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Mortality, Morbidity and Population Health Dynamics

A Thesis submitted in partial fulfilment
of the Degree of Doctor of Philosophy
at the University of Waikato

M. Jit Cheung

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The
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ABSTRACT

This thesis is concerned with the dynamics of the health of populations with low levels of mortality, using the non-Maori population in New Zealand as a case study. The primary objective is to study patterns and trends in mortality and morbidity, their interactions, and their implications for the future.

The key questions addressed in this thesis are central to the future evolution of population health. Given the significant declines in mortality, especially those at older ages that have occurred over the last two decades, will mortality declines at advanced ages continue much longer? Or will mortality become increasingly compressed into a narrow range of ages at death? Moreover, what are the implications for morbidity trends and any indications at present of future trends in population health? Will increased survivorship be accompanied by long periods in ill-health, or will morbidity move in tandem with mortality, or will durations surviving but in ill-health actually decrease?

The thesis first establishes a theoretical platform by reviewing and synthesising transition theories and empirical observations into a general framework to guide the understanding of population health dynamics. It also forges a theoretical linkage between variations in the individual senescence process, which underpin the validity of demographic models of mortality and morbidity, and their population level manifestations. These theoretical extensions provide the context for an evaluation of contemporary theories on mortality trends and changes in morbidity in relation to mortality.

In addition to using long term historical data spanning over a century and complemented by a population-based true cohort approach, this thesis also makes methodological advancements by adapting and developing a tool, known as health expectancy, for the analysis of population health. In particular, a new measure termed "hospitalisation expectancy" is developed. It integrates data on hospital utilisation and mortality to yield a summary measure of population health.

The empirical study documents the shift in the forces of mortality of non-Maori, from younger to older ages, and from communicable causes of death to chronic degenerative diseases. Results of the mortality analysis by cohorts show that while there may be some indications of a future trend towards mortality compression, further increases in life expectancy at birth can be realistically expected. Empirical evidence of compression of morbidity is found in the trends of hospitalisation and the patterns of severe forms of disability. The causes of morbidity are becoming more diversified, dominated by non-lethal conditions; whereas the causes of mortality are becoming increasingly compressed into a few chronic conditions, namely cancers and cardiovascular diseases. It is also established in this thesis that the older population represents a heterogeneous group in terms of levels of susceptibility to ill-health. An empirical method for identifying heterogeneity is developed to show non-Maori inter-cohort differences in this property.

Later in the thesis, a cohort based mortality projection of non-Maori at adult ages is undertaken. Various scenarios of morbidity responses to the projected mortality changes, incorporating the leading theories on future morbidity changes, are then modelled. To conclude the thesis, the implications for policy of further mortality declines at older age among an increasingly heterogeneous population are discussed.

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When opening up a thesis, the first section I am always eager to read is the acknowledgements, and it is just as well that the section is strategically placed at the very beginning of the thesis. In writing their acknowledgements, authors have already gone through arguably the worst part of an enduring, and often pain-staking, labour-intensive process. A sense of satisfaction and relief radiates from their writings, and I can share in their ecstasy of this most rewarding moment.

Writing this thesis was a monumental task which, to many of my friends and family, seemed a life-long obsession. It was made particularly difficult since for most of the period I was in full time employment. I would not wish any reasonable human being to subject oneself to the same process. And yet the joy in seeing the final product is most up-lifting, which I would equate to giving birth to a newborn; a sensation which at least half of the population, myself included, could never claim apperception.

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Professor Brown's unflagging persuasion to complete the thesis proved critical at the end. His distinctive American flare on the basketball court also provided me with refreshing moments. I was also fortunate to be able to work with Dr Martin Tobais, Senior Adviser at the Ministry of Health, with whom I had held many valuable discussions on developing the methodological aspect of the thesis.

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PART ONE:

INTRODUCTION

CHAPTER ONE
INTRODUCTION - THE HEALTH OF POPULATIONS

Demography concerns itself with the dynamics of population behaviour. A fundamental determinant of which is mortality. This factor has major impacts on demographic growth, and population sizes and structures (Myers, 1989; Omran, 1971; Pool, 1994). The last century and a half provides the most dramatic example in our demographic history of the impacts of mortality change. Life expectancy at birth in most developed countries has doubled from 40 years to around 80 years (Coale, 1996; Olshansky, 1990). The remarkable declines in mortality, especially those in the last half century, had created a "new demography" of longer lives (Vaupel, 1998), and an accompanying rise in the size and the changing health status of the older population.

The present study is structured principally around the theme of mortality, its long-term past trends and future developments. At the same time there is recognition that trends in population health are a part of wider demographic transitions. As a secondary theme, it analyses the impact of changes in morbidity, which, in relation to mortality changes, are becoming increasingly critical in determining the health status of populations. The overall substantive aim of the present study is to develop an insight into the past, the current and the future evolutionary pathway of population health for non-Maori New Zealanders, those persons predominantly of European origin, constituting about 85 percent of the total population. But New Zealand provides a case-study which has general application across other populations and/or countries.

The theoretical component of the study gives full attention to and integrates the analysis of mortality and morbidity. Because of limitations in the available data, however, the empirical analysis of morbidity historically over the longer-term is limited compared to that of mortality. For this reason, and because this is the first detailed study of the mortality of the elderly in relation to morbidity for New Zealand, this thesis gives more attention to theoretical and methodological questions than might otherwise be the case.

The key questions which this thesis seeks to address are central to the future evolution of population health. Given persistent declines in mortality, especially those at older adult ages that have occurred over the last two decades, will mortality declines at advanced

ages continue much longer; or will they come to an end at some point so that mortality would become increasingly concentrated into a narrow range of ages at death? This sets the theme for the central theoretical concerns of this thesis which investigates whether or not this concentration, termed "compression", occurs, and then seeks to analyse its causes and consequences.

Following these mortality changes at older ages projected for the immediate future, what are the implications for morbidity trends and are there indications at present which may indicate future trends in population health? These questions are highly topical for the health systems in New Zealand and other more developed countries, in which around three-quarters of all deaths are currently occurring at ages 65 and above, and where causes of death are concentrating around a limited range of chronic degenerative diseases. Of particular importance for New Zealand analysts is the fact that population level morbidity information are sparse and that which are available do not permit high quality trend analyses[1]. Unlike many of its developed counterparts such as Australia, France, Canada and the United States, New Zealand has no repeated population level health surveys which might yield comparable information at two or more points in time. Trends in morbidity are often inferred from mortality and/or morbidity studies done overseas.

This thesis takes as its starting point the idea of a heavily concentrated mortality pattern at older ages conceptualised by the notion of the "compression of mortality" by Fries (1980), whose work has been the inspiration for many studies including the present one. Rather than inherently wanting to prove or to disprove theoretical conjectures raised by Fries, the present study uses the hypothesis to conduct investigations on issues affecting population health.

The postulate of "mortality compression" has sparked intense debate because of its extensive influence on views regarding future trends and their impacts on the health status of the population as a whole. It also carries profound policy implications, not only relevant to the size of the future older population, but because the validity this theory

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[1] With the exception of public hospital admission and discharge data, which have many inherent problems for time series analysis (see Chapters Seven and Nine), few population level morbidity data exist elsewhere in New Zealand.

would help to shape population level policies regarding activities and interventions at these ages.

Implicit in this is the recognition that compression, or any of its alternatives, merely the population level average of countless variations in individual experiences of health, disability and death. The notion of compression does imply a dampening of these variations, yet some understanding of differentials within population is essential even in a macro-level analysis. The idea of epidemiologic polarisation (Frenk et al, 1989; see Chapter Two) recognises this. In the present study the notion of population, especially cohort, heterogeneity is introduced and explored both theoretically and empirically as an underpinning sub-theme.

The issue of possible mortality compression was first analysed for New Zealand by Pool (1994). By using a crude percentile analysis of cross-sectional data he found empirical support for mortality compression in its simplest form: a narrowing range for age at death. More recent research carried out in other developed countries generally supports Pool's argument (Nusselder and Mackenbach, 1996; Paccaud et al, 1998; see also Rothenberg, 1991). The present study extends from Pool's earlier work, examining possible mortality compression of non-Maori in New Zealand using both cross-sectional and cohort data. Evidence for this phenomenon in the accompanying morbidity patterns are also investigated. At the end of this analysis the results are synthesised for cohort level determinants of changes in future population health dynamics.

Finally this thesis adopts another strategy explored briefly by Pool (1994). This thesis is built around a long-term analysis of both cross-sectional and synthetic, plus true cohort rates, life tables and health expectancy models (and a variant developed specially for this thesis). As will be raised in Chapter Three, such a longer-term perspective is essential if fuller understanding of the health of ageing population is to be better understood.

This thesis therefore has the following listed set of substantive methodological and theoretically objectives.

- At a theoretical level, to add to a growing knowledge-base on population health, particularly at older ages, by
  - identifying the role of population health trends as a constituent of the wider demographic transition, and more narrowly seeking the links between shifts in patterns of longevity and age-structural transition at older ages;
  - distinguishing, and thus drawing linkages between individual senescence processes, individual variations of these, and their relationships with population health and heterogeneity; and
  - integrating and synthesising different theories on (i) future changes in mortality, and (ii) on morbidity in relation to mortality, so as to develop a theoretical framework for the analysis of mortality, morbidity and population health dynamics.
- At a methodological level, to develop a framework for the analysis of mortality, morbidity and population health dynamics, and to apply this to a case-study, the non-Maori population in New Zealand, by
  - deriving a cohort based approach to the analysis of population health, complementing the more conventional cross-sectionally based synthetic studies;
  - analysing long-term trends in non-Maori mortality and morbidity to set the context for substantive issues;
  - advancing population health measures which integrate patterns of mortality and morbidity, and have direct practical applications in the New Zealand context;
  - testing empirically the occurrence, or not, of compression of mortality, and where possible, morbidity, to provide a baseline for projecting future mortality and morbidity changes; and
  - investigating, for older non-Maori cohorts, the impacts of their accumulated exposure to the risks of mortality at younger ages on their mortality experience at older ages.
- At a policy level, to identify the policy implications of the present study, by
  - providing a policy context in formulating responses to major changes in population health in the short-term future;
  - underlining potential sources of concern and thus policy focus in population health; and
  - highlighting the needs for developing a better understanding of issues on population health, and informing future policy directions as well as research agenda.

The rest of this chapter sets out in greater detail the themes to be addressed by this thesis. The present chapter analyses the conceptual and methodological issues involved in measuring population health. It then defines the specific aspects of emerging issues in population health which are to be investigated in this thesis. Later in the chapter, an overview of the structure of the thesis is given.

1.1 Reasons for Measuring Population Health

While there is a high level of academic and theoretical interest in the analysis of population health patterns and trends, their implications for future health policy planning are even more far-reaching. The continuous evolution of population health, particularly over the last few decades, has placed a heavy strain on health and the wider social policies. Population health status is becoming an increasingly major concern to policy planners, both at local and national levels. This trend is evident in many developed countries, as is demonstrated by the growing momentum behind the push for health system restructuring in New Zealand, in the United Kingdom, in the United States and elsewhere. The most prominent features of changes in population health status are the continuing improvements in survivorship through younger ages and today into the advanced ages, and an accompanying longer-term numerical ageing of the population (see below). The question of future population health trends, and the subsequent costs of looking after the older population, will be a challenge to health administrators for the years to come.

Policy is written for population groups, not individuals, and therefore measures of health status at a population level are inevitably required to support this exercise. The need for population based health analysis is also self-evident in an era of growing discussion of the curative versus preventative and caring capacities of the medical system, the possible emergence of negative impacts of medical advances and social expectations on population health as reflected by an upsurgence in morbidity rates, and rapid increases in health spending as a percentage of government's annual budget across developed counties. In the face of intense competition for resources, the growing investments in the health care delivery system, either by government or the private sector, calls for more effective use of personnel, equipment and institutions. Devising a means to measure the

health status of populations whom the system seeks to service thus becomes imperative. These policy implications have elevated this area of research from a somewhat academic research pursuit to mainstream health policy research.

Simply stated, the primary purpose of measuring population health is to keep a "score" on the present state of population health and progress being made toward its betterment. Scores need to be kept in a valid and reliable manner so that longitudinal and cross-sectional comparisons are possible. In today's context, the objective of population health status measures are to specify the dynamics between the contemporary pattern of mainly chronic morbidity and mortality. From a policy point of view, three central underlying purposes can be identified:

1. *Resource Allocation*: concerned with channelling limited resources into different areas in the most efficient manner possible, a function ideally carried out in the early stage of health planning, though this is not always the case in practice. An adequate resources allocation process involves a number of sub-steps.
   - Global evaluation of the patterns and trends of population health status (the primary objective of this thesis).
   - Appraisal of capacity to gain and resources needed to achieve these gains.
   - The setting of priorities for the wider health sector and allocate resources.
2. *Programme Prioritisation*: where two or more proposed programmes compete for the same type of resources, competing programmes can be compared so that the most desirable combination can be identified.
3. *Impact Evaluation*: designing measures of outcomes — of the efficiency and the effectiveness of the health programme(s) or the system at large — as a part of the monitoring process.

This list coincides with different stages of health administration. Resources allocation takes place at the first stage, primarily responsible for identifying areas of concern. Once the tasks are defined, means to tackle them can be investigated at the second stage of programme prioritisation. Mainly quantitative, but sometimes qualitative, evaluation is part of the ongoing process in health planning. The steps in this hierarchy are closely linked with one another. Siegmann categorised measurement processes in the first two

groups as drawing on disciplinary research indicators, designed to summarise population health status in general; whereas the last group comprises policy research indicators, designed to identify the effect on population health status of particular programmes (cited in Mooney and Rives, 1978). Distinctions such as this, especially the labels used by Siegmann, may be accentuated at the time they are proposed, but are less accurate in the current context when research and policy are becoming increasingly integrated.

1.2 Issues in Measuring Population Health

1.2.1 Conceptualising Health

Many attempts to quantify the dynamics of population health have stumbled at the first major hurdle: the defining and measuring of what is essentially an abstract notion, *health*. Measuring health of either an individual or a community, or a population as a whole is fraught with many problems, because health and its counterpart, ill-health, are notoriously ambiguous concepts.

A simple, unified, conceptually rigorous definition of population health is lacking, but obstacles against achieving this goal are obvious (Alter and Riley, 1989; Bunker et al, 1996; Chen, 1976; Lerner, 1973; Mooney and Rives, 1978; Saracci, 1997). An inherent problem is the multi-dimensionalilty of its central concept, health, which can be extended to include a number of dimensions, and can be interpreted from "clinical, demographic, social, biological, genetic, psychological, or humanistic perspectives" (Fries, 1989:16). Most of the definitions of health proposed over the years do not lend themselves to operational applications. The most notable example is that used by the World Health Organisation (WHO).

> *"Health is a state of complete physical, mental, and social well-being, and not merely the absence of disease or infirmity."*
>
> (WHO, 1992)

Inscribed in the constitution of the World Health Organisation in the aftermath of the Second World War, this definition has the advantage of stressing the inadequacy of delineating health in terms of disease and infirmity alone. While comprehensive and covering all three dimensions of health, physical, mental and social, this now classic definition of health has, however, been widely criticised for its ambiguity and simplicity (Colvez and Blanchet, 1981; Doll, 1992; Goldsmith, 1972; Saracci, 1997; Twaddle, 1974). It offers little substantial basis for measurement since it is difficult to see how "complete physical, mental, and social well-being" can be translated into operational terms. The notion of "perfect health" is more utopian than real. Even the precise meaning of "absence of diseases and infirmity" can be ambiguous. These criticisms highlight the fact that, though the desirability of adopting a positive definition of health is widely advocated, the lack of axes on which to base classification hinders its effective operation.

The notion of positive health is a major component of many endeavours, but one universal feature shared by all is an absence of data on positive health, and thus a reliance on measures of ill-health. This is an age old paradox because, historically, measurements of health status have been conceptually framed in terms of disease, disability and death. Terms such as "disease", "disability", "impairments" have been used interchangeably to imply the manifest inverse stages to good health. Furthermore, the presence of positive health, though favoured ideologically, can hardly be proved with any rigorous evidence (Patrick and Bergner, 1990). Maybe everyone suffers some minor episodes of ill-health; it is the degree to which such sub-clinical events interrupt their normal life, or impairs or disables them which is difficult if not impossible to define and measure with fine degrees of calibration. Confounding this are individual and even cultural variations in the way these episodes are identified as interrupting normal daily life (see below).

Definitions of the physical and mental health of an individual have been heavily influenced by clinical diagnostic classifications. Not surprisingly, medical data principally on physical conditions have long served also as the bases of population health analysis. The high profile for chronic diseases that has emerged in recent decades has, however, rendered clinical conceptualisation and measurement of health problematic because of the occurrence of multiple states of clinical manifestation, variations in disease progression and lengthy pre-symptomatic stages often associated with these diseases. Definitions of

mental and social health are even more elusive (Saracci, 1997). The terms mental health/illness have traditionally embarrassed psychologists for their inability to conceptualise the concept satisfactorily (Moriyama, 1968).

The health of an individual can also be conceptualised from a physiological or functional perspective, but essentially this is an extension of the clinical tradition. Two major components of health are identified: (i) an individual's level of functioning at one point in time, and (ii) the individual's expected transition probability from the present functional level to some other functional levels. From this perspective health at a given time can be defined as a composite of a range of functional statuses and prognoses. This definition is underpined by the notion of the role performance of an individual in a given socio-biological context, for the health of an individual is closely associated with the individual's ability to fulfil his or her social and biological role.

Personal ability to perform important life functions, however, is influenced by factors falling both inside and outside the parameters of health. Cultural and economic factors, to name two, exert substantial influences on health thus defined (Riley, 1992). Differences in levels of motivation and in the effort required also alter an individual's performance. The abstract notion of health is thus difficult to be measured objectively through performance based health assessments (Hoeymans et al, 1997). Conceptually, it should be stressed that disability is not the same as dependency; persons with certain functional disabilities may still be able to perform a large proportion of remaining functions.

A range of alternative definitions of health and associated problems have been investigated by many researchers over the years (Bunker et al, 1996; Chiang and Cohen, 1973; Chen and Bryant, 1975; Doll, 1992; Goldsmith, 1972; Moriyama, 1968; Patrick and Bergner, 1990; Saracci, 1997; Twaddle, 1974). The diversity of opinion on these conceptual issues is manifested at the methodological level. An operational definition of "health" is often tailor-made for a specific task in consonance with the needs of its author, and ways of measuring it are then devised. The possibility of even finding a single concept of health which may eventually be encapsulated by an operational definition has long been dismissed by some (see Lawton et al, 1967).

These issues become both easier and more difficult to resolve at a population level. Easier because variations between individuals are dampened down in the process of computing measures of central tendency and other demographic rates. More difficult, because the health of a population is a far more abstract concept than the health of an individual, because group norms, values and perceptions of what constitutes health may confound inter-group comparisons, and because of classical issues relating to macro-micro linkages (Lipton, 1998:28), above all the risk of "ecological fallacy".

1.2.2 Methodological Issues

A direct consequence of the lack of unifying theory of health at the conceptual phase is the absence of a universal unit of measurement. The concept of a single unit of health does not exist and is unlikely to be developed in the near future. To the extent that what constitutes good health cannot be agreed upon, the notion of health remains abstract and cannot be objectively measured. In response to these inherent problems, techniques used in studies of population health vary considerably, ranging from purely empirical mortality and morbidity data analysis to sophisticated theoretical framework of population heterogeneity.

The design of health status measures has a long history dating back to the nineteenth century. Mortality statistics have long been the traditional tool of demographers, a tendency reinforced by the availability of long and generally comparable series of death statistics. The importance of mortality-based measures was heightened in an era when mortality from infectious disease was the prominent health problem as the population was confronted by high incidence and case-fatality rates of these diseases. Arguments in favour of morbidity measures by which to analyse disease incidence and prevalence, the central components of classic epidemiology, came to prominence only after the Second World War. As chronic diseases increased in relative frequency among causes of death, problems arose over the classification and interpretation of cause of death statistics. Growing dissatisfaction with the power of mortality statistics in health analysis set the stage for new developments in morbidity indicators. Morbidity data were, however, scarcely ever collected in a systematic way.

