      SUBROUTINE LXMX(ALX,AMX,N)
      DIMENSION ALX(80),AMX(80)
      DO 100 I=15,N-1
      AQX=(ALX(I)-ALX(I+1))/ALX(I)
      AMX(I)=AQX/(1-(.5*AQX))
  100 CONTINUE
      RETURN
      END
C*********************************************************************
```

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