The increase, beginning in the mid 1940s, in the relative importance of chronic degenerative diseases also changed general perceptions of population health. Reflecting growing appreciation of the multi-dimensionality of the concept of health, a new stage of development in health measures was marked by increasing controversy over the conceptualisation of the phenomenon being measured. Traditional clinical definitions of health had expanded to embrace psychological, personal and social dimensions. The arsenal of health status measures greatly diversified though there was still no consensus over which parameters should be included in the definition of health. These methodological developments unfolded in many different directions. For example, the concept of utility was introduced for measuring and aggregating population preferences for alternative health outcomes; formal probabilistic models of disease prognoses were developed; cost-effectiveness models were used for programme evaluation and for prospective resource allocation. However, an essential feature of this early stage of unsystematic research was that health status indices, when applied, nearly always were restricted to a few selected disease programmes aimed at particular population subgroups.

The current stage of development of research in this area is marked by a growing interest in the search for some form of more global or integrative population health status index, to replace sets of specific indices. Increasingly attention is also being given to morbidity and how to integrate some measure of it into the traditional mortality-based indices. A major obstacle to achieving this at a population health level, as against studies in epidemiology or case-control analyses, has been the absence of morbidity data, particularly historical time series.

This thesis adopts an international emerging methodology, termed "health expectancy", which, however, depends on the availability of data on disability. To attempt to overcome this constraint, it then constructs a new measure designed specially for this thesis and called "hospitalisation expectancy". The advantage of this strategy is that hospital data are more widely available for longer time periods (and at a sub-national level) than any other data on morbidity. They have large numbers at the population-level,

and cover clinical causes. Against this, they do have drawbacks which will be analysed further and strategies developed to minimise these effects in Chapter Seven.

1.2.3 Measuring Ill-health to Study Population Health

On a population level, mortality and morbidity rates must be used as indices of population health because, paradoxically, they are the most direct measures of "ill-health" in the population, through its attempt to assess "health". The occurrence of "perfect health" is essentially not measurable, but its absence is more readily defined and measured by evidence on poor health. Measuring patterns of ill-health in the population is therefore the most pragmatic, albeit counter-intuitive, approach to population health analyses. For the purpose of this thesis, the basic parameters of population health are said to be defined by both mortality and morbidity patterns and trends.

It should also be noted that mortality and morbidity statistics alone do not adequately describe all aspects of population health. For example, social well-being, an integral component of the World Health Organisation's definition of health, cannot be adequately defined or measured by these statistics. The analysis of social and environmental aspects of population health thus require a much wider perspective than the one adopted in this thesis. Studies of these wider aspects of population health constitute almost a separate discipline in their own right.

Later in Part Two of this thesis, the issue around measuring ill-health to study population health will be more extensively analysed through discussions on the evolution of population health. These will highlight the multi-faceted characteristics of population health, and will provide further justifications for identifying mortality and morbidity as the principal attributes which underline its evolutionary processes. Investigations into the leading contemporary theories on mortality and morbidity trends in Chapters Four and Five will further emphasise the properties of these two foremost dynamic attributes of population health.

1.3 Background to the Present Study

Underlying the century long secular decline in mortality is a shift in the causes of death, from infectious diseases to chronic degenerative diseases, and the subsequent redistribution of deaths from the young to the old. The earlier stages of these transitions, which occurred in the nineteenth century and the first decade of the twentieth century mostly in more developed countries, have been encapsulated in Omran's epidemiologic transition theory (Omran, 1971; 1982, see Chapter Two of this thesis for a more thorough discussion). The more recent trends in these and other countries have aroused intense international debate (see Chapters Four and Five). Nevertheless, and despite their different outlooks on the future, health researchers around the world are generally unified in the view that the persistent declines in mortality and the seeming rise in the prevalence of selected morbidity conditions have massively transformed the landscape of population health in developed countries, and that the same process is working at a fast pace in developing countries. These transformations form the backbone for contemporary health transition theories. Also embedded in these theories are explanations based around changes in public health care delivery and personal health-related behaviours. The combination of these factors have reshaped the population's perception of what constitutes "health". Consequently, the direction of study of population health has been adjusted constantly in the light of these theories as well as by other more empirical considerations.

As a more immediate issue, recent changes in patterns of mortality at older ages, particularly among low mortality populations in developed countries, are taking on an heightened international significance. Competing theories on, and estimates of, the limits to life expectancy have proliferated over the decades. The IUSSP (International Union of Scientific Studies of Population) has set up a longevity committee to co-ordinate efforts in this area of research (I. Pool, personal communication); a plenary session of its last general conference in Beijing (1997) was devoted to this topic; and emerging technical issues are now the concern of an international "Network on Health Expectancies" (Réseau Espérance de Vie en Santé, REVES).

Coinciding with the demographic phenomenon of increasing concentration of risk of mortality and morbidity at older ages is the emergence of demographic forces which are distorting

population age structures. Ageing of the population is a real demographic issue with profound social policy implications. "Numerical" ageing of the population (Pool, 1994), that is, the rapid increases in the absolute numbers at older ages, stems from variations in the sizes of birth cohorts, as these are modified by migration and, of major importance in the context of this thesis, by improved survival at younger ages. "Structural" ageing of the population, or the increases in the proportions at older ages, is a function of very different forces, mainly decreases in fertility (see Chapter Two). In most developed English-speaking countries, including New Zealand, numerical ageing of the population is further reinforced by the demographic waves created by the post war baby-boomer cohorts, which is itself a key contributor of "structural" ageing.

1.4 Analytical Strategy

This thesis first establishes a theoretical platform by reviewing contemporary theories on mortality trends and changes in morbidity in relation to mortality. From this, a framework for the analysis of population health patterns and trends of the non-Maori population in New Zealand is developed for the purpose of this thesis.

In addition to understanding trends in cross-sectional data, a considerable part of the present study is invested in the analysis of cohort patterns and trends. A cohort approach is an integral component of the present study and sets it apart from conventional time series analyses mainly of synthetic patterns. A synthetic analysis is confounded by the range of experiences of the various cohorts involved, who, earlier in their lives, are likely to have had different patterns of exposure to the risk of mortality and morbidity. A cohort based approach is therefore theoretically superior to a synthetic one. Issues relating to cohort mortality and morbidity experience are explored throughout this thesis.

In the empirical component of the study, trends in non-Maori mortality and morbidity are investigated wherever data are available. The test for mortality compression is extended from investigating trends in synthetic life tables to examining trends in cohort life tables constructed by the present author. Results of the cohort analysis are synthesised through a retrospective analysis at a cohort level of the antecedents of recent population health

dynamics. Particular attention is given to the synthesis of relationships between patterns of earlier cohort exposure to mortality and morbidity risks, and the mortality experience of each cohort later in life. In the later part of this thesis, a cohort based mortality projection of non-Maori at adult ages is undertaken. Various scenarios of morbidity responses to the projected mortality changes, incorporating the leading theories on future morbidity changes, are then modelled.

Detailed reasons for choosing non-Maori (the overwhelming majority of New Zealanders, 85 percent) as the focus of this study are given in full in Chapter Seven. But in sum, the non-Maori mortality transition is at a more advanced stage compared to that for the other principal New Zealand population, the minority Maori (about 15 percent of the total). Other smaller minority populations, notably Pacific Islanders and Asians, are included in the non-Maori figures.

An appealing aspect of this focus on non-Maori is that their mortality trends present many strong parallels with changes in other developed countries. A major topic in the present study concerns the implications of the more recent significant mortality reductions at older ages, a phenomenon in New Zealand mainly affecting non-Maori, but shared by many other European-origin populations. The non-Maori population presents therefore a good case-study for understanding the future evolution of mortality in developed countries. Many of the key findings from this study can, however, also be inferred to the Maori population and to other delayed transitional societies, to use Omran's term (1982; see also Pool, 1994).

Focusing on the non-Maori population also has methodological advantages. The overwhelming majority of deaths now occurring at the older ages in New Zealand (95 percent in 1996) are non-Maori. More importantly, the collection of non-Maori demographic data in New Zealand dates back to the middle of the nineteenth century. These data are sufficiently adequate and robust to support good time series analysis. Furthermore, detailed mortality analysis by birth cohorts is made possible through the availability of these long series demographic data. Finally, hospital admission and discharge data, generally of reasonable quality, are available for most years since 1951.

1.5 Organisation of the Thesis

This thesis is organised into six parts. Part One, the introduction, consists of the present chapter which sets out the theme and the substantive issues to be addressed by this thesis. Conceptual and methodological issues in measuring population health are also dealt with in the chapter.

Part Two covers contemporary theoretical issues emerged in the field of population health. Chapter Two provides a macro-level review of population transition theories across a wide range of disciplines relating to the evolution of population health. Included in these discussions are the demographic transition, the fertility transition, the epidemiologic transition, the mortality and the morbidity transitions, and the more recently proposed health transition. A general transition framework linking the various transition models by their major constituting elements is developed later in Chapter Two, which provides the essential background for developing a theoretical framework for the present study. Chapter Three performs a major theoretical role by forging micro-macro linkages between individual and population health. It first details theoretical explanations of health status changes at the micro-level. For this, differences in perspectives on senescence, which govern the biological ageing process of individuals, are summarised from a range of disciplinary stand points. The focus of Chapter Three then turns to inter-linking the macro-level views of population health as presented in Chapter Two and micro-level explanations put forward in Chapter Three thus far. The concept of population heterogeneity is introduced in the latter part of Chapter Three, a perspective which translates up to the population level the effects of variations in the senescence process of individuals.

The presentation of these issues as viewed from the micro and macro-levels, leads into Chapters Four and Five which theoretically forge links between patterns of mortality and morbidity. Chapter Four sets out in detail the central question of this thesis concerning mortality changes at older ages, taking as a lead-in the hypothesis of compression of mortality, plus its corollaries and alternative hypotheses. Arguments both in favour of and opposed to this hypothesis are systematically laid out for subsequent testing in the body of the thesis. Chapter Five takes this further by integrating morbidity and mortality

trends. It investigates leading theories on recent morbidity trends in relation to mortality changes.

Part Three outlines a framework developed for this thesis for the empirical analysis of non-Maori health patterns and trends. Chapter Six summarises the emerging theoretical issues in mortality and morbidity discussed in Part Two, and integrates elements of the various theories into a broader framework. Chapter Seven translates the framework developed in Chapter Six into an operational model. Methodologies for the analysis of non-Maori mortality and morbidity as applied in the subsequent chapters are explained. Issues around data availability and quality are also discussed in Chapter Seven.

Parts Four, Five and Six present the results of empirical study of non-Maori mortality and morbidity patterns and trends. These three parts adopt a progressive sequence, from descriptive analyses in Part Four to explanatory analyses in Part Five to synthesis in Part Six.

Part Four starts the empirical sequence with detailed descriptive analyses of non-Maori mortality and morbidity patterns and trends. Chapter Eight provides a review of non-Maori mortality trends over the last century. Issues emerging as a result of this review are also discussed. Chapter Nine proceeds with a review of non-Maori morbidity patterns and trends in relation to mortality over the more recent period for which data are available. Chapter Ten continues the descriptive analysis with a review of trends in non-Maori cohort mortality experiences.

Part Five moves forward with explanatory analyses of non-Maori mortality transition, using Fries' compression of the mortality hypothesis as its investigative framework. Chapter Eleven presents results of an in-depth analysis of the shift in non-Maori age at death. The trends in the recent series of synthetic life tables are tested for mortality compression. Chapter Twelve repeats the same exercise, but from a cohort perspective, testing for mortality compression among non-Maori cohorts. The results are compared and contrasted with those from Chapter Eleven, and the discussion lays the foundation for projecting future cohort mortality trends later in this thesis.

Part Six synthesises the results of the empirical study and discusses their policy implications. Chapter Thirteen summarises the key findings from the chapters in Parts Four and Five, and then synthesises these descriptions and explanations. It does this by exploiting the long-term cohort analyses presented earlier, showing how the mortality and morbidity experiences of cohorts at younger ages combined to determine intra- and inter-cohort changes in mortality and morbidity at older ages. Chapter Fourteen presents cohort-based non-Maori mortality projections for adult age groups over the next 30 years from 1996 to 2026. The results are presented by both cohort and synthetically for census years. Later in Chapter Fourteen future morbidity trends are investigated by modelling the various scenarios postulated in the leading theories in Chapter Five. Chapter Fifteen brings the focus back to policy and the health system, and concludes the thesis with a discussion on the policy implications of the present study.

PART TWO:

EMERGING THEORETICAL ISSUES IN POPULATION HEALTH

CHAPTER TWO
THE EVOLUTION OF POPULATION HEALTH

Health and disease patterns in a society evolve in a complicated manner, responding to a wide array of demographic, socio-economic, biological, medical, technological, political, and cultural factors. These factors are identified in what are termed transition models which attempt to outline and explain these changes.

This chapter thus presents a detailed review of the evolution of population health over the last century and beyond, mainly for more developed countries. The approach followed is to review major population transition theories, describing key elements of demographic changes which played a part in, and provided a context for, the evolution of population health. The purpose is to provide a macro-level overview of issues emerging in the contemporary study of population health.

The present chapter serves a number of objectives. By providing an historical perspective to the changes in population health under investigation, the significance of these changes can be more adequately understood in terms of historical trends, of their major determinants, of their pace of change and of their future implications. Furthermore, discussions on the theoretical context of population health issues help to formulate a general framework for later analyses. Population transition theories have long dominated demographers' thinking of both longitudinal and cross-sectional variations in the population behaviour because of the valuable broad framework these models provide.

The evolution of population health in each country is unique to its prevailing social and epidemiological environments, whereas population transition theories are constructed around commonalities drawn across populations in their behaviour patterns. As will be implied in this chapter, the unfolding of new developments in population health inevitably challenges aspects of the transitions theories established over the years. This thesis is, however, less concerned with these debates than with the fact that transition theories provide a convenient, even if somewhat imperfect, means of organising and synthesising important questions relating to aspects of population health.

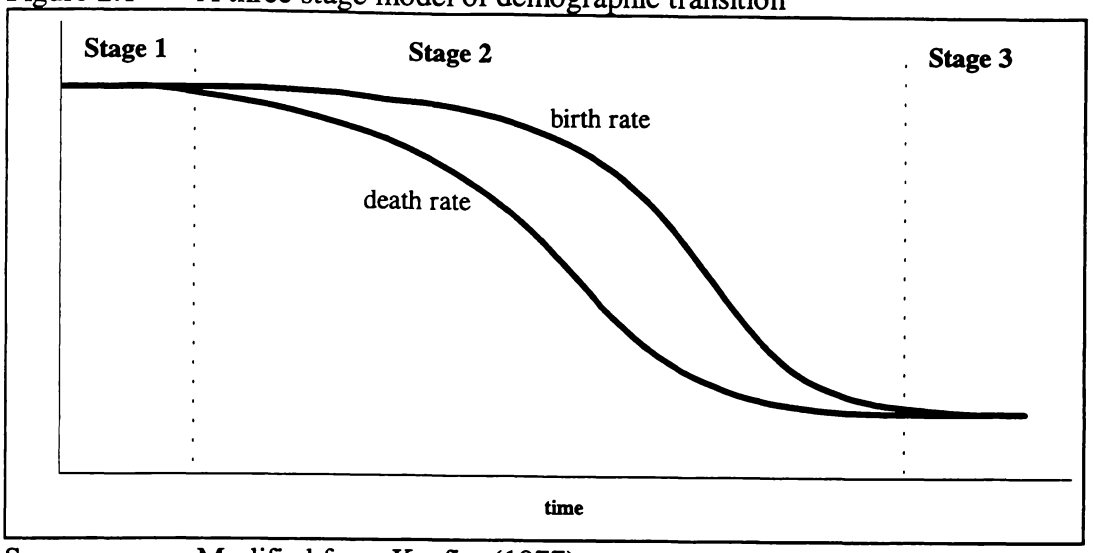
The demographic transition, which describes a transformation from high mortality and high fertility to low mortality and low fertility, presents the broadest conventional framework. It identifies and sets out the relationships between the various constituents that are each the focus of other related transition models. This chapter starts with a brief review of the demographic transition. It is then followed by an analysis of the epidemiologic transition model, which describes major changes in epidemiological environments, mainly in developed countries. Their trends are described and the general patterns are discussed. The attention then turns to the related concept of health transitions and their relationships with the other transitions. In the final part of this chapter, the various transition theories and their major components are synthesised into a general transition framework. From this some key theoretical arguments for this thesis are formulated.

2.1 The Demographic Transition

The concept of demographic transition recapitulates the dramatic shift of populations from high mortality and fertility to low mortality and fertility, a change which coincides with the modernisation process. The term was first used by Warren Thompson in 1929, but entered general use with Notestein's elaborations of it in 1945 (Hodgson, 1983:7; Lassonde, 1997:16; see also McNamara, 1982) to describe demographic changes of Western Europe at the time. The concept has since been applied to both the developed and the developing countries.

The demographic transition theory was formulated as a three stage model, as is illustrated in Figure 2.1. Stage one represents a phase of equilibrium when both birth rates and death rates were high, leading to quasi-stationarity. Stage two is a transitional stage of rapid population growth. The death rate falls, the birth rate stays high but then falls later. Stage three is a new balanced stage with low levels of mortality and fertility, and receding prospects for future population growth.

Figure 2.1 A three stage model of demographic transition



Source: Modified from Keyfitz (1977)

The widening "demographic gap" between birth rates and death rates gave rise to accelerated population increase. The demographic transition also had other consequences, including changes in the age and sex structure and in the dependency ratios of the population.

Contrary to conventional belief, mortality declines in the early part of the demographic transition tended to produce a younger population structure (Coale, 1974). This was because mortality declines at that time were highly age-selective, mainly concentrated at younger ages, especially the first few years of life. "New survivors", referring to those who would have died had it not been for the declines in mortality, were mostly those below the average age of the population. More of these "new survivors" survived to the reproductive ages and were able to raise more children. This tendency towards a younger population age structure was not very strong in the developed countries because of the fall in the fertility rate. By slowing down the rate of introduction of newborns into the population, the proportion of the overall population composed of older members increased, raising the average age of the population. This is termed the "structural ageing" of the population (Pool, 1994).

At later stages of the demographic transition, especially in the now developed countries, improvements in survivorship started to spread progressively to middle then to later adult ages. The improved survivorship at adult ages was further compounded by the large post Second World War birth cohorts, mainly in English-speaking countries, known as the

"baby-boomers". The combined effects of improvements in survivorship and large birth-cohort sizes are leading to an accelerated increase in the absolute number of older people in the population. This is termed the "numerical ageing" of the population (Pool, 1994), or simply "the momentum of growth" (Rowland, 1996).

Population ageing, in both the structural and numerical senses, has far-reaching socio-economic and political implications. The chief driving forces behind numerical ageing and its impacts on population health constitutes the subject matter of the present study. A sub-theme is the effect of improved survivorship and thus life expectancy on changes in age structure, especially within older ages.

2.1.1 The Fertility Transition

The mortality component of the demographic transition is discussed in detail in Section 2.2 on the epidemiolgoic transition. The rest of this section is devoted to a discussion of the fertility transition in relation to changes in population health. The most important linkages are the effects of changes in fertility rates on the age structure of the population as noted earlier and the delicate interactions between the level of mortality and the level of fertility.

It has been argued that in much of the twentieth century, the dramatic reductions in fertility in countries as they underwent the demographic transition has been the prime determinant of demographic patterns and trends worldwide (Myers, 1989; United Nations, 1992). If fertility were to remain low in the future, mortality, particularly at older ages, would resume its major role in population dynamics (Myers, 1989; Pool, 1994). This is already happening in most developed countries as exemplified by the structural as well as numerical ageing of the populations.

From an evolutionary perspective, in the long run the level of fertility is likely to be dominated by the ruling mortality environment. In a high mortality setting such as during the pre-transitional era, fertility rate must be maintained at a high level to avoid the otherwise inevitable extinction of the species. A low mortality environment, on the other

hand, cannot support an exponential population increase for any substantial period of time. Prolonged geometric increases or decreases in population size are clearly unsustainable, and the level of fertility, sooner or later, must respond as a controlling mechanism to changing population dynamics. This inherent controlling mechanistic nature of fertility was well illustrated by the widespread introduction of family planning programmes in developing countries undergoing the second stage of demographic transition. At the other extreme, after 20 years of net reproduction rates lower than the replacement level, a renewed interest aiming at increasing fertility level has developed in many European countries. Some demographers called the decline to sub-replacement fertility the "second demographic transition" (United Nations, 1992:60; van de Kaa, 1987).

From a societal and cultural perspective, the lead-lag relationship between level of mortality and level of fertility is more complicated than may have been implied in Figure 2.1. The redistribution of the age composition of the population and greater longevity enjoyed by females at reproductive ages should have favoured high birth rates. However, social changes emerged from the industrial revolution, for example the demand for a larger female workforce, had redefined the general perception on the economics of child-bearing. A fall in fertility shortly followed, and the momentum swung to an older population structure. Reduced rates of repeated pregnancy also contributed directly to mortality declines of females at menopausal ages.

Like any other transition, the fertility transition occurred over a long period of time. The process is, to a large extent, governed by the prevailing culture, which in itself is constantly changing. High or low levels of fertility are themselves strongly resistant to change. In many typically peasant societies, traditional values encourage the formation of large family. Departures from traditions require changes in the old order, and are often met with resistance. Similarly, attempts to reverse the decreasing fertility trend in some European countries require a new revolution in social attitudes towards child-bearing. Most governments' recent pronatalistic policies have at most temporarily deflected the downward fertility trend to a barely discernible extent (van de Kaa, 1987).

2.2 The Epidemiologic Transition

In retrospect, the secular decline in mortality over the last century primarily reflects a shift in the patterns of causes of death, from infectious diseases to chronic degenerative diseases. The term, epidemiologic transition, was proposed by Omran (1971) and since updated (1982) to delineate this shift in disease patterns. It was originally an academic exercise, set forth to "describe and disentangle the determinants and consequences of changing disease patterns that have accompanied modernisation in most Western countries" (Omran, 1971:511).

The concept of epidemiologic transition provides a contemporary framework which can be applied to the study of population health dynamics, and their interactions with the demographic, socio-economic, policy and eco-biologic environments in different time spaces. The more recent trends identified in the model are at the heart of the present study, and therefore a more comprehensive examination of the epidemiologic transition is provided in this chapter.

2.2.1 The Epidemiologic Transition Theory

The epidemiologic transition theory is essentially disease pattern oriented. It describes the process of transformation of a population from a state of high levels of mortality, dominated by infectious diseases at younger ages, to a state of low levels of mortality due mainly to chronic degenerative diseases at older ages. Like the demographic transition, the epidemiologic transition theory was originally constructed based upon the experience of the West, and drew a close association between the epidemiologic transition and trends in economic and social modernisation. That said, Omran (1982) did give it a far more universal currency by identifying a number of different transitions.

The essential structure of the epidemiologic transition theory as proposed by Omran (1971) is in the form of five basic propositions. The first of these claims that mortality is a fundamental factor in population dynamics as population growth is determined by the difference between the birth and the death rates. As argued in Section 2.1.1, in the long

run fertility is likely to be the dynamic controlling mechanism in response to the changing levels of mortality. Omran observed that, before the middle of the eighteenth century, mortality had moved in a rather "trendless" fashion, with fluctuations in levels being far more extreme than was true for the course of fertility. A reduction in the degree of fluctuation, and a gradual decline in the middle of the eighteenth century produced the initial period of sustained population growth. An accelerated growth starting the turn of the nineteenth century, when the death rate was gradually declining, resulted in a widening vital gap as illustrated in Figure 2.1.

The second proposition is central to Omran's transition theory. It states a long term trend in mortality and diseases patterns, where what he termed degenerative and "man-made" diseases gradually displace infectious diseases as the leading forms of morbidity and primary causes of death. Such a shift is progressive but not complete, and it takes the form of three successive stages:

1. "*The Age of Pestilence and Famine*" - when mortality is high and fluctuating, life expectancy at birth is about 20 years, and fluctuations in levels of mortality is the major causes of changes in population growth rates;
2. "*The Age of Receding Pandemics*"- when mortality falls at an accelerating pace while fertility is still high or lagging behind on its downward path, so that natural increase begins to follow an exponentially increasing curve; and
3. "*The Age of Degenerative and Man-Made Diseases*" - when mortality stabilises at a low level, life expectancy at birth increases to 70 plus years, and fertility assumes a crucial role in trends of natural increase

According to the third proposition, the epidemiologic transition is particularly beneficial to children and to females at adolescent and reproductive ages. This is largely due to their high susceptibility to infectious and deficiency diseases and for females decreases in maternal mortality. Social class differentials are often maintained and the affluent and more privileged are advantaged. Latin American demographers (Frenk et al, 1989) have elaborated this into the sub-theory of epidemiological polarisation. This is the implicit recognition of a factor to be analysed further next chapter (Three) - the notion of heterogeneity in population health trends, and thus an understanding at an aggregate

level that health and the biological process of senescence (see Chapter Three) are both factors which are subject to wide individual and inter-group variations.

Omran's fourth proposition holds that the transitions took off in the nineteenth century and were linked to rising standards of living and improved nutrition, which together constitute modernisation. In comparison, twentieth century transitions in the developing regions are initiated by imported medical technology and public health determinants, which are mostly independent of the socio-economic conditions of the countries concerned. Further improvements come from the combined effects of public health care and socio-economic progress.

The fifth proposition argues that there are four basic models defined by variations in patterns; in pace of change; in their determinants and in their consequences for population change. These are:

1. *Classical or Western Model* - describes the gradual change in the developed Western societies over the past two centuries;
2. *Accelerated Variant of the Classical Model* - describes the mortality transition in non-Western European developed countries occurring over a relatively short time frame, and, in most cases, shortly after modernisation had begun in the twentieth century;
3. *Delayed Model* - describes the changes in most of the Third World countries where transitions emerged largely as a product of international co-operation, so that progress in mortality declines has typically outpaced the rise in living standards; and
4. *Transitional Variant of the Delayed Model* - describes some developing countries where fertility and socio-economic development have responded to the rapid fall of mortality.

By 1950, virtually all the countries classified by the United Nations as developed had "completed" the epidemiologic transition, although there were large discrepancies in the timing and magnitude of these. Broadly speaking, these countries can be divided into two major categories according to their starting point and the pace of change. The first group

includes those in Northern and Western Europe, plus the United States, Canada, Australia and New Zealand (at least the non-Maori population, Pool, 1994), whose transitions are best described by the Classical Model. The second group consists of the Eastern (include the former Soviet Union) and Central Europe, and Japan, which display the features of the Accelerated Variant of the Classical Model. These classifications are based on the majority of the population, but some countries, like New Zealand, have two distinct transitions (Pool, 1993). The mortality transition path of the non-Maori population corresponds to the three-stage Classical model, whereas the Maori population follows the Transitional Variant of the Delayed Model with a later onset of mortality decline but at a much rapid pace.

It is noteworthy that the Transitional Variant of the Delayed Model was not included in Omran's original work (1971) but added in his 1982 update. It was in an answer to the point that sub-models were warranted by the important differences between Latin America, Africa and Asia areas (Omran, 1971).

2.2.2 Historical Trends in the Mortality Transition

In this and the next sub-section, the trends of the two key constituents of the epidemiologic transition are reviewed, namely mortality and morbidity. The focus is on the historical trends as their theoretical backgrounds are provided in the epidemiologic transition theory in Section 2.2.1

Mortality in the pre-modern period can only be estimated from fragmentary documentation. Up until the late eighteenth century, the essential Malthusian check - famine, diseases, and wars - generally applied to areas of the European settlements. Mortality was high, and so was fertility. Demographic historians put the maximum life expectancy at birth in that period at 40 years (Stolnitz, 1955; see also Livi-Bacci, 1968; cited in Pool, 1991:77). During this era periods of crisis mortality led to large fluctuations in level of mortality.

Not until the late eighteenth century did the fluctuations in mortality level gradually diminish, giving rise to a slight decline in death rates between 1780 and 1830. From then mortality trends in some European countries, like France and England, entered a phase of stagnation, reflecting the negative impacts of rapid industrialisation on the populations' health. Mortality in Scandinavian countries continued to fall.

The rise in life expectancy at birth became marked again late in the nineteenth century. In most developed countries, improvements since the 1890s have been more than double the gains over the preceding half century; reaching 50 by 1900 and 70 by 1950 (Preston, 1977; United Nations, 1973). By far the largest gain took place in the 30 years ending in the First World War. Countries fitting the Accelerated Variant of the Classical Model of Omran's epidemiologic transition had a comparatively late onset, but more rapid pace of mortality decline. For example, mortality decline in Italy began in 1875, and in the next 55 years, the crude death rate had dropped from 30 per 1,000 to around 15, a change which had taken 150 years in France, and 125 years in England to accomplish (United Nations, 1973). Like most of its Eastern European counterparts, Japan's major developments came much later yet much faster.

Declines in overall mortality rates in developed countries have levelled off in the 1960s, with life expectancy at birth ranging from 65 to 75. This shift was followed by some minor reverses in certain age-sex groups. Deaths from parasitic and infectious diseases were at low levels and were no longer significantly influencing general mortality trends. From the 1970s, however, moderate but steady declines in mortality became observable at older ages. This latter trend, which has persisted to this day, is the focus of the present study. The theoretical implications of these recent mortality trends are analysed in Chapter Four.

2.2.3 Historical Trends in the Morbidity Transition

Due to inherent difficulties in the collection of morbidity data and its subsequent measurement, far more is said and known about changes in mortality than about morbidity. Data regarding population morbidity experiences in the nineteenth and the early

twentieth century are scarce. Some investigators draw upon British insurance records, insurance claims and the records on amounts of work loss, reflecting the ill-health in the male labour force (Alter and Riley, 1989; Riley, 1990b). Although the data covered only the working class at the time, it has, nevertheless, shed some valuable light on this subject.

Up until the eve of receding pandemics, morbidity generally followed the course of mortality, varying greatly from year to year. This fluctuation declined over time, similar to the pattern observed in mortality. During this period, acute sickness tended to dominate mainly because of the younger age structure of the population, and the disease profile to which the population was exposed to.

After the middle of the nineteenth century, fluctuations in morbidity levels reduced slowly. The available records show a strong upward trend in age specific morbidity rate after 1870 when the mortality decline was already under way. While the incidence and the fatality of illness and injures decreased, the duration of morbidity, especially protracted illness, increased at every age (Riley, 1990a). Studies suggest that the disease profile remained much the same, but their lethality had reduced. People lived longer in some morbid conditions, and recovered more often than in the earlier periods. Riley (1990a) concludes that there was a change in the age structure of death rather than in the disease profile.

At the turn of the twentieth century mortality had declined and stabilised at low levels. Morbidity, on the other hand, had evolved into complex configurations: the incidence of morbidity remained stable or declined slightly and the duration of morbidity continued to increase at all ages. The incidence of acute sickness fell, whereas the prevalence of chronic, often non-fatal, morbidity increased substantially, largely due to the progressive elimination of other risks and increased heterogeneity in the older population. Morbidity had become increasingly more protracted, and increases in the duration of morbidity contributed to the sharp rise in the overall level of morbidity, despite a declining trend in the incidence rates at that time.

Morbidity data started to be collected regularly and systematically in many developed countries from the end of Second World War. Studies in most of the developed countries since the 1950s indicate a continuous increase in morbidity, whereas these same countries

also enjoyed a significant decline in mortality (Alter and Riley, 1989; Cole, 1974; Olshansky and Ault, 1986; Riley, 1990a; 1990b; Verbrugge, 1984). The largest increase in morbidity again occurred at older age groups. Based on the United States prevalence rates in the 1950s and 1960s, Cole (1974) concludes that the consistent rises in the prevalence rate of chronic infirmity is unmistakable. On the whole, the trend of increasing morbidity has lasted to this day. Factors contributing to this apparent rise in morbidity included earlier and better diagnosis of chronic disease; better awareness of the public about morbid conditions, completed with rising expectations that "healthiness" was an attainable norm and thus that a remedy must be sought for any shortfall below that norm; changes in the classifications (for example, a lower threshold) of people suffering minor non-life threatening conditions; and more willingness to seek professional help on the part of the individuals. The more recent morbidity trends and theories developed to explain them are provided in Chapter Five.

While there has been some theoretical development, research, at least at population health (as against epidemiological) level has often fallen behind. In part, this is because, compared to those on mortality, data on morbidity are still generally inadequate for comprehensive studies. Moreover the necessary methodologies are continuously evolving, and, not surprisingly, controversies often arise surrounding the interpretations of current trends and future projections. Considering the problems inherent in morbidity measurements Olshansky and Ault (1986) have warned about difficulties inherent in gauging morbidity trends in the future.

2.2.4 Interactions Between Mortality and Morbidity

Given the general lack of morbidity data, it is not surprising that studies on the historic interactions between mortality and morbidity at a national level are rare. The relationship between mortality and morbidity once looked simple. Deducing from the hypothesis that death relates directly to illness, declining mortality rates would imply improvements in population health and a transformation towards low morbidity. This led to the assumption that mortality and morbidity moved in tandem with each other.

In contrast, however, empirically based analyses suggest that mortality and morbidity progressed on paths quite opposite to each other, as has been outlined in Sections 2.2.2 and 2.2.3. Mortality declines over the last century and a half were in fact strongly associated with morbidity increases. The phenomenon of an "inverse association" between mortality and morbidity (Alter and Riley, 1989) is a logical outcome of the combined effect of shifts in the age composition of the population and in the disease profile, and the persistent decline in mortality at older ages. The latest social and medical developments are often more effective in reducing the fatality than the incidence of diseases. As a result, cause of death pattern tends to diverge from the reigning morbidity profile.

Complex interactions between mortality and morbidity have significant effects on the evolution of population health, and serve to highlight the complexities of the health transition. One significant relationship between mortality and morbidity which underlines this is that the level of mortality is a key determinant of the level of morbidity, but the reverse does not necessarily hold true. High mortality levels leave fewer years for the population to be exposed to the risk of developing morbid conditions. Lower mortality levels, on the other hand, are more likely to preserve and prolong lives in a state of episodic morbidity.

Increased survival at older ages has increased the average frailty of the older population. A reverse selection effect is examined by many authors (Alter and Riley, 1989; Kannisto, 1988; Manton, 1982). Genetically weaker individuals survive longer but in turn are subject to higher rates of disease incidence. However, the rising morbidity as a result of this reverse selection effect can also be expected to be partially offset by improvements in cohort health experiences. These issues, discussed here in terms of the notion of population heterogeneity, are investigated in Chapter Three.

2.2.5 A Critical Review of the Epidemiologic Transition Theory

A shift in the patterns of cause of death towards degenerative diseases, as a direct result of reductions in other causes, is a principal feature of the epidemiologic transition. This pattern is often accompanied by a change in the age distribution of death, because

parasitic, infectious diseases, on the one hand, and chronic, degenerative diseases, on the other hand, affect people at different ends of the age spectrum. Following the third of Omran's proposition (that "the most profound changes in health and disease patterns obtain among children and young women", 1971:521), the shift-share in causes of death has redistributed deaths from the young to the old; and also from females at maternal ages to females at older ages. In fact, improvements in maternal mortality usually come earlier than those in infant and childhood. Fertility may rise in the early stage of the transition. As a result of the changes in risk factors and the subsequent redistribution of age at death, life expectancy at all ages makes substantial, but not uniform, improvements. The largest gains in number of years of life are concentrated among children, and females at child-bearing ages.

In his 1982 update, Omran acknowledged the importance of medical advances, health care, life-style and environmental changes in delivering further gains in life expectancy. Consequently, widening mortality differentials are expected to converge once public health programmes can reach the vast majority of the population at a low cost. Moreover, as further improvements become difficult and take longer to achieve, socially disadvantaged groups will catch up. However, directions in government's health policy plays a crucial role in determining the longer term outcomes.

The epidemiologic transition theory poses a picture of a sequential three stage transition. Omran implicitly dismissed the possibility of over-lapping stages, and effectively implied an orderly, non-reversible sequence of population evolution. Thus the third and final stage represents the end stage of transition. Mortality is low and stable, whilst fertility is low but variable, as occurred for example, in the short lived baby boom which followed the Two World Wars. Life expectancy at birth approximates biological limits, and further gains will be harder to achieve.

The notion of three successive stages of the epidemiologic transition has, however, been criticised on the ground that it implies an orderly, progressive and uni-directional sequence of changes. Omran's deduction of a high mortality of the Age of Pestilence and Famine is labelled by Heenan (1990) as "naive expectation". In fact, according to archaeological evidence, pre-historic hunting and gathering populations might have had a

higher expectation of life than agrarians societies because the spread of acute person-to-person infections were facilitated only by the dense habitation pattern of the latter populations (Stolnitz, 1955). Moreover, from the end of the Middle Ages exploration and mass inter-continental migration and settlement mainly from Europe brought indigenous people, such as Maori in New Zealand, into contact with diseases against which they had no immunity (Crosby, 1986). Consequently, life expectancy at birth of indigenous population actually fell (Pool, 1991).

Furthermore, the theory implies that the epidemiologic transition, parallelled by modernisation, is a sign of societal progress. This view is reinforced by its uni-directional perspective. This assumption ignores the possible effects of, however, many negative aspects of modernisation on public health. In particular, rapid urbanisation at the onset of industrialisation even in developed countries was detrimental to community health. While it is also doubtful if mortality related to occupational hazards, mental stress and work accidents can be considered a more civilised form of death (Frenk et al, 1989).

As new developments unveiled, several aspects of Omran's theory have often been challenged and various modifications have been proposed. The third stage of his model is seemingly exclusive and conclusive. These features are in sharp contrast with the dynamic nature of the epidemiologic environment, where attributes often regroup into new configurations. For example, the theory concentrates on two types of deaths, namely infectious, and chronic degenerative, but masks interactions between these two categories (Rogers and Hackenberg, 1987). Scientific and technological advances have in fact provided the opportunities for the interplay of infectious and chronic diseases. Sufferers of one chronic disease often die of other complications, or from an acute respiratory, gastro intestinal or other type of infectious diseases. Similarly, the theory fails to foresee the decreases in some chronic, degenerative deaths such as ischaemic heart disease (Olshansky and Ault, 1986; Rogers and Hackenberg, 1987).

In the light of recent studies based on the disease profiles affecting contemporary populations in developed regions, it has been suggested that life has been prolonged, but more often in a state of worsening health (Gruenberg, 1977; Kramer, 1980; Robine and Ritchie, 1993; Verbrugge, 1984). The degenerative diseases commonly involve some

type of wearing out of the organism, enough to restrict sufferers from performing certain daily activities. It must also be noted that behaviour-related mortality, including violent deaths, sexually transmitted diseases (AIDS), alcoholism, and drug abuse, typically affecting traditionally low-risk groups have become the leading causes of death (Rogers and Hackenberg, 1987). In fact, in 1984 four of the 10 leading causes of death in the United States were termed "social pathological" in nature (Heenan, 1990:304).

The explanations of these changes have produced a great deal of controversy among researchers. One result of this is that for some commentators the need for a fourth stage for Omran's model is often suggested. This concern is generally based on a number of factors: a declining rate of death from degenerative ailments; the continuous improvement in life expectancy, especially at older ages; diminishing class and gender differentials; and the increasing influences of individual behaviours and life-styles.

Suggested fourth stages have come in different forms, reflecting the differences in the authors' perceptions on the mechanics behind these developments. Rogers and Hackenberg, for example, suggested a "Hybristic Stage" where individual behaviours and life-styles are largely responsible for changes (1987). This view is dismissed by Trlin (1994), who argues that this puts the blame on the victim for adopting their life style and is thus morally irresponsible. Instead, he proposed the "Age of Preventable Diseases Maintained or Generated by Social Forces". Drawing on recent experiences in the United States, Olshansky and Ault (1986) advance a more generalised extension of Omran's model - "the Age of Delayed Degenerative Diseases" - a stage distinguished by rapid mortality declines in advanced ages. Deaths are delayed because of the postponement of the ages at which degenerative diseases tend to kill. This point on the extension of Omran's epidemiologic transition model is revisited latter in Section 2.4 below.

2.3 The Health Transition

The concept of health transition was first proposed by Lerner in 1973 (cited in Frenk *et al.*, 1989; 1991) and later brought into the limelight by Caldwell (1990). The emergence of the notion of health transition and its research is a direct result of observations about

changes in the epidemiologic environment, and by social responses to these changes. The concept of health transition attempts to model the changes in population health status as well as associated social and behavioural changes. Murray and Chen (1992) saw the health transition as a convenient concept that is meant to capture the changing pattern of morbidity and mortality, the socio-economic determinants of such change, and the response of the health care system. It is a dynamic concept, tracing the long term changes in the patterns of death and diseases and the accompanying transformations in demographic, social and economic structures. That said, the identification of the major forces behind the health transition has been a source of continuous debate. The task is inherently problematic methodologically, while the data available for conducting analyses of this issue fall short of adequate.

This section reviews some major changes observed during the health transition in many developed countries. Major epidemiological changes have already been discussed in Section 2.2. The focus of this present section is therefore on the accompanying "social and behavioural changes" in addition to those already discussed in the last section.

Capturing the essence of Omran's epidemiologic transition and relating them to their concomitant expression in health problem and services, Frenk and colleagues (1989) further specified that two transitions form the backbone of the health transition. The first deals with changes in the *health conditions* at the population level, that is, a transition covering, strictly speaking, changes in the epidemiologic profile of the population expressed in terms of mortality and morbidity statistics. The second refers to the organised *social response* to these changes, in the form of the health care system. This was coined the "health care transition". They suggested that it started between the early and middle of the twentieth century with the adoption of a technology-driven, hospital based model, aided by the later development of a community-based comprehensive approach to health protection and preservation. These strategies form a combined response to the epidemiologic transition, and to related socio-economic, political and technological changes (Frenk et al, 1989).

There are high levels of interaction between the epidemiologic transition and the health care transition. On the one hand, the health care transition was part of public health

reforms, making a direct contribution to the advances in life expectancy and to a rise in community awareness about health matters. Its impact on the epidemiologic transition is reflected in reductions in case-fatality rates, which brought mortality to a lower level and, at the same time, fuelled the upsurge of morbidity. These issues are analysed empirically for non-Maori in Chapter Nine of this thesis, using historical mortality and hospital utilisation data. On the other hand, the improved expectation of life has increased the exposure of the population to other risk factors and contacts with the health care system. Increases in morbidity, a prominent feature in the third stage of the epidemiologic transition, demands far more and better delivery of the social health care services.

2.3.1 Changing Concepts of Health

Accompanying these transitions are marked changes in the concept of what is population health. Under pre-transitional high mortality regimes, the major concern was the fight against communicable diseases which had ruled the lives of most people. The concept of health was typically expressed as *the ability to survive*. Following dramatic declines in mortality and shifts in disease patterns from parasitic, infectious diseases to chronic, degenerative diseases, the community's perception of health had been reshaped substantially, transforming from a passive stance of mere survival to a more positive one - *the quality of life*. What eludes researchers nowadays is the question of whether the additional years gained are spent in a state of good health or prolonged suffering of chronic degenerative conditions. This issue is central to public health research as it has caused us to reflect on the consequences of public health policy on the evolution of health, and on the selection of future health targets (Robine and Ritchie, 1993), to produce decreases in death rates, or improvements in quality of life for those surviving.

The multi-dimensionality of health becomes more evident as morbidity assumes an increasingly prominent role. The concept of health has changed as a function of the changing socioeconomic, medical, technological, and cultural environments (Riley, 1992). Rising living standards, medical and technological advances and the accompanying increases in level of sophistication in knowledge about health matters together reshape contemporary cultural perceptions of health (Crimmins et al, 1989;1997a; Johansson, 1991). Dramatic changes in

society's attitude towards health and ill-health are one manifestation of these underlying changes. An excellent example of the influence of societal changes on the population health is provided in the case of developing countries. For example, studies on health issues in Africa have shown that diarrhoea is accepted as a part of normal childhood development (Yoder, 1991). A simple conceptual alteration by imposing "Western" medical thinking on the locals would profoundly change their perceptions and possibly their responses.

Changes are also generated through government policies. Public health care provision has been high on the government's health planning agendas in many developed countries. Burdens traditionally born by the family have been shifted towards the wider community. The community as whole is more willing to look after the sick and the old, as is reflected in declines in discrimination against people with long term disability. Recently, however, this has been challenged because of the rapidly rising costs of health care, plus a generic shift away from a Keynesian to a market perspective (emphasising personal and family responsibility) for social welfare and health.

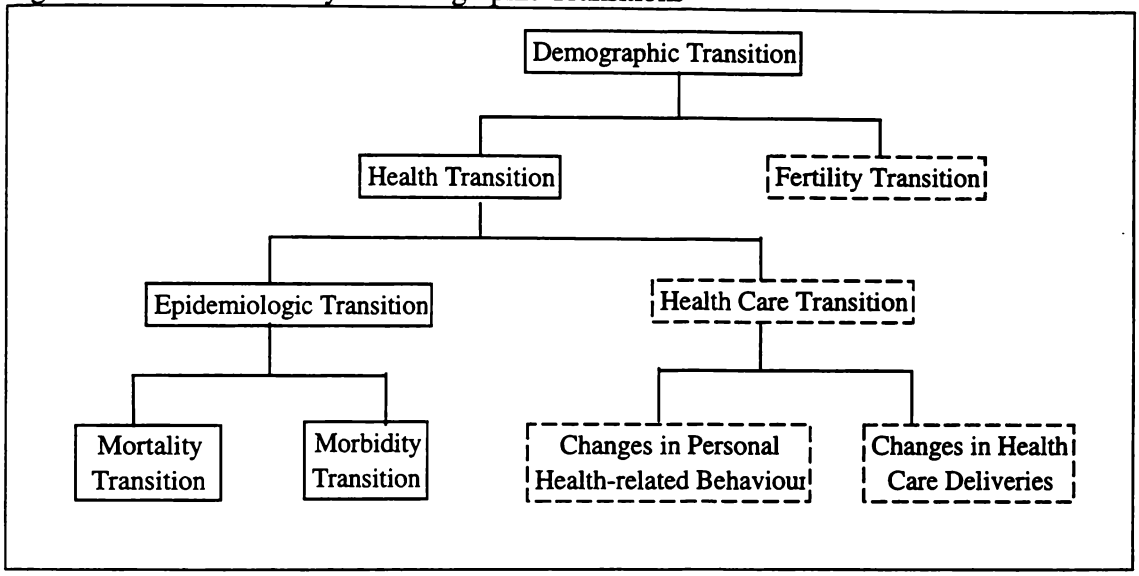
2.4 Discussion

In summary, both the epidemiologic transition and the health transition are developed from general transition theory in demography. Since its publication, the concept of epidemiologic transition has often been interpreted in a rather loose manner. In particular, the epidemiologic transition and the health transition have been used interchangeably despite conceptual differences. One common misinterpretation holds that the health transition is essentially the epidemiologic transition with more emphases on the upsurge of the morbidity trend. Instead, as eloquently summarised by Caldwell, the concept of health transition intends to emphasis "social and behavioral changes which parallel the epidemiological transition and may do much to propel it" (1990:xi).

To put the various transitions into their broader context and into a unified perspective, a transition framework nested into the demographic transition theory is developed by the present author. This is presented in Figure 2.2, which shows the components of major transitions, and, in doing so, differentiates the various dimensions. For example, the two

transitions which make up the health transition are clearly shown, and hence the epidemiological changes and the parallel social changes are given equal emphasis in considering the health transition. Boxes with solid boundaries in Figure 2.2 indicate areas which are elaborated in this and/or the subsequent chapters of this thesis; and boxes with dash boundaries indicate otherwise. It is not the intention of Figure 2.2 to describe the complete matrix of complex inter-relationships between these various transitions, although some of the inter-relationships have been discussed earlier in this chapter, as for example, that between fertility, and mortality and morbidity.

Figure 2.2 The Family of Demographic Transitions



Source: Prepared by the author

Focusing on the constituents of the health transition, the epidemiologic transition is an outcome measure of a series of epidemiological changes, incorporating both the mortality and the morbidity transitions. Levels of mortality and morbidity are indicative of changes in the health status of the population. The health care transition, a term borrowed from Frenk et al (1989), reflects medical and social responses of these changes.

This chapter has concentrated on the left hand side of the tree diagram in Figure 2.2, pivoted around the epidemiologic transition, one of the main foci of this chapter, a well-established field of research. Despite its apparent limitations, the theory provides a convenient framework and a platform for the development of the health transition theory.

A transition is not about just any change, but changes which evolves over a reasonably long period of time and follow an identifiable pattern. Two underlying arguments formulated for this thesis by its author are that:

1. a transition is not simply a time oriented sequence of identifiable patterns, but instead a process of change where the exact timing is less important than the patterns of change;
2. a transition is a dynamic process involving multi-level re-configuration of population dynamics; thus a static uni-directional perspective is inadequate to capture the essence of this dynamic process.

A misconception of transition as a time oriented sequence of changes is reinforced by the sequential three stage model by Omran. Transformation of any demographic element, whether it is mortality, morbidity, or fertility, has never been a smooth process. Interruptions in time series trends, large scale differentials between gender or ethnic groups, or even temporary reversals are the standard features of many transitions. By turning away from a time-dependent dimension, one may save the embarrassment of endless variants to the proposed models and be more able to focus on the process of change itself, its determinants and consequences.

This problem arises in other ways. It is implicit, for example, in the notion of epidemiological polarisation noted earlier (Frenk et al, 1989) which infers a link between health and patterns of social stratification. There is, however, another associated question: the way in which a wide range of individual variations in the experience of health affect population-level trends. Thus in the next chapter (Three) this issue will be further elaborated by introducing theoretically the wider notion of "heterogeneity".

The epidemiologic transition and the heath transition evolved from general transition theories. They are dynamic concepts for they focus on the evolution of the health profile of a population. The characteristic of the dynamic process is best demonstrated by the discussion on a fourth stage of the epidemiologic transition (see Section 2.2.5). Common to the various proposals is the feature of a dynamic, instead of a static state. However, most of the proposed models are essentially transitional since longevity cannot grow indefinitely. This point is confirmed by the constructors of one the proposed fourth stage:

"... we know that this fourth stage must eventually come to an end given the likelihood of a finite lifespan, ..."

(Olshansky and Ault, 1986:384)

Provided the fourth stage is not the last, the next stage, if not the final one, must be either a return to the third stage or an advancement into a fifth stage. The former is contrary to Omran's uni-directional progression and the new stage would lack the characteristics of the third. A fifth stage is more likely, characterised by dynamic equilibrium levels of mortality and morbidity, and life expectancy at birth reaching the biological limits to life. This may signal the start of another major transition in population health.

This hypothesis carries profound implications for contemporary health policy issues, such as dilemmas posed by the higher levels of morbidity in the immediate future due to the postponement of death, and questions about the feasibility of pouring in more resources for diminishing returns in terms of quality health. This position also opens up theoretical, empirical and philosophical questions about human longevity. The next chapter (Three) examines these questions.

CHAPTER THREE
DISTINGUISHING BETWEEN SENESCENCE AT AN INDIVIDUAL LEVEL AND AT A POPULATION LEVEL

The phenomenon of population ageing as introduced in Chapter One is gathering momentum in most developed countries and is also becoming perceptible in many developing countries. For policy purposes there is thus a need to study macro-level mortality and morbidity patterns and trends in order to measure population health, particularly for an ageing population

Apart from often rather naive basic demographic analyses, for example the dependency ratio, much of the policy concern over ageing is typically based around models which are often extrapolated out from the micro-level (Pool, 1999a). To look at population health it is thus necessary to review ageing at an individual level, by looking at the mechanisms governing an individual's senescence process, one in which many age-related changes in body composition, physiological functions, disease expressions, leading to the ultimate death. Some understanding of the patterns of senescence at the micro-level and different explanatory paradigms for these underpins any analysis of the linkages between individual trends and macro-level analogues observed when we study population health. This chapter attempts to resolve these concerns before preceding with more specific theoretical issues on population health *per se* in the remaining chapters (Four and Five) of Part Two.

In this chapter, then, the subject matter of human senescence is explored first on a micro-level, from an evolutionary and then a clinical disciplinary perspective. Micro-macro linkages between individual senescence and population health are investigated through demographic elaborations of theories originating from other disciplines. This leads to the introduction of the notion of population heterogeneity which is essential in establishing macro-micro linkages. This concept is based on the recognition of the senescence process of individuals or groups of individuals within any cohort and is thus is central to demographic modelling. The links between individual senescence and population ageing, and the role of heterogeneity are important to an understanding of emerging theoretical issues in mortality and morbidity to be presented in the next two chapters (Four and Five).

3.1 An Micro-Level Overview of the Senescence Process

The central importance of theories of senescence in formulating a framework for the development of a model of human mortality is well recognised (Carnes and Olshansky, 1993; Manton et al, 1991; Nesse, 1984). These theories underpin the validity of many mortality and morbidity models. Without a theory of senescence, much of the current research on future mortality and morbidity developments cannot develop much depth or go much further beyond extrapolations of past and present trends. It is not the intention of this section to critically evaluate the different theories on the subject of senescence, a task which is beyond the scope of this thesis. The purpose instead is to introduce the concept of senescence, the reasons why it occurs, and how it expresses itself on an individual level.

It is critical to make a fundamental conceptual distinction between ageing and senescence. The former is simply *the passage of chronological time* from birth. It is a purely deterministic process, and therefore its observation is relatively straightforward at both the individual and population levels. For the latter, a more rigorous definition puts it as "the time-dependent accumulation of damage at the molecular level that begins at fertilisation and is eventually expressed as nonspecific vulnerability, impaired function, disease, and ultimately death" (Carnes and Olshansky, 1993:795). Thus senescence is a stochastic process governed by a combination of biological, genetic, behavioural and environmental factors. It is most conveniently viewed as *the passage of biological time*, but its stochastic property deems its precise measurement impossible at present. In practice, senescence is often defined somewhat tautologically in terms of systematic increments in the age-specific mortality rates which are suggestive of the presence of senescence in the population. The theoretical justification of this starting point is questioned by some (Carey et al, 1992).

Senescence is accepted as a standard feature of the life histories of higher animals (Kirkwood and Rose, 1991). The evolutionary theory of senescence, developed from biology, bases its arguments on the declining force of natural selection at adulthood and the importance of successful reproductions to ensure ultimate survival of the species. Contrary to popular belief, in this paradigm senescence is not the result of some

predetermined genetic programme, but the combined effect of rapidly diminishing force of natural selection and survival beyond the reproductive ages. Past the reproductive period, the force of natural selection is considerably weakened or non-existent, and therefore the body is allowed to maintain its biological functioning, but with declining efficiency. Senescence is therefore the inadvertent consequence of the diminishing force of natural selection before death occurs.

Other commentators view human senescence from a clinical perspective, through various forms of expression of disease. This is the most convenient and the most popular starting point of many contemporary research. It is generally believed that functional losses at old ages are due to increased susceptibility to diseases or a reduction in the efficiency of homoeostasis maintenance (the tendency to maintain a constant internal environment regardless of varying external conditions) after the cessation of physiological growth. Deteriorations in the measurable parameters of biological functioning thus correspond to the cycle of growth and development. Since most of the clinically observable deteriorating biological functioning are highly age-related, this construct leads to the formulation of the concept of normality of ageing which is discussed latter in the chapter. The amendability of, or the postponement of, the senescence process is a major feature of the clinical paradigm.

The rest of this section discusses micro-level explanations for the senescence of individuals from evolutionary and clinical paradigms. The questions to be addressed are: how senescence became part of human life history; and how this phenomenon is understood on a clinical level.

3.1.1 Explanations of the Senescence Process from the Evolutionary Discipline

The reasons for including evolutionary explanations on human senescence in this section require some further elaboration. First, evolutionary theory serves to place the historical importance of the recent mortality transitions into its proper perspectives. It is indisputable that the dramatic changes in human longevity occurring over the last 100 to 200 years took place over a period which constitutes merely "a blink of an eye on the

evolutionary time scale" (Carnes and Olshansky, 1993:793) and that its historical significance is yet to be fully appreciated. Survival much beyond reproductive ages has historically been limited to a minute fraction of the population. The combination of high attrition through death at birth and during infancy, and a low probability of surviving to older ages means that little historical information is left in this area for researchers (Williams, 1957). Recognising the historical unstable trend of high mortality and its recent deviation, over the years proponents of the evolutionary theory have long argued that demographic estimates of future longevity should be considered within the context of the evolution (and survival) of the human species (Carnes and Olshansky, 1993; Kirkwood, 1977; Kirkwood and Holliday, 1979; Kirkwood and Rose, 1991; Medawar, 1946; Sacher, 1978; Williams, 1957).

Second, quantitative mathematical models utilising mortality data are well developed in the field of demography. The basic biological mechanisms influencing senescence and ultimately mortality have not, however, received adequate attention from demographers. In the words of Carnes and Olshansky: "demographers typically do not receive training in the basic biology of the organism upon which their mathematical models are applied - humans" (1993:794). For example, contemporary practice in mortality forecasting relies heavily on the analysis of short-term trends in death statistics, assuming that the future will see some variations of current trends (Carnes and Olshansky, 1993). The historical fact that the mortality changes observed over the last 200 years represent dramatic departures from the patterns of mortality that must have existed for the past 100,000 years is largely overlooked. The situation is more serious than this for many government forecasts base their future mortality assumptions on trends observed during the 15-20 years preceding the forecasts (Olshansky and Carnes, 1994).

It is for these reasons, as was noted in Chapter One, that, where data permit, this thesis has extended out the analysis of mortality by cohort, and even by cause, back into the nineteenth century. It also has formulated a measure of health expectancy integrating mortality and morbidity components which is taken back to 1951, further back than most analyses of this sort go. Cohort based mortality projections to be presented in Chapter Fourteen are based on data spanning over a century and therefore represent a major methodological advancement from extrapolation of trends over the last few decades.

The underlying premise of the evolutionary theory is that evolution occurs through the changing gene pool of any population over time (Carnes and Olshansky, 1993; Kirkwood, 1977; Kirkwood and Rose, 1991; Sacher, 1978; Williams, 1957). In sexually reproducing species like humans, every individual carries a unique assemblage (genotype) of the available genes which make up the genetic composition of the population. The genetic composition is slowly changing through differentials in the survival and reproduction of each individuals. For a specific gene to remain in the pool over time, successful reproduction by the individuals who possess the gene is critical. Evolution operates through the primary mechanism of natural selection, which is characterised by differentials in the survival and reproduction of individuals with different genetic (heritable) traits. The evolutionary process systematically eliminates weaker individuals through mortality differentials before they are able to reproduce.

It is argued that the intensity of natural selection is at its greatest before reproduction begins, decreases as the reproductive potential of the individual is achieved, and is at its weakest after reproduction is accomplished (Williams, 1957). Mortality differentials operating after the reproductive ages bear no effect on the composition of the gene pool of the generations that follow. The high concentration of the force of natural selection at younger ages is also reflected, in that all sexually reproducing species have invariably adopted the common strategy of early reproduction. A shift toward early reproduction must have been favoured by natural selection.

The evolutionary theories of senescence maintain that there is no predetermined genetic programme responsible for senescence (Christensen and Vaupel, 1996; Sacher, 1978). Instead, senescence is the sacrifice of late survival in favour of enhanced early reproduction. It is postulated that the force of natural selection is weaker or nonexistent at older ages than at younger ages; and the push toward greater longevity incurs a cost to the body's biological mechanisms. Senescence, rather than being part of an optimal evolutionary strategy, thus arises as a by-product of the priority that natural selection places on reproduction (Carnes and Olshansky, 1993; Kirkwood and Rose, 1991).

3.1.2 Clinical Observations of Senescence and the Concept of Normality of Ageing

From a clinical perspective, senescence is often expressed as observable signs of bodily changes (Fries, 1988; Kohn, 1982; Shimokata and Kuzuya, 1993). For example, the cessation of reproduction and the onset of menopause among women are themselves phenomena of biological ageing. The clinical view of human senescence differs from the evolutionary view in that the former generally believes senescence is present in our cells and is sometimes referred to as the genetic clock which also responds to changes in the external environment. It is argued that an individual's overall "genetic frailty" consists of some form of innate frailty obtained at conception and is modified by "acquired frailty" which may be temporary or permanent (Kannisto, 1991). Individual survival relies on the functioning of several vital organs which are not fully developed at birth, but reach their maximum capacities in the early years of life. Biological or physiological ageing, or senescence, is the clinical expression of age-related reductions in the function of these organs. Death occurs upon the breakdown of the weakest vital organ(s) with the lowest level of reserve (Fries, 1983; 1988; Fries and Crapo, 1981).

Clinical explanations of senescence tend to anchor on observable clinical manifestations of diseases. For example, Kohn (1982) re-categorised the conventional classifications of fatal and non-fatal degenerative diseases and acute diseases among the older population into three types of disease and ageing relationships. These are:

1. Diseases that are part of the "normal ageing" process, that satisfy the criteria of progressive and irreversible under normal conditions, and that are widely observable in the population. Examples are arteriosclerosis / atherosclerosis, degenerative joint disease, emphysema, and osteoporosis.
2. Diseases in which the incidence increases with increasing age. The most prominent examples as cause of death are malignant neoplasms and hypertension.
3. Diseases (or processes) that have more severe effect on older suffers than younger ones. Examples are trauma / accidents and infections, particularly respiratory tract infections such as bronchopneumonia and influenza.

An "age-illness" interaction (Kohn, 1982) is inherent in each of the above categories. Clinical opinion maintains that declines of the physiological functions over age can be delayed, and that progress towards disability can be subjected to modification. The plasticity of the "age-illness" interactions would be best illustrated by declining lethality at advanced ages of certain diseases in Kohn's third category, because these diseases are presented among both the young and the old but tend to have a more virulent effect in the latter. In fact, Minaker and Rowe (1985) argued that because of the changing physiologic environment it is possible that many diseases that occur in both the young and the old become less lethal at advanced ages. One of the examples they cite to support their argument is breast cancer which is believed to run a less aggressive course in post-menopausal than pre-menopausal women.

Implicit to Kohn's categorisation is the concept of the normality of ageing which has enjoyed a certain level of acceptance in the fields of physiology, psychology and sociology (Rowe and Kohn, 1987, Shimokata and Kuzuya, 1993). The relationship between age and many clinically relevant variables such as hearing, vision, glucose tolerance, bone density and so on are well documented (Kohn, 1982; Minaker and Rowe, 1985). Changes that occur to the vast majority of the population may represent what is called the "normal ageing" process. This concept is operationalised by excluding individuals whose pathological changes cannot be attributed to ageing *per se*. Part of the operation involves attempts by gerontologists and geriatricians to make clinical divisions between body changes that occur with age and those that are associated with specific disease states.

However, the idea of "normality of ageing" carries two major implications which can be detrimental. First, it implies a false sense of harmlessness or lack of risk as the process is supposed to be natural. Certain physiologic changes such as menopause conform to the notion of the normality of ageing but also carry adverse clinical sequels. While menopause is accepted as part of the female life-cycle, this normal change also brings about increased risk of osteoporosis and atherosclerosis and other symptomatic clinical manifestations. Second, the study of risks associated with "normal ageing" may lead one to conclude that ageing itself, in some ways, represent a disease state or "ageing syndrome" (Kohn, 1982); or similarly, one may hold that what is natural is beyond

purposeful intervention and modification. In some countries ageing is listed as one of the causes of death, though it is infrequently recorded as a cause, partly because ageing is not generally accepted as a genuine cause of death (Minaker and Rowe, 1985). The arguments around the concept of normality of ageing is re-visited later in this chapter.

3.2 Demographic Perspectives on the Impacts of Senescence of Individuals on Population Health

Some researchers (Manton, 1989; Manton et al, 1991; 1997b) have argued that there is an inter-relationship between many of today's major fatal diseases and predisposing risk factors. This delicate inter-relationship is primarily responsible for senescence, and for responses to interventions and mediating factors. Their opinions are categorised by Olshansky and Carnes (1994) as "a demographic perspectives on senescence", for they are developed from a population level modelling, and include in their explanations population level variables in addition to genetic, physiological or biological ones. It is argued that improved risk factors at the population level will translate into better population health. This paradigm has a wide appeal and as a consequence is used to form the bases of health intervention and prevention programmes in many countries.

Micro-level explanations of human senescence explored in Section 3.1 underpin the understanding of its impact on population health. From a demographic perspective, however, susceptibility to diseases and death are filtered through a number of factors in addition to biological ones, including environmental, socio-economical, behavioural. These factors are themselves changing. The conventional ideas that functional loss and impairment are necessary and universal correlates of the senescence process is challenged as being counterproductive (Manton, 1989). It is argued that a sense of senescence as the sum of irreversible, immutable losses of individual functions gives rise to social biases in relation to ageing (Schneider and Reed, 1985). Many negative attitudes toward ageing thus derived leads to the formulation of "a type of self-fulfilling prophecy" (Manton, 1989:14). Roles for the health professionals are passively defined as chiefly supportive to those experiencing such inevitable losses.

The risk factor model of mortality and morbidity developed by Manton (1989) is an extension of purely micro-level explanations of senescence so as to include the wider societal changes and their effects on individuals in determining their biological courses later in life. Risk factors responsible for senescence identified for population sub-groups help to explain increasing population heterogeneity, especially at older ages. Both of these two concepts: risk factors and population heterogeneity, are discussed separately in the rest of this section.

3.2.1 Risk Factor Model

In recognition of the inter-relationship between major fatal diseases and predisposing risk factors, the latter are held responsible as the primary cause of senescence. For example, predisposing risk factors like smoking are closely related to many major fatal diseases, and are clearly amenable to modification. The most prominent feature of the risk factor model is its emphases on early interventions and/or changes in life-style.

Traditional epidemiologic research of chronic degenerative diseases have focused on lethal conditions. This leads to the identification of factors of more fatal diseases such as coronary heart disease, stroke, and cancer. Factors thus identified provide scope for secondary control but much less scope for primary prevention. Once the condition of the acute, lethal diseases manifest at a clinical level, additional pathological processes are initiated that may be more difficult to reverse.

In view of the high prevalence of morbidity due to non-fatal diseases, identifying factors having linkages to chronic degenerative diseases which most affect the active portion of life expectancy assumes a more important role. Exercise, for example, clearly has beneficial effects on certain age-related disorders, such as cardiovascular disease, but its effect on longevity is not conclusive (Christensen and Vaupel, 1996; Schneider and Reed, 1985). Accordingly, changes in the direction of contemporary practice is required not only from the medical profession, but also in institutional responses.

3.2.2 Population Heterogeneity

Population heterogeneity refers to the variability in a population of its individuals' susceptibility to diseases and disorders. It is therefore a population level manifestation of individual variations in senescence processes. Often the variability, or the level of heterogeneity, is considerable at advanced ages.

Heterogeneity as a "congenital endowment for longevity" is a fact in any population (Kannisto, 1991), but the level of variation can be expected to differ from population to population. It can be shown that in two heterogeneous populations with respect to the endowment for longevity of individuals, cross-overs in the age specific mortality rates of the two populations (where mortality rates of one population are higher than the other at younger ages, but lower at advanced ages) can occur if one population has significantly poor mortality rates at early ages (Rogers, 1992; Vaupel et al, 1979; Vaupel and Yashin, 1985). The cross-over is explained by the differential mortality at early ages which selects the least robust persons from the disadvantaged population, leaving proportionately more robust persons at advanced ages.

For reasons of simplicity, most studies of senescence tend to group people by chronological age. Implicitly assumed under this approach, however, are: i) that a linear relationship between age and senescence exists on the level of individuals; and ii) that the same relationship is universally applicable for the entire population. Not only does this confuse senescence with ageing, as defined at the beginning of the chapter, but such practice ignores the fact that dissimilarity is present, and may even increase by age, among the older population. Clinical observations indicate that variability in the degree of both physiologic changes and disease states is greater among the very old ages than in the younger old, making generalisations about their frailty more hazardous. Reasons for the marked variability in the clinical manifestations of a given illness in the older population include variability in underlying physiological changes; other diseases that the individual has accumulated over time, that is, accumulated risk exposure; pattern of responses to illness and interaction with health care professionals that is characteristic of the individual; and varying degrees of severity of patho-physiologic processes (Minaker and Rowe, 1985).

Several demographic studies have identified a deceleration in the rate of mortality increase after age 85 (Gavrilov and Gavrilova, 1991:129; Kannisto, 1988; Manton, 1992; Thatcher, 1981; Vaupel, 1998). A slowing of the mortality increment with age may be an artifact of compositional change in the cohort, resulting from increased heterogeneity and the associated patterns of mortality differential (Carey et al, 1992). The explanations of this and other related phenomena are investigated extensively by Vaupel and Yashin (1985).

The concept of population heterogeneity is included as part of the demographic paradigm of senescence for two major reasons. First, apart from genetic and biologic factors, heterogeneity exists in the older population with respect to a combination of demographic factors such as ethnic, gender, and socio-economic characteristics (Crimmins et al, 1997a; Hayward and Heron, 1999; Manton et al, 1997b; Reynolds et al, 1998). Second, the issue of population heterogeneity has some profound policy implications. Until recently, research on ageing has placed a heavy emphasis on the age-related functional losses and neglected the substantial heterogeneity among the older population (Rowe and Kohn, 1987). It has led to the exaggeration of the ageing process and an over-readiness to treat age as if it were itself a sufficient explanatory variable (see Section 3.1.2), underestimating physiological and personal behavioural factors. Rising awareness of the level of heterogeneity in the older population help to refine many aspects of health policy. The shift from institution-based to more individualised health care provides a good example.

3.3 Discussion

The possibilities of finding some univariate process of intervention by which to arrest senescence is somewhat simplisticly encouraged by proponents of various theories. But it is also argued that a diversity of views on senescence is essential to allow one to unveil multiple explanatory mechanisms operating at different levels of observation (Schneider and Reed, 1985), ranging from a cellular to a population level. Understanding, and viewing as complementary arguments presented from different perspectives can therefore

help us to broaden our knowledge on this subject. In contrast, treating these views as competing theories will only limit our understandings.

Other than the inevitability of senescence among all sexually reproducing species, the evolutionary theories of senescence also have some profound implications for the fine-tuning of demographic models of human longevity. These theories rest upon the linkage between reproduction and senescence, and the onsets of these two processes are predicted to coincide with each other (Carnes and Olshansky, 1993). Two conclusions can be drawn from these theories. First, a biological limit to human longevity is predicted from the diminishing efficiency of somatic maintenance and repair. The law of diminishing returns will ensure that further extension of survival beyond the reproductive period becomes an unending uphill battle. Second, by successfully controlling many of the extrinsic forces affecting mortality, the stage is set for a rapid increases in the prevalence of morbidity. A proliferation of degenerative diseases in the population among survivors beyond the reproductive ages is the natural consequence. Many degenerative diseases associated with senescence inevitably result from the unavoidable declines in physiological functions.

However, not all aspects of these disciplinary theories on senescence are harmonious. For example, the concept of normality of ageing may serve the purpose of much physiologic and psychologic research, but has only limited value for demographic modelling of human longevity. It is argued by the present author that the concept is a rather crude attempt to re-interpret micro-level individual variations in the senescence process as a macro-level phenomenon. Dividing the population into "normal" and "diseased", and clinical findings into "disease-related" and "age-determined" would neglect much of the heterogeneity present in the older population. The concept of heterogeneity more adequately captures the great diversity in population health status.

Central to the notion of population heterogeneity are observed cohort mortality patterns and the effects of their accumulated prior exposure to the risks of diseases and death (Manton and Vaupel, 1995). The example of cross-overs in mortality rates noted earlier is itself an observation of cohort mortality experience. Similarly, deceleration in mortality rate increases at advanced ages can be explained by changes in cohort composition,

which in turn are influenced by the epidemiological environments under which these cohorts received their disease exposure. Failure to recognise population heterogeneity and associated dynamic selection effects may unnecessarily complicate theoretical and empirical research (Rogers, 1992). Vaupel and Yashin point out that the "[n]aive acceptance of observed population patterns may lead to erroneous policy recommendations if an intervention depends on the response of individuals" (1985:176).

In a data environment such as New Zealand where individual-based longitudinal data is lacking for empirical modelling of heterogeneity and thus of frailty, a cohort approach is therefore the closest substitute for disentangling "heterogeneity's ruses" (Vaupel and Yashin, 1985). Macro-level cohort mortality and morbidity patterns and trends are thus, at a population level, the ramifications of individual heterogeneity. Demographic modelling using cohort mortality and morbidity data can integrate the individual probabilistic process of frailty into the concept of population heterogeneity. However, as will be shown later in this thesis, even at a population level empirical cohort studies are also constrained by issues of data availability.

The remaining chapters of Part Two of this thesis will take a theoretical focus on a population level the manifestations of the senescence process. Chapters Four and Five are devoted to detailed discussions on leading contemporary theories on the patterns and trends of mortality and morbidity.

CHAPTER FOUR
CHANGING PATTERNS OF MORTALITY

The central element of Omran's epidemiologic transition model reviewed in Chapter Two is the shift in disease patterns from infectious diseases to chronic degenerative diseases and a subsequent shift in the force of mortality from the young to the old. In the second half of the twentieth century, the age at death distribution in developed countries, and to a lesser extent, in developing countries, has shifted and is becoming concentrated at the upper end of the age spectrum. The focus for population health analyses has also moved up the age scale accordingly.

In this chapter emerging issues in mortality from the shift in mortality patterns are further investigated, drawing on the notion of "compression of mortality" introduced in Chapter One as its framework. The discussion in Chapter Three on the senescence process which governs the individual's mortality path and the population level ramifications of this provides the platform for evaluating the theoretical conjectures presented in this chapter. This chapter also sets the scene for in-depth discussions of the leading theories on morbidity trends in relation to mortality in the next chapter (Five).

The concept of the compression of mortality has been put forward by Fries (1980; 1983; 1988; 1989; 1992) to describe the recent observations of an increasing concentration of age at death at older ages. The idea of a compressed mortality pattern at the very old ages was first raised by Pearl (1940) as one of the theoretical boundaries to observed variations in survival curves in the life table. This idea was later discussed extensively by Comfort (1964). Turning this hypothesis into a broader theoretical construct was achieved by Fries, but in doing so he has created some intense disagreements. Fries went on to put forward his controversial theory of the "compression of morbidity" (1980; 1988; 1992; 1996), basing it around the observed postponement in the onset of long term morbidity to the last few years of life (see Chapter Five). Despite the limited empirical evidence that was available at the time (Myers and Manton, 1984a), Fries (1980; 1983; 1984) took the notion of mortality compression as a given and presented it as a pre-condition to his associated theory on morbidity compression. Some recent population-level studies have lent limited empirical support to the mortality compression hypothesis

(Coale, 1996; Go et al, 1995; Nusselder and Mackenbach, 1996; 1997; Paccaud et al, 1998).

In this chapter, the concept of compression of mortality and its corollaries are critically reviewed. Views from its proponents as well as its opponents are considered. Irrespective of the eventual validation or falsification of Fries' compression of mortality hypothesis, the concept plays a role in its own right for it provides a convenient framework for the analysis of mortality trends and patterns. This framework is further elaborated as schema around which to build analyses in the later chapters of this thesis for the study on New Zealand non-Maori population.

4.1 The Concept of the Compression of Mortality

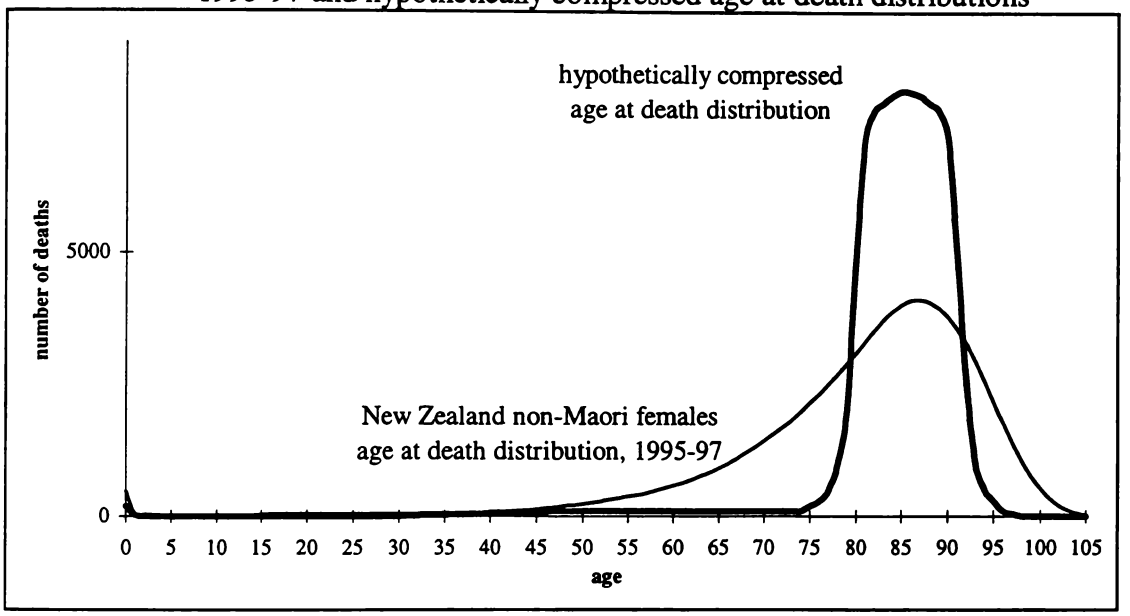
In a series of articles, the first published in 1980, Fries outlined a challenging view about the health of future older populations. He argued that human life span is subjected to a fixed biological limit of approximately 115 years of age, while the average length of life, measured by life expectancy at birth, would be unlikely to exceed 85 years of age. The lower age mark was estimated by extrapolating life expectancy changes at different ages to a point of convergence. A more recent study by Coale (1996), which extrapolated an empirical inverse linear relationship between life expectancy at birth and annual changes in life expectancy at birth, arrived at the same estimate for female life expectancy at birth, at between 84 and 85 years.

The reference point of life expectancy at birth of 85 years of age is rapidly approaching in many low mortality countries. The combination of a fixed boundary and the momentum of the shift in age at death toward this boundary will naturally leads to a congestion of the age at death distribution at around the age of 85 years, and thus the risks of mortality for the majority of each cohort compressed between a narrow range of ages. In its simplest form, the concept of the compression of mortality argues that death is continuously compressed to an upper limit, and the range of age at death becomes increasingly narrower. Fries (1980; 1984) estimated that, under the conditions of a fixed tail of the survival curve and normal distribution of age at death, an age of 100 years is

about four standard deviations from the mean of 85 years. At the final stage of mortality compression under "ideal societal conditions", that is in the absence of premature death (deaths due to diseases or accidents), 66 percent of natural deaths (deaths due to senescent frailty) would occur between ages 81 and 89 years, and 95 percent between ages 77 and 93 years.

The concept is illustrated in Figure 4.1, using a hypothetically compressed age at death distribution from the life table (the ${}_nd_x$ function). To make comparisons with the current situation, the age at death distribution for non-Maori females in 1995-97, representing the lowest overall level of mortality ever achieved in New Zealand, is also included in Figure 4.1. The exact magnitude of the hypothetical curve is of secondary importance to its general shape in comparison with the non-Maori female curve.

Figure 4.1 New Zealand non-Maori females life table age at death distribution in 1995-97 and hypothetically compressed age at death distributions



Sources: Non-Maori female age at death distribution extracted from the 1995-97 complete life table constructed by Statistics New Zealand
The hypothetical age at death distribution, generated by the author based on the curve proposed by Fries (1980)

Compared to the 1995-97 non-Maori female curve, the peak of the hypothetically compressed age at death distribution has a much narrower base of around 15 years. Mortality, instead of being a hazard at all stages of life, is becoming increasingly compressed into the later years, as represented by the much taller and sharper peak of the hypothetical distribution. However, it is worth noticing that non-Maori female deaths at

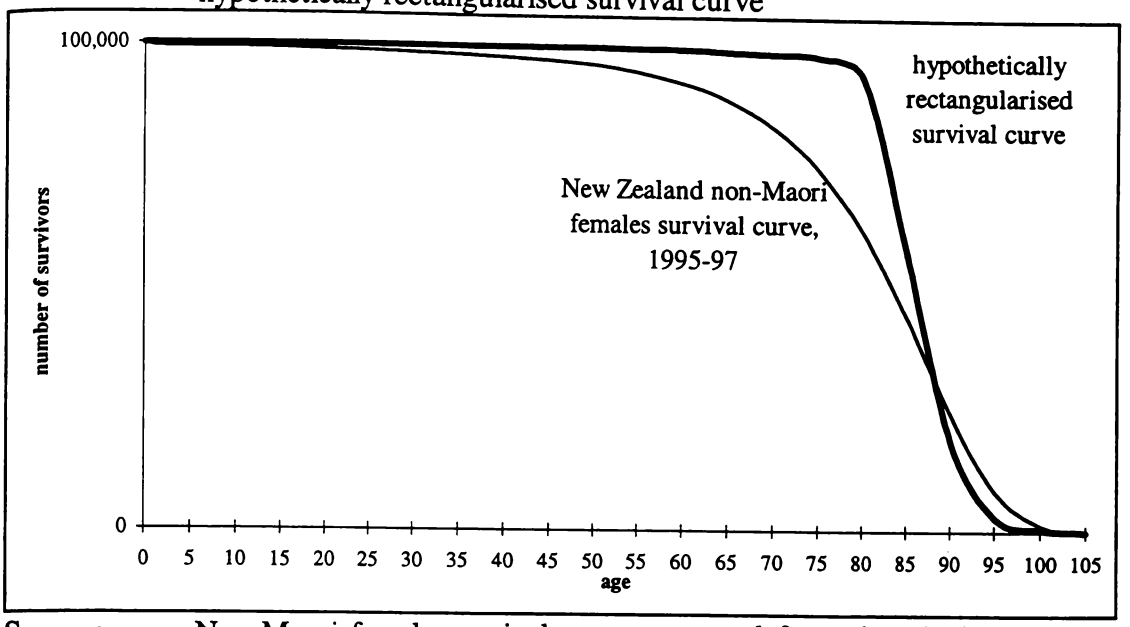
ages older than 93 years in 1995-97 already accounted for over 10 percent of all life table deaths. It appears that Fries' specifications of a compressed mortality pattern at the upper age range is unrealistic, as reflected by the considerable space between the two curves at ages 93 years and above. For 95 percent of all "natural deaths" to occur before the age of 93 years, a deterioration in age specific mortality rates between ages 85 and 93 years is required to bring forward more than half of deaths currently observed at ages older than 93 years.

While Fries' curve seems unrealistic under present conditions this does not exclude the possibility that in future it could more accurately reflect actual experience, even for ages 93 years and over. The reason for this argument rests with the fact that the observed distribution comes from a synthetic life table. It is entirely possible that the oldest cohorts in the 1995-97 tables could have atypically high levels of longevity as hardy survivors of higher rates of mortality at much younger ages early the twentieth century. This alternative scenario will be investigated here in later chapters.

To operationalise the abstract concept of compression of mortality, Fries proposed the idea of "rectangularisation of the survival curve" (Fries, 1980; 1983; 1988), capitalising on general familiarity with the survival curve of the life table. The latter notion is essentially a statistical manifestation of the former. It is based on the idea that as deaths continue to concentrate in a narrow range at older ages, the survival curve will stay flat before reaching this range, but because of senescent frailty will drop nearly vertically within this age range for "natural death". The age at death distributions in Figure 4.1 are re-organised as survival curves in Figure 4.2 to help explaining this latter concept.

The hypothetical survival curve stays horizontal before age 81 years, but between ages 81 and 89 years, when the mortality compression is taking place, the survival curve drops sharply. The horizontal property of the hypothetical curve at younger ages and the vertical tendency at older age combine to produce a visual "rectangular" effect. The hypothetical survival curve drops below the non-Maori female curve at around age 87 years, indicating that the hypothetically compressed mortality pattern as specified by Fries has under-estimated survivorship at ages 85 years and above, as noted earlier.

Figure 4.2 New Zealand non-Maori females life table survival curve in 1995-97 and hypothetically rectangularised survival curve



Sources: Non-Maori female survival curve extracted from the 1995-97 complete life table constructed by Statistics New Zealand
The hypothetical survival curve, generated by the author based on the curve proposed by Fries (1980)

The building block of Fries' compression of mortality hypothesis is that the biological limit to the human life span is fixed at about age 115 years, but realistically reduced to 85 years due to non-predetermined factors. Life expectancy at birth in many developed countries is rapidly approaching this limit of 85 years of age. Relying on this presupposed fixed biological limit to life expectancy at birth, Fries foresaw two crucial developments in future mortality patterns: i) death rates will remain low before reaching an age just before age 85 years to extend the horizontal section of the survival curve; and ii) an over-whelming majority of deaths will occur over a short span of years to complete the vertical component of a rectangularised survival curve. These thus raise two key issues:

1. the existence and the effect of a fixed upper limit to life expectancy at birth; and
2. whether mortality declines at the very old ages will decelerate, or even reverse, so to confine the vast majority of deaths within a narrow age range.

The rest of this chapter and the subsequent ones in this thesis relating to the empirical analyses of mortality compression in the New Zealand non-Maori population are less concerned with the numerical expressions of mortality compression laid down by Fries (1980; 1984; 1988) and their discrepancies from reality. Rather, the focus is on the above

two issues, that is, the operating mechanisms of mortality compression and their implications. These two issues are examined separately in the next two sections in exploring arguments both for and against the Fries hypothesis.

4.1.1 Definitional and Methodological Issues

In presenting his view, Fries (1980) failed to give a precise definition or an appropriate operational basis for empirical evaluations of either mortality compression or morbidity compression. The supposed end result of mortality compression, a mix of premature deaths and natural deaths as noted earlier, was only implied by Fries as what could be expected under "ideal societal conditions" (see Figure 4.1). Factors constituting "ideal societal conditions" were not, however, specified. The concepts of premature death and natural death are difficult to translate into operational definitions. According to Fries, these concepts "are complementary rather than antagonistic, and any dividing line must be an arbitrary one" (1983:400).

Rectangularisation of the survival curve, the alternative statistical expression of the compression effect, is no easier to measure. Though intuitively appealing, the concept can have a number of operational definitions (Anson, 1992). Much of the controversy arises because of disagreements over the relevant age range of the curve to be measured. For example, the cut-off point at the upper open-end of the curve can, at best, only be arbitrarily defined or assumed. As demonstrated by Fries himself (1984), results are often highly sensitive to the age cut-off points at both ends (see also Myers and Manton, 1984a). It is unclear whether the conventional standard deviation technique is an appropriate one to apply to analyses of the spread of survivorship at older ages because behaviour of the tail of the distribution is erratic, yet critical. Study the exact nature of the tail end of this distribution is always problematic since data are sparse and not necessarily reliable, as they are, among other problems, subject to both numerator and denominator age reporting errors.

In fact, the appropriateness of the survival curve as a tool for analysing mortality has been questioned by Carey et al (1992). These authors pointed out that survival curves

are used not because they are the best curve for studying mortality patterns, but because they are smooth and decrease systematically, even for small populations. Yet these curves may give mis-leading impressions, as mortality differences between age groups may not be systematically correlated with age. That is, sequential age groups may not systematically have lower levels of mortality, and there may well be fluctuations in age specific death rates (e.g. male youth aged between 15 and 24 years often have higher rates than do the age groups either side of these ones). Thus age specific demographic patterns may often be closer to reality than those which incorporate some sort of statistical cumulative effect (Rogers, 1992; Vaupel and Yashin, 1985).

Methods other than those based in the life table have been suggested and experimented, ranging from the more sophisticated involving "numerator analysis" (Myers and Manton, 1984a), which focuses exclusively on death statistics, to rather crude studies based on the empirical population and death statistics (Go et al, 1995). These moves away from life table methodologies have, however, been criticised, for the simple reason that changes in the age structure of the population would affect the results and undermine their comparability (Nusselder and Mackenbach, 1996).

In Chapter Eleven, the definitional and methodological issues associated with empirical testing for mortality compression in the non-Maori population are re-addressed and working solutions proposed.

4.2 Support for the Compression of Mortality Hypothesis

As is typical of such conjectures, Fries drew on theoretical deductions on less tangible events such as biologically pre-determined natural life span. The empirical evidence in support of a biological limit to life, so he argued, comes from the recent declines in old age mortality and the absence of an observed significant extension to the tail of the distribution. As will be discussed later in the chapter, this is highly contentious.

4.2.1 Biological Limits to Length of Life

Fries (1988) justified the important cornerstone of his hypothesis on several fronts: a genetically pre-determined human life span to which we are rapidly approaching. Empirically, there is no scientific documentations on the existence of any population with exceptional longevity. The range of highest age lived of 110s is confined to a selected few and has remain largely unchanged. The greatest authenticated age of 122 years was recorded in France (cited in Paccaud et al, 1998). The tail of survival curves generally confirm to those observed for the right-hand of normal distributions, tapering off at the upper-end. Olshansky et al (1993) further supported this empirical deduction of Fries with arguments based on evolutionary theories of senescence, despite disagreements about some sort of pre-determined genetic clock (see Chapter Three). These authors asserted that humans are generally not capable of living much beyond the age of 110 years regardless of the observed declines in death rates form major fatal diseases.

Over the last century life expectancy has made significant gains, but life span has remained almost constant (Carnes et al, 1996; Fries, 1988; 1992; Olshansky et al., 1993). Notwithstanding significant increases in life expectancy, so they argue, there have been few detectable changes in the number of centenarians, a point argued otherwise by others (Kannisto, 1988; Kannisto et al, 1994; Thatcher, 1981; 1992; Vaupel, 1998).

Fries (1984) cited the reducing gender differentials in life expectancy in Japan, dropping to less than five years in early 1980s. Japanese life expectancy of both genders are the highest in the world. Moreover, if life expectancy increases are constrained by an upper boundary then, by logic, populations with the highest life expectancy should exhibit the greatest constraints (Coale, 1996). Following this argument, increases in the Japanese female life expectancy will slow relative to changes for males as women's levels approach this boundary, and this will allow their male counterparts to catch up.

Theoretical arguments across a number of disciplines in support of the existence of a biological limit to life expectancy are drawn upon by Fries (1988). The actuarial law of mortality, represented by the Gompertz function, states that the rate of mortality doubles every eight years beginning in early adulthood. Indeed, the characteristic shape, but not

the magnitude, of the human mortality curve stayed essentially unchanged throughout the twentieth century despite substantial improvements in survivorship at all ages (Olshansky et al, 1993, see also Olshansky and Carnes, 1997). An exponentially increasing mortality function theoretically converges to a boundary.

On the biological front, Hayflick demonstrated a finite number of cell doubling in the life span of a species (cited in Fries, 1988). Some believe that the length of life is ultimately limited by the genetic constitution of individuals (Chapman et al, 1986). This limit, though not a function of a pre-determined genetic programme, can also be derived from the evolutionary theories on senescence (Olshansky et al, 1993).

Quantitative models of senescence derived from biological and physiological data suggest the existence of a hazard function (a function which describes the instantaneous rate of mortality, also known as the actuarial force of mortality) which is associated with the ageing process (Fries and Crapo, 1981). The force of the hazard function is relatively minimal before the age of 70 years, but increases rapidly thereafter and dominates the disease-related hazard function over the age of 100 years (Fries, 1984). It is further argued that this type of hazard function is not included in mortality models such as the competing risk one, and therefore that survival is overestimated at advanced ages (Fries, 1983; 1989).

At the level of the organism, Fries (1980; 1988) contended that, in young adults, the functional capacity of human organs is four to 10 times that required to sustain life. This "organ reserve" declines linearly over time beginning at about the age of 30 years. He holds that an age-associated progressive loss of organ reserve, to approximately 20 percent of basal level required for the maintenance of basic life processes, can lead to natural death, even without disease. Clinical observations cited by Fries suggest as many as 25 percent of deaths occurring in individuals with minimal organ reserves, are essentially from natural death (the simultaneous multiple failure of body systems at the end of the life span).

4.2.2 Compression Towards Biological Limits of Length of Life

A pre-condition of mortality compression is a stationarity in mortality rates at very old ages. In general, the momentum of mortality reductions has been proportionately greatest at younger age groups and has declined progressively with age (Grundy, 1984; Paccaud et al, 1998; Pool, 1994). The survival curves for some populations with low levels of mortality are showing an increasingly acute decline at older ages (Riley, 1990a). This signifies a shift towards stationarity of mortality at very old ages, while the force of mortality is progressively concentrated at these age groups.

Until the 1970s, significant ground was made in improvements in life expectancy at birth, but life expectancy at middle and older ages had changed a little (Fries, 1996). This reflects progress in the elimination of "premature death" as defined by Fries. It is a well known statistical fact that a life saved in infancy contributes more substantially to overall life expectancy than a life saved at, say, age 60 years.

Sequential survival curves over the last century analysed by Fries demonstrate a progressive rectangularisation effects (1980; 1984; 1989). The progressive elimination of the risk of premature deaths produces a horizontal survival curve before reaching the age when the risk of natural death becomes perceptible. If it is governed by the biologically pre-determined natural life span, then once survivors survive the risk of premature death they face the inevitability of natural death.

4.3 Alternative Theories to the Compression of Mortality Hypothesis

Opponents to the hypothesis of mortality compression argue that recent mortality patterns in developed countries are largely contradictory to what is predicted by the hypothesis. For example, at ages 65 years and above significant improvements in survivorship have been recorded in every age group (Kannisto et al, 1994; Myers and Manton, 1984b; Schneider and Brody, 1983; Thatcher, 1992; Vaupel, 1998). If there is well-defined limit to the increases in life expectancy, critics argue, it should cause rapid acceleration in mortality rates at the very old ages in order to produce a near vertical

drop of the survival curve (Curtsinger et al, 1992). Instead, the highest age group, 85 years and over in most studies has enjoyed a disproportionately large share of mortality declines.

Based on data from the United States from 1962 to 1984, Rothenberg et al. (1991) used various statistical techniques to evaluate the compression hypothesis. They came to the conclusion that their findings support the notion of compression only in its simplest sense, but the more specific notion of compression, wherein deaths clustered around some fixed limit or up against a fixed boundary, is not supported.

4.3.1 Diverging Views on the Biological Limits to Length of Life

Generally, two schools of thought on the biological limit to life expectancy can be identified. Some believe in its existence, but finds no agreement among themselves as to the exact position of this limit. There have been numerous attempts to estimate this upper boundary (Brody, 1985; Coale, 1996; Lohman et al, 1992; Manton et al, 1991; Olshansky et al, 1990; 1993; Paccaud et al, 1998; Rothenberg et al, 1991; Schatzkin, 1980), but as there are no empirical data on which to base this search, so far no firm conclusions can be drawn. Predictions generally range from 80 to 120 years. Similarly, how far future extension can be achieved by slowing the rate of senescence is the subject of much speculation.

The second group of critics of compression hypothesis argue against a biological upper limit to life. The Gompertz function is found to be an inadequate tool by which to describe mortality patterns at advanced ages. To the contrary, several studies have suggested a slowing in the rate of mortality increases after age 85 years (Gavrilov and Gavrilova, 1991:129; Kannisto, 1988; Manton, 1992; Thatcher, 1981; Vaupel, 1998). The rate of mortality does not necessarily follow the same exponential path during the last decades of life (Kannisto, 1988), but tends to stabilise into a pattern such that the chance of survival from one year to the next is roughly half (Thatcher, 1981; 1992). This hypothesis would suggest that rather than being governed by a biological clock, life at very old ages is determined by the law of probability.

Whichever way the debate on the biological limits to the human life span unfold, it is hard to see the relevance of some such limits to issues of immediate concern. Manton and his colleagues (Manton et al, 1991; Myers and Manton, 1984a) acknowledged that so long as we do not have data on a mortality configuration allowing us to compute the portion of deaths to which the intrinsic biological limit relates, we can hardly prove conclusively the presence of mortality compression. The differences between the concepts of natural and premature deaths are blurred. Until a separation of "disease" and "senescence" components of mortality is made, assuming they are independent of each other, the precise biological limits to life expectancy will remain elusive.

4.3.2 Mortality Shifts at Very Old Ages

The opponents of the compression hypothesis maintain that mortality is expanding at the older age groups (Rothenberg et al, 1991). For rectangularisation to materialise, mortality rates must stay high and stabilise in age groups immediately below and above the parameter of age 85 years as specified by Fries in order to produce a near vertical drop in survivorship (see Figure 4.2). The observation that the greatest proportional mortality declines have occurred among the young, an empirical observation which is still debated by some, does not mean that there are no changes in mortality rates at older ages. Far from that, data have revealed significant changes even at most advanced ages (Kannisto et al, 1994). For example, British data has shown that for the cohort born in 1880, within the space of 30 years the chance of becoming a centenarian has increased fourfold (Thatcher, 1992). If we focus specifically on and around the age of 85 years, then currently the rate of mortality reductions at these ages seem to show no sign of deceleration. Continuous substantial reductions in mortality rates at these and even higher ages cause the survival curve to "derectangularise".

Some try to test the empirical validity of mortality compression through the inverse correlation between variance in age at death and increases in life expectancy (Myers and Manton, 1984a; and Olshansky et al, 1990). It is argued that as a maximum life expectancy is approached and mortality is becoming progressively compressed, the variations in the age at death distribution, measured by the standard deviation, should

decrease until they reflect only the heterogeneity in the endowment for longevity. This standard deviation argument, however, carries some flaws in its statistical base[1], and is disputed on theoretical grounds by Fries (1984).

Those who believe in the existence of a biological limit to life expectancy hold that the shift in the distribution of death cannot go on indefinitely, and that the maximum natural life span will act as the ultimate barrier to the current expansion of survival curve (Olshansky and Ault, 1986; Olshansky et al, 1993; Rothenberg et al, 1991). Under these conditions, some argue, it is inevitable that mortality compression will eventually take place. Others, citing the notion of heterogeneity among the older population, dispute the above argument by the inherent assumption of homogeneity in a population (Manton et al, 1991; 1997b; Nusselder and Mackenbach, 1996). Individual differences in the biological endowment for longevity will prevent the survival curve from rectangularising. For example, heterogeneity among the older population may be able to explain the levelling off of mortality rates at advanced ages as noted above. Darwinian-type selection, which is less subject to control by intervening processes at higher ages, is more effective in ensuring the survival of the fittest.

4.4 Conclusion

Simply stated, the compression of mortality is a redistribution of age at death, with the risk being shifted from the young to the old, but becoming most concentrated within a narrow age frame. Efforts on empirical testing of this hypothesis, however, is hampered by the imprecision in the technical details and its measurements. Depending on the methodology adopted, findings can vary considerably. The root to many controversies is an old problem: how much emphasis should be given to the mortality rates at the very old ages? Some argue that few people survive beyond age 100 years which makes their number under consideration demographically insignificant. Concentrating on selected very old age groups tends to inflate the overall impacts. While others hold that stabilised

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[1] Standard deviation is essentially a measure of the spread of the distribution away from the mean, and its value is heavily influenced by the position of the mean. Given that mortality at the lower age range has been reduced to a minimum, the higher the mean age at death, the greater the weight deaths at relatively younger ages attract in the standard deviation calculations.

mortality declines at advanced ages is the first milestone in the march towards mortality compression. Persistent mortality declines at all ages will lead to the expansion of the entire survival curve in its characteristic shape, including the extension of the tail. As will be shown in Chapter Six, the movement towards compression is not uni-directional and restricted to older ages. Instead, at the start of the epidemiologic transition the force of mortality would be left-skewed and would produce an inverse-retangularisation effect of the survival curve.

The concept of mortality compression has raised intense debate in the international research community. Many express scepticism on the theoretical basis of the hypothesis. Questions on the existence of a fixed biological limit to life expectancy are central to the compression debate. Convincing proof in favour of either side of the argument remains elusive, yet the answer to this question has the most significant bearing in determining the eventual epidemiologic pathway. Despite some heavy criticisms it has received, the conceptual and methodological issues raised by the notion of mortality compression cannot be easily dismissed. Though it is debatable whether the limit of life expectancy is a biological one or whether pursuing this question is very practical (Lohman et al, 1992), its policy implications in areas such as defining preventive health measures and evaluating programme priorities are far-reaching. Many economic and health issues could be considerably affected by its nature and its position. The framework derived from the compression of mortality hypothesis carries profound policy implications, directly affecting forecasts and the planning of social and medical programmes which must be tied to the size and health status of the older population.

It should be noted that much of the attention is currently centred on trends from cross-sectional views of the survival curve. These period trends are "the product of interactions of multiple dynamic period and cohort factors" (Manton et al, 1997b:135), yet in most of the deliberations cohort effects tend to be overlooked.

It can be argued that period trends in mortality, especially those involving the older members of the population, are potentially confounded by historical events and conditions that different birth cohorts experienced at different points in their lives (Manton and Vaupel, 1995; Manton et al, 1997b; Reynolds et al, 1998). Events such as

the two World Wars and the Great Depression; and conditions such as differential exposure to the influenza epidemic in the late 1910's and medical advances in recent decades may have had lasting effects on the health status of the cohorts. The health experience of each birth cohort viewed in a cross-sectional snapshot is filtered through its unique pattern of prior exposure to the risk of mortality and morbidity. Furthermore, differences in their patterns of health-related behaviour including smoking and dietary patterns are arguably reflections of different cohort experiences in education, in the labour force, and in socio-economic circumstances (Crimmins et al, 1997a; Leigh and Fries, 1994; Reynolds et al, 1998). As explained in Chapter Three, cohort differentials in exposure to risk and their accumulated effects are key determining factors for population heterogeneity. Synthetic measures which collapse together cohorts at different stage in these mortality changes tend to make assumptions of population homogeneity.

Since cross-sectional data extracted from synthetic life tables encompass mortality patterns of multiple cohorts and are essentially hypothetical measures of mortality, a cohort approach is necessary to provide a different picture of mortality changes. For example, as mortality has been decreasing over most of the last century, cohort survival curves, for which we have complete data, are likely to show an upper right-hand extensive stretching even further than that shown by the cross-sectional curves. A cohort based approach to the analysis of population health patterns and trends is further developed for the present study in Chapter Seven. The issue of possible mortality compression by non-Maori birth cohorts is tested empirically in Chapter Twelve.

Withstanding unresolved issues on various aspects of the hypothesis, Fries' compression of mortality still provides perhaps the most easily modelled framework for investigations. The present study uses Fries' schema and elaborate it as a useful starting point, a point which the present author elaborates but often also diverges from (see Chapter Six). In the next chapter (Five), however, the focus is turned to the interconnected topic of morbidity, and its trends in relation to mortality changes. Leading theories on recent morbidity changes will be investigated.

CHAPTER FIVE
CHANGING PATTERNS OF MORBIDITY IN RELATION TO MORTALITY

Mortality is the extreme end point of ill-health. Demographic trends in mortality, the subject matter of the last chapter (Four), not only reflect population health status, but, more importantly, differences in the tempo of mortality changes across population sub-groups and over time determine the size and the demographic composition of populations which also have enormous implications for population health. Within the population parameters circumscribed by mortality, variations in population morbidity patterns are the determinants of the changing level of mortality. The analysis of mortality trends in Chapter Four paves the way for the discussion in this chapter on the changing patterns of morbidity in relation to mortality changes. In this chapter, three leading theories on the evolution of morbidity in relation to mortality are introduced, and are used as an investigative framework. The focus of this chapter is on analysing and contrasting elements of these leading theories. In doing so, by implication, it may suggest that these theories have competing paths. This is not necessarily a true reflection of the situation, as demonstrated in the next chapter (Six) which draws all three theories on morbidity changes into an integrated framework. Although most aspects of these theoretical conjectures cannot be empirically tested in New Zealand at present because of data unavailability, insights gained from a theoretical analysis enrich understandings and improve the modelling of future population health dynamics in Part Six of this thesis.

The significance of morbidity for the contemporary field of population health research has grown out of an attempt to understand the rapid increases in life expectancy. The continuous mortality declines over the last century, particularly declines at late adult ages over the last two decades, have pushed morbidity high up on a research agendas. For a greater part of the twentieth century, as a results of the shift in the disease profile toward degenerative diseases and the accompanying shift in the force of mortality towards older ages, mortality and morbidity have generally moved on divergent paths, as was reviewed in Chapter Two. Starting in the late 1970s in most developed countries the majority of mortality risks had already become concentrated at older ages and "compressed" around a few chronic degenerative diseases (Pool, 1994). This set for a new stage in the morbidity transition.

Studies on the postponement of mortality pose important questions which become more apparent when assessed from a morbidity framework. Additional years of life gained certainly represent a triumph for civilisation, but the effects of this gain on the health of populations have been perceived as a mixed blessing. Views regarding the present and the future morbidity trends vary greatly. Some pessimists hold that the improved longevity arising from arresting the progression of chronic diseases is accompanied by a pandemic of disease and disability (Gruenberg, 1977; Kramer, 1980), or may even mean an impending disaster for society (Olshansky et al, 1993). Optimists, on the other hand, maintain that by delaying the onset of morbid conditions a greater proportion of life is likely be spent in good health (Fries, 1980; 1988; 1996). Still there are others who have taken an intermediate position, arguing for a trade-off between the prevalence and the severity of morbidity (Manton, 1982). These diverging views have quickly developed into a three-way debate with the protagonist theories, termed the "pandemic, compression, or dynamic equilibrium of morbidity" approaches, following the chronological order of their formulation. Each of the three leading theories on the evolution of population health is reviewed in this chapter.

The implications of future morbidity movements in relation to mortality trends have far-reaching public health policy consequences. Several international research forums on this subject were held and their proceedings were published (see *Gerontologica Perspecta*, 1987; *Journal of Aging and Health*, 1991; *Milbank Memorial Fund Quarterly*, 1985). Efforts in this area of research are, however, often hindered by even more obstacles than are studies on mortality. Documentation on the qualitative aspects of life extension and life satisfaction are inconclusive. Quality of life is often measured by the presence or the absence of morbid conditions. The multi-dimensionality of morbidity, however, makes it notoriously difficult to be defined and measured. Morbidity statistics generally include a vast array of health related states, ranging from a lengthy pre-symptomatic stage for many chronic diseases, when patients often experience no apparent discomfort, to a final stage just prior to death. In addition, the definitions of diseases and morbid conditions have changed over time and between cultures, varying according to prevailing medical and social settings.

5.1 The Pandemic of Morbidity

In a influential article published in 1977, Gruenberg first raised the alarm about the "pandemic effect" of morbidity. He argued that the extension of life expectancy, due to advances in medical care in treatments of lethal sequels of formerly fatal diseases, has led to the rising prevalence of certain chronic conditions. Mortality declines, he argued, are induced by declines in the fatality rates of chronic diseases rather than declines in the incidence or the rate of progression of these diseases. That is, reductions in case fatality rates have resulted in increases in the prevalence of some chronic conditions (Chapman et al, 1986). Life extension is therefore equated with the extension of time spent in states with disease and/or disability, and often may involve severe conditions. Gruenberg concludes that "our life-saving technology of the past four decades has outstripped our health-preserving technology and that the net effect has been to worsen the people's health" (1977:22). His view is built upon by Kramer (1980), drawing on data mainly from the field of mental health. Minor reductions through preventive measures in the incidence of some chronic conditions have been more than offset by the increased duration of time spent suffering from a wide range of conditions whose fatal complications have been successfully postponed.

Olshansky and colleagues (Olshansky, 1985; Olshansky et al, 1993) further supported this view with arguments from evolutionary biology (see Chapter Three). They maintained that protracted chronic morbidity is a necessary consequence and a corollary to the evolutionary theories of senescence. As a higher proportion of the population reaches advanced ages, morbidity will increase if the frailty distribution shifts from "disease-dependent causes" to the inherent "age-dependent physiological decrements" (Olshansky et al, 1990), though differences between these two states are arbitrary at present. Senescent diseases, including new diseases not previously experienced, stand at a much greater chance of manifesting themselves among an older and more heterogeneous population.

Hospital data from the Minnesota Heart Survey from 1970 to 1985 reveal a considerable decline in coronary heart disease mortality rate, particularly at older ages where the prevalence of coronary heart disease is high. However, hospital discharges due to acute coronary heart disease have increased at an alarming rate over the same period (Burke et

al, 1989). Stout and Crawford (1988) also detected an increase in the length of hospital stay among older patients. Though increases in the active portion of life expectancy and postponement of the onset of terminal dependency are also observed, increases in the absolute length of stay and, more importantly, increases in the proportion of life spent in long-term hospital care are more significant. Working on data on the health status of elderly persons in Manitoba, Canada, Roos et al (1993) concluded that while elderly individuals were living longer, but were in poorer health status. "Longer life but worsening health" is the well-known theme of Verbrugge's (1984) findings of a detailed investigation of population health in the United States on a broader array of health indicators and over a longer time frame.

5.1.1 Determinants and Mechanisms of the Pandemic Effect

Secondary prevention measures, characterised by the early detection of diseases and subsequent interventions to slow its progress, have been the major emphases in medical care and personal health care domain. Improved survival is therefore identified as one of the major determinants of the pandemic effect; among others, changing cohort size, the rising awareness and expectations of the population, improved diagnostic techniques and early accommodation for the disease, greater availability of health care services at lower costs, are the possible explanations.

Greater readiness to resort to elective and acute therapeutic procedures and increased admissions for procedures or diagnostic evaluations are believed to be some of the contributing factors to the increased hospital discharge statistics. Hospital treatments and managements of the frail older patients aiming at improving the quality of their remaining life invariably improve also their chance of survival (Stout and Crawford, 1988). Medical advances are more successful in restricting the progress of chronic diseases than eliminating chronic disabling conditions, and therefore contribute to the eventual terminal outcome and the prolonged duration of these conditions.

Kramer (1980) formulated two major mechanisms responsible for this situation. First, population ageing significantly increases the relative size of the older population who are

generally at high risk of developing chronic conditions. This mechanism is chiefly mortality driven, and fuels the crude prevalence rate of chronic conditions. Second, the average duration of chronic diseases has increased due to the "failures of success" by medical advances (Gruenberg, 1977). That is, the cures postpone death, but either the condition continues or undergoes recrudescence. The net result is a longer cumulative duration in sickness. This situation, therefore, is responsible for increases in the age specific prevalence rates of diseases, which are at the heart of the problem. It is doubtful if progress in the past has changed the basic parameters of senescence, and further progress could extend the scope for untreatable disability now common among the very old, or even some less common but more debilitating ones (Olshansky, et al, 1993).

5.1.2 Implications of the Pandemic of Morbidity

It is believed that advances in medical technology combined with more aggressive therapeutic interventions have been successful in prolonging the risk of death. Bio-medical advances have also extended the range of "salvage capacity", and produce further problems of morbidity among the aged (Olshansky et al, 1990). This is reflected by Pool in his assessment: "once sick, however, they are less likely to die" (1994:37).

The alarms about the "failures of success" by medical advances and the rising pandemic of chronic diseases has raised the general awareness of this issue, and has injected a sense of urgency into the search for preventable causes of the chronic diseases which are increasing in prevalence. To arrest the rising prevalence of chronic conditions and to produce a parallel effect of improvements in survival and in quality of life, directions in current health research and policy need to turn away from a curative emphasis and more towards the prevention of chronic diseases. Gruenberg (1977) argued that resources are disproportionately invested in research on treatment, but are inadequate in the area of disease prevention, producing a high prevalence of protracted periods of morbidity.

5.2 The Compression of Morbidity

A completely opposite view to the pandemic theory is raised by Fries (1980; 1983; 1988; 1992; 1996), who envisages a state where the onset of permanent morbidity is significantly delayed to later ages, and thereby prolongs health. Since the life span is genetically fixed at a theoretical maximum, the validity and the exact position of which was examined in the last chapter (Four), and in the absence of any further extension of life expectancy, morbidity is compressed into the last few years of life by postponing the age of first chronic infirmity. A more comprehensive definition of the theory is later provided by Fries (1983; 1988; 1989), which states that the average age of the onset of morbidity, usually a significant permanent infirmity, may increase at a faster pace than life expectancy. Morbidity will be compressed in relative terms if the proportion of life spent infirm in relation to the life expectancy is shortened. An extension of this relative compression is the absolute compression where the period of morbidity is shortened in absolute magnitude if the age specific morbidity rates decrease more rapidly than do the age specific mortality rates.

A more formal definition, provided in Fries' 1988 update, lays down the general formulation of the theory.

> *[F]or the average person, (a) if morbidity may be defined as that period from the onset of the first irreversible chronic disease or ageing marker until death, (b) if the date of occurrence of that marker can be postponed until later in life, and (c) if the rate of such postponement can be greater than the rate of increase in adult life expectancy, then (d) morbidity for the average person can be compressed into a shorter period of time.*
>
> (Fries, 1988:408)

The theory of compression of morbidity is built essentially on two premises. First, the length of average life is limited by a biological maximum. This average maximum, projected by Fries as age 85 years, is acting as a theoretical limit to the increases in life expectancy at birth. Second, risk factors of chronic diseases can be modified so that their

onset can be postponed to later ages. Combining these two factors, the average period of infirmity will be reduced.

The first premise and its necessary corollaries of the rectangularisation of the survival curve, the validity of both of which is still in question, was the focus of last chapter (Four). Fries has made mortality compression a pre-requiste to morbidity compression. If the period of morbidity is measured from its onset to its end point, a rectangularised survival curve will act as an boundary to circumscribe the upper end. Others have argued, however, that the relevance of mortality compression to morbidity compression is not necessarily the lead-follow relationship portrayed by Fries (Myers and Manton, 1984b; Robine et al, 1997). This point is re-addressed in greater detail in the next chapter (Six).

The second underlying premise rests heavily on the ability of the health sciences to delay the onset of morbidity. Risk factor modification, such as changes in diet, exercise and daily routines will help to better manage the senescence process (Fries, 1992; 1996, Leigh and Fries, 1994). It is hypothesised that, as the delay in onset occurs mainly for what are normally fatal diseases, it will therefore lower both the incidence of mortality and morbidity prevalence rates (Fries, 1983; 1988; 1989).

5.2.1 Empirical Evidence in Support of Morbidity Compression

Over the last few decades proportionately more people than ever before have survived to advanced ages and are thus should be at greater risk of chronic conditions. Yet morbidity due to some chronic diseases, such as atherosclerosis and lung cancer, have shown downward trends (Fries, 1988; 1992). This reduction has principally come from incidence rather than from survival after the first clinical event. It is suggested, on the basis of medical evidence, that the onset of chronic infirmity has shown signs of postponement to even later ages (Lubitz and Prihoda, 1984; Roos et al, 1987). For some conditions, morbidity is thus being compressed between an increasingly late onset and a relatively fixed life expectancy. Drawing on data from clinical literature and randomised-control trials, Fries (1988; 1989) maintained that higher reductions in some cause specific morbidity than in mortality have been achieved.

From this empirical evidence he deduced that it is easier to reduce morbidity than to reduce mortality in some major causes, and hence that substantial reductions in morbidity due to many other causes are theoretically possible.

Indeed, cross-sectional surveys in Britain indicate that today's older population appears fitter than were previous cohorts. Moreover, the prevalence rates at different levels of disability among the older community showed a general decline from 1981 to 1988 (Jagger et al, 1991). Similar cohort effects have also been observed in the United States (Arber, 1996; Manton et al, 1997a; see also Palmore, 1976; 1986).

Others stressed the notion of multiplicity in the road to morbidity compression (Branch et al, 1984; Rogers et al, 1990; Suzman et al, 1992). According to them, a preoccupation with the onset of chronic morbidity and dependence has diverted attention from both the possibility of, and the effects of, recovery from morbid or dependent states.

Morbidity compression is also reflected in the cost of health care. The commonly held belief that advanced age dramatically increases the consumption of all types of health care is not supported by many studies into the health care utilisation of the older population (Cohen et al, 1997; Henderson et al, 1990; House et al, 1990; Roos et al, 1987; Scitovsky, 1984; 1988; van Weel and Michels, 1997). Analysis of health patterns of the older population based on hospital discharge data, which by definition focus on usage rather than non-usage (Payne et al, 1993), mask the fact that the majority of the older population, even the very old, are healthy and infrequent users of acute and chronic hospitals (Cohen et al, 1997; Roos et al, 1984). Studies on the costs of caring for terminal patients indicate that age-specific increases in medical costs are largely an artefact of the high costs incurred in the last years of life. Significant portions of health care utilisation are associated with dying rather than ageing (House et al, 1990; van Weel and Michels, 1997). According to these studies, decedents in the last year of life greatly exceeded survivors for all measures of hospital use. Once the impact of mortality is removed, the apparent rapid increase in age-specific health care expenses is greatly reduced. Lubitz and Prihoda (1984) also observed a decline by age among decedents in their medical cost. An explanation put forward is that very old, frail individuals are less able to benefit from large amounts of costly intervention (Cohen et al, 1997; Scitovsky, 1988).

The growing concentration of medical costs at a time close to dying (van Weel and Michels, 1997) is an indication of compression of health care needs in an individual's life. It is estimated that 18 percent of lifetime medical costs are incurred in the last year of life (Fries, 1989), and an even further concentration in the last one or two months of life is suggested by some studies (Cohen et al, 1997:4; Lubitz and Prihoda, 1984). As an indirect implication of this economic compression of morbidity, deferral of death to later ages also postpones the significant costs associated with dying. Medical expenses exhibits a higher correlation with time to death than time since birth. Methodologically, chronological age alone may no longer be adequate in forecasting medical costs (Cohen et al, 1997); instead time to death may better reflect an individual's medical need.

5.2.2 The Plasticity of Senescence

Fries (1989; 1992; 1996) contends that only a minute fraction of the population is at the forefront of the battle against senescence, while most of us are treading along below our personal best. Individuals not performing at a maximum level might readily improve so that their physical and psychological risk factors would be modified. That is, pandemic of chronic conditions is essentially a result of individuals performing below their own maximum personal physical and psychological levels, and thus could be reversed.

Aided by medical advances in postponing chronic diseases, morbidity compression is achieved through the gradual elimination of premature chronic conditions and modified senescence processes. The modifiability, or the plasticity, of many senescent phenomena holds the key to morbidity compression. Studies indicate that many physical, mental, and social capacities can be preserved and are modifiable by the individual at any age. For example, dietary restrictions seems to help preserve somatic maintenance for a longer time (Olshansky et al, 1993), though it may not be practical to expect the whole population to adopt a calorically restricted diet. The outlook of the compression theorists are indeed optimistic, assuming a rapid success in the development of prevention techniques and their subsequent widespread applications, and an equally swift change in individual health practices and life-stylies.

The optimistic outlook of Fries if further heightened by the notion of "natural death" (Fries, 1980) introduced in Chapter Four: the simultaneous multiple failure of body systems at the end of the life span. Fries argues that "organ reserve" declines with age, and so the body loses the ability to restore homoeostasis. A logical outcome is natural death, even without disease. Fries implicitly divides causes of morbidity and mortality into two broad groups: those resulting in premature death and those resulting in natural death. The latter can be considered as accelerated loss of organ reserve, and the only effective strategy in dealing with this group of diseases is postponement rather than cure. Accordingly, policy planners might direct their attention at the formulation of "successful ageing" programmes (Fries, 1989; 1996); and at measures aiming at eliminating or reducing the risks of non-fatal illness, which would, however, have relatively less impact on mortality.

The notion of "natural death" is, however, heavily attacked by Schneider and Brody (1983), who asserted that most organs are not compromised sufficiently for death to result in the absence of disease. Beyond this, they argue, fruitful breakthroughs in curative and preventive interventions, as main ingredients of the recipe for "successful ageing" programmes, are at present beyond the capacity of the health sciences and society.

5.2.3 Implications of the Compression of Morbidity

Population homogeneity in the endowment for both longevity and health is the underlying premise of Fries' compression argument. It follows that the process of population ageing, independent of any specific disease, will constitute the core of health burden of the society (Fries, 1984). A direct implication of this paradigm is that the era of high prevalence of chronic disease will slowly give way to a new era when diseases directly related to the senescence process, some still be unknown, will constitute the major health problems (Olshansky et al, 1993).

The compression of morbidity theory is delivered by Fries through a series of predictions. Most of these predictions currently cannot be fully tested since life expectancy at birth in most low mortality populations has not yet reached the predefined theoretical maximum of 85 years. Furthermore, data on the incidence of many chronic diseases is inadequate.

The compression effect has been observed in some sectors, but whether it is an achievable phenomenon on a much wider scale is debatable.

It is generally agreed that for morbidity to move in parallel with mortality, preventive measures must be more effective in reducing morbidity than mortality. More effective primary health care rather secondary or tertiary is the leading contender as the catalyst of this process. The call for a shift away from a curative emphasis in health care research and delivery is also echoed by the supporters of the compression theory. Objections to various aspects of the compression of morbidity theory are, however, raised by other researchers (Myers and Manton, 1984a; Schneider and Brody, 1983; 1984). They argued that risk factor modification through behavioural changes do not change the onset and progression of most senescent-related chronic diseases. Even if the benefits of risk factor modification is proven, its wide adoption across the population is highly questionable.

In addition to the above criticisms, the present author is also critical of Fries' reliance on medical interventions and on medical costs in his arguments. Data on medical interventions emphases on the curative power of medical technology, and reflect utilisation of health services at secondary and tertiary levels. These data are not, however, a good reflection of the population's primary health needs, but are influenced by other factors including socio-economic status, geographical access, public awareness, and other social and cultural barriers. Differences in health for these factors can be seen as indirect measures of the failures of primary health care. Furthermore, in some cases, medical costs bear no direct relationship to the severity and lethality of the condition. For example, one can cite the orthopaedic area where some causes are not life threatening, but profoundly affect the quality of the patient's life.

In many of his theoretical arguments Fries drawn on clinical and experimental studies. Related, then, to the issue of data support drawn on in information of Fries' arguments, the present author would argue, is the implicit assumption that the population will respond to future interventions in the same way as is now observed under controlled experimental conditions. Samples in clinical studies reflect a certain degree of selectivity, and the results may be influenced by a range of other intervening factors. Questions thus arise on the appropriateness of the extrapolation of these results to a population health level.

5.3 Dynamic Equilibrium of Morbidity

At the heat of the morbidity pandemic versus compression debate, Manton (1982) put forward a less radical argument by proposing a "dynamic equilibrium" state. He suggested that decreases in fatality rates are also accompanied by declines in the incidence and progression of chronic diseases. Increases in the duration of morbidity, under an environment of improved survivorship, are offset by decreases in severity through delayed onset and therapeutic interventions, which slow the progression of many diseases.

Manton (1982; 1991) predicted that the prevalence would grow as the length of life increased since chronic diseases are the manifestations of the senescence of human organisms. A chain of casual relationships is the basis of Manton's prediction. He inferred that in the absence of significant reductions in the incidence rate of chronic diseases life expectancy increases can only occur by slowing the progress of the disease process, and, in doing so, increasing the average duration of these diseases. The underlying increases in morbidity duration, compounded by improved survivorship, are responsible for the observed increases in the prevalence of chronic diseases in the population. Advances in medical intervention, which are credited for slowing the progress of chronic diseases, also have the effect of reducing the severity of the diseases.

Multiple-cause-of-death data from the United States suggest a greater prevalence of chronic disease associated with population ageing (Manton et al, 1993). Also reflected in these statistics is that chronic diseases often play a contributory role as causes of death, but with diminishing severity. For most types of cancer, the number of underlying cause of death as percentage of total cause (both underlying and associated causes) increases with age and over time (Manton, 1991), which is a direct indication of the subsiding lethality of cancers and indirectly indicates their reducing severity.

Robine and his colleagues (Robine and Ritchie, 1993; Robine et al, 1997) analysed studies done in developed countries on disability and disability-free life expectancy, measuring expectation of life free from disability (see Chapter Seven for an analysis of the properties of this index and Appendix Three for its statistical specifications). They found that the proportion of disability-free years drops as the disability severity level increases. From that

they concluded that the relationship between life expectancy and disability-free life expectancy is more complex than had generally been thought; disability-free life expectancy does not simply vary in relation to life expectancy. Indicators measuring mild to severe disability have shown some pandemic effects, while most severe disability exhibits neither pandemic nor compression effects (see also Crimmins et al, 1989; 1997a). These findings tend to support the dynamic equilibrium theory.

5.3.1 A Dynamic Relationship Between Mortality and Morbidity

Most prominent in the theory of dynamic equilibrium is a dynamic and multi-dimensional relationship between morbidity and mortality. It is specified in the dynamic equilibrium theory that the rate of progression and severity of chronic morbidity are positively correlated with changes in levels of mortality. Associated with mortality reductions at older ages are therefore declines in the severity and the rate of progression of senescence of various vital organs.

Morbidity compression requires an across-the-board slow-down in *all* the physiological parameters of senescence. The prospect of this scenario becoming real seems remote in the current epidemiological environment where the identification of these parameters is yet to be completed. A more realistic scenario would be the modification of some, but not all, of the parameters of senescence, such that the added years are likely to result in a prolonged period of disability (Olshansky et al, 1993). Under this second scenario, the severity of disability is likely to be lower since the individual's senescence process is subjected to partial amendment. The final result would thus comply with the notion of dynamic equilibrium.

Riley and Bond (1983) found that there is co-existence of a high degree of individual variability in rates of age-related changes, and a relatively intact maintenance of certain physiological functions. These findings are interpreted by Manton and Soldo (1985) as indicating that many of the physiological changes associated with ageing can be identified with age-related pathological changes and are not directly tied to intrinsic senescence processes. They deduce that mortality and morbidity at different levels are interrelated,

so that changes or interventions at one level will have effects through a feedback loop to other health outcomes. That is, improvements in morbidity or a slowing of the rate of progression of chronic disease will have a positive impact on reducing mortality rates.

The theory of dynamic equilibrium is in harmony, in a number of important aspects, with both the pandemic and compression theories. For example, the prediction of rising prevalence rates can be found in both pandemic and equilibrium views. The basic parameters of senescence also play a major role in both the compression and equilibrium arguments, and the plasticity of senescence is emphasised in both. However, neither pandemic nor compression theories specifically recognise the dynamic relationship between morbidity and mortality in relation to chronic diseases, which is the building block of the dynamic equilibrium theory.

5.3.2 Implications of Dynamic Equilibrium

The dynamic equilibrium theory posits a mixed message of population health status. Successful management of many chronic conditions such as diabetes and hypertension (Manton, 1982) has resulted in an increase in their average duration and hence their prevalence. On the other hand, the quality of life of many suffers of these conditions has also been significantly improved through interventions and changes in personal health-related behaviour. On an individual level, a positive health outcome is produced, in the form of improved survival and less suffering from chronic conditions. Whether the aggregation of individual level benefits, allowing for population heterogeneity, can be translated into improvements in population health is still to be tested (Hayward and Heron, 1999).

Positive societal benefits can be derived from both primary prevention and secondary interventions. Through a dynamic relationship future mortality reduction is directly linked with the long term management of diseases, which needs to be integrated as a important component of primary health care. Life expectancy benefits in two ways: first from postponing the onset of morbidity, and second from reducing the severity of the chronic disease, or slowing its rate of progression. Further reductions in mortality risk

are likely to be at the expenses of accelerated resources demanded for arresting the progression of many chronic diseases. Linking reductions in disease severity and mortality decreases will be vital in future health planning.

5.4 Conclusion

Research on contemporary morbidity patterns and trends has evolved into a three-way debate. Each side of this debate has its merits in the face of inconclusive data and their subsequent measurements. There were a great deal of interest in this general area in the late 1970s and early 1980s, when the three leading theories were formulated. Efforts on studying them were, however, hampered by the generally poor availability of good quality population-level morbidity data. This situation started to improve from the early 1990s, with a greater availability of population-level time series data mainly in developed countries, particularly in the United States. Empirical analyses on the leading theories on morbidity trends began to accumulate.

Trends and patterns in morbidity need to be interpreted with caution because of the known problems with data reliability and comparability. Brody (1985) warned of the dangers of extrapolating conclusions from soft data. Circumstantial evidence can be found to support every one of four possible combinations of increasing/decreasing trends of mortality and morbidity. The driving forces of these changes are not consistent in any particular direction. For example, contemporary medical and public health measures coupled with changes in personal health-related behaviour are argued to have affected health in both ways: postponing the age at which degenerative diseases tend to kill, and therefore prolonging life in more years of compromised health; and delaying the onset and progression of these diseases, giving rise to more years of healthy life.

We are presently short of the achievable limits to better population health which might have been set by any of the three leading theories. Morbidity and population health are clearly in transition, and estimating transition parameters in a dynamic environment is inevitably fraught with difficulties. Despite the continuing disagreements on other issues, however, views are unified on the fundamental issue of preventive approaches to managing chronic

morbidity. This debate has highlighted the need for improved primary health care and better integration with policies affecting medical and socio-economic environments.

The continuing ageing of the population is likely to put even heavier strain on social provision. Fries (1984; 1992; 1996) acknowledged the current problem of widespread chronic conditions and cautioned that the problem would grow worse before turning for the better, even under the most favourable scenarios of projected changes. Health policies in the future need to aim to bring morbidity under control rather than mortality. This forces us to reflect on the consequences of contemporary health policies, so that their role will be more of a determinant than a mere response to the changes in public health status (Robine and Ritchie, 1993).

These leading theories on morbidity trends together with the compression of mortality hypothesis reviewed in Chapter Four are synthesised in the next chapter (Six) to formulate an integrated theoretical framework for the analysis of mortality, morbidity and population health dynamics. The operational elaborations of this framework for the purpose of a case study of non-Maori are investigated in Chapter Seven.

PART THREE:

TOWARDS A FRAMEWORK FOR THE ANALYSIS OF HEALTH PATTERNS AND TRENDS

CHAPTER SIX
A THEORETICAL FRAMEWORK FOR THE ANALYSIS OF POPULATION HEALTH

The wide ranging theoretical conjectures on past, present and future trends in mortality and morbidity studied in Part Two help to synthesis emerging issues in population health. The purpose of this chapter is to put these different schools of thought into a common perspective for the purpose of formulating a theoretical framework for the analysis of population health patterns and trends. The framework developed in this chapter, and integrated with more practical considerations, such as data availability, in the next chapter (Seven), guides the empirical analysis of the non-Maori population in New Zealand presented in Parts Four to Six of this thesis.

This chapter starts with an investigation into a population level conceptual model linking mortality and morbidity, the fundamental parameters defining population health (see Chapter One). This model provides the foundation of a framework developed in this and the next chapters (Six and Seven). The attention of this chapter then turns to a critical evaluation of the compression of mortality hypothesis reviewed in Chapter Four. Particular attention is paid to its interpretation, its relationship with the compression of morbidity and its theoretical significance. This is followed by an evaluation of the leading theories on morbidity reviewed in Chapter Five. The focus is on integrating the various theories into a unified framework.

6.1 A Model of Mortality and Morbidity

Current efforts in population level research on mortality and morbidity patterns and trends, and the complex interactions between these variables can benefit from adopting a common perspective. This can be achieved through the use of a population level conceptual model general enough to describe the most significant changes in population health. The purpose of developing a model is to make use of the way a model organises and focuses information so to present a convenient perspective from which the current situation can be viewed and a strategy for research on it can be developed (Tu, 1999).

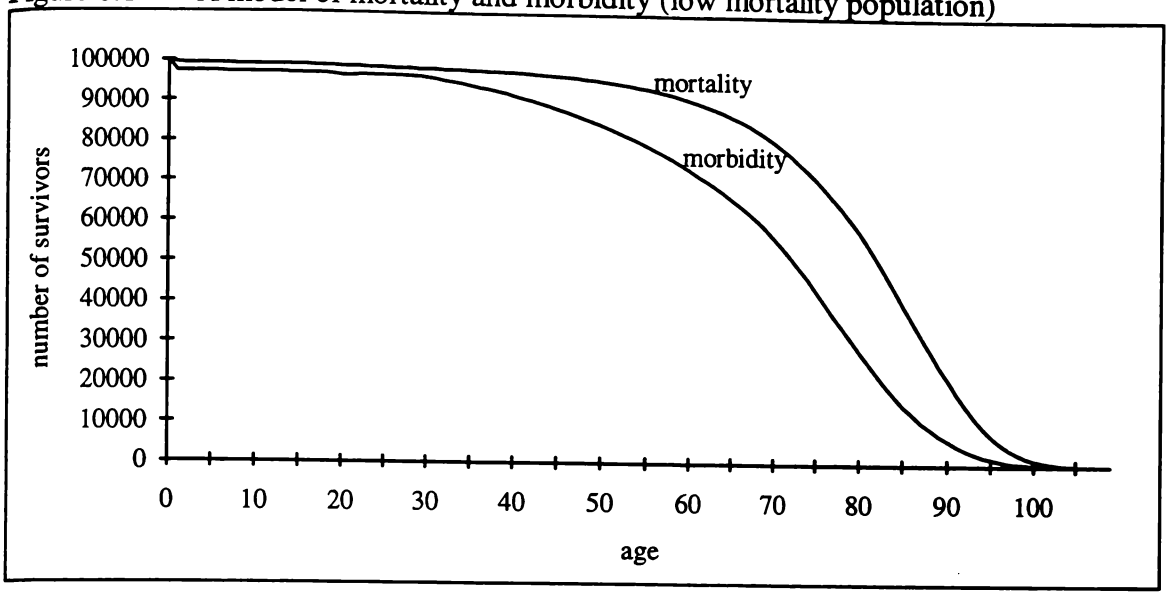
The elaboration of a research strategy development and of the measures of population health status is the function of the next chapter (Seven).

The survivorship function of the life table, which describes the number of people surviving from the original birth cohort as they advance in age, and which is often illustrated by the survival curve (see Figure 4.2), is a versatile tool for analysing mortality. (See Johnstone et al, 1998 for a general introduction to the life table, Namboodiri and Suchindran, 1987 and Shryock and Siegel, 1976 for a thorough understanding of the life table techniques and applications.) The survival curve incorporates two principal determining factors which hold the key to the biological relationship between mortality and morbidity: age and survival. Provided that all other conditions stay constant, each extra year gained, however, entails an added risk of morbidity.

Building on the analytical power embedded in life table techniques, a model of mortality and morbidity was developed at a World Health Organisation Scientific Group meeting on the "Epidemiology of Aging" in 1983 (cited in Manton and Soldo, 1985), with the survival curve as its core and two more curves added to depict the number of survivors at different ages who are free from morbidity or disability. For the purpose of this thesis, morbidity is considered in a broader sense with disability as one of its subsets. The separation of disability as a distinct different state from morbidity without specification of its causes would be mostly semantic and would serve no further purpose. The above-mentioned model is modified accordingly and presented in Figure 6.1.

By linking mortality and morbidity in a very natural and yet simple way, this model serves as the foundation for developing a framework for describing multi-faceted population health changes. The value of this simple model lies in its ability to describe different theories of changes in population health as demonstrated in the next two sections of this chapter.

Figure 6.1 A model of mortality and morbidity (low mortality population)



Source: Modified by the author based on Manton and Soldo (1985)

However, the above model exhibits a number of common pitfalls in mortality and morbidity modelling. First, a uni-directional flow is implied by the model, from morbidity-free, to with morbidity, to death. This is not necessarily congruent with reality for partial or full recovery would warrant a reverse of this transition. Second, the notion of population heterogeneity cannot be adequately reflected in the model. A cohort approach can go some way to alleviate this problem. Both problems could, however, be mitigated through elaboration and careful applications of the model.

6.2 Re-examining the Compression of Mortality Hypothesis

Despite nearly two decades of research, the validity of the compression of mortality hypothesis is yet to be satisfactorily established. Disagreements evolve from loose interpretations of the hypothesis, for, as was shown in Chapter Four, the same piece of information can potentially be used to support both sides of the argument. Problems with the loose statistical understanding of the hypothesis are further complicated by inconclusive research into the biological limits of life expectancy, which otherwise could be an extremely useful reference point in seeking empirical evidence.

As an example of the loose interpretation of the hypothesis, the compression of mortality has typically been perceived by its analysts as a smooth transition towards a heavy

clustering of age at death into a narrow age range (a point argued otherwise by the present author in the following paragraphs). Any bumps along this smooth transition pathway will concern observers who might see it as major contradictory evidence. Following this logic, the compression of mortality and its counter-effect, the expansion of mortality, are mutually exclusive phenomena, but in a purely static sense.

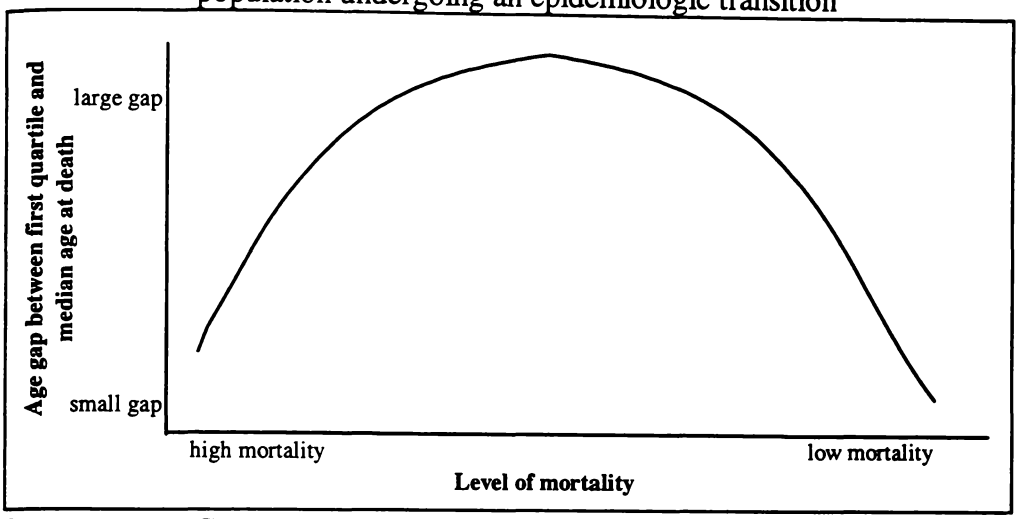
However, the present author would argue that a temporary expansion in the age at death distribution could be part of the dynamic process towards the eventual mortality compression. The explanation is simple, and lies in differentiating the pathways to mortality compression from its supposed end-point (see Figure 4.1). A heavy clustering of the distribution of the age at death is merely the final stage of mortality compression, but the intermediate steps do not necessarily take on a uniform appearance.

Let us explore this point further. For arguments sake, should the theoretical limit to life expectancy at birth be set at 90 years instead of 85 years as hypothesised by Fries, the current peak of the age at death distribution at around age 85 years would move further up the age scale, pulling along with it part of the age at death distribution, but not the entire curve. The new peak would eventually settle at around age 90 years, but before this happened the age at death distribution would become more dispersed, creating almost a plateau between ages 85 and 90 years. Comparing this against the current age at death distribution, with a peak at age 85 years, then a most definitive but temporary mortality expansion would be seen.

This point can be taken back in time to earlier in the epidemiologic transition. Using Pool's method of comparing first quartile and the median age at death, it can be shown that the age gap between these two indicators for the Maori population in New Zealand had actually increased, then decreased over the last century (Pool, 1994). That is, far from a smooth uni-directional transition, mortality compression had actually been preceded by an expansion of mortality during earlier phases in the mortality declines. The relationship between the spread of age at death distribution and level of mortality as illustrated in Figure 6.2 is therefore not a linear one. The curve in Figure 6.2, depicting the correlation between the age gap in first quartile and median age at death and level of

mortality, is for illustrative purpose, and may not be congruent with the exact magnitudes.

Figure 6.2 A hypothetical curve illustrating the relationship between the age gap in the first quartile and median age at death and the level of mortality of population undergoing an epidemiologic transition



Source: Generated by the author based on a study by Pool (1994) on the Maori mortality transition

Experience of the Maori population can be inferred to non-Maori and other low mortality populations, but over a longer time horizon. The key point is, however, that the contemporary controversy over the compression of mortality concerns itself only with the bottom right corner of Figure 6.2. Its counterpart at the bottom left corner may suggest precedents of mortality compression in history occurring at the youngest age groups.

6.2.1 Differentiating Mortality Compression and Morbidity Compression

Death in itself is an unambiguous, once-off event. Life, however, does not generally follow a smooth mechanistic progression from disease to illness to death. Morbid conditions in an individual can be recursive. In fact, partial or full recovery from non-fatal diseases is relatively common in a low mortality environment. The differences between mortality and morbidity become more apparent statistically. In theory, each individual can only make one contribution to the numerator in population mortality rate calculations. In contrast, individuals are potentially capable of making multiple

contributions to the numerator of population morbidity rate calculations in a great number of ways. This widened scope for an individual in the enumeration of morbidity can be extended to most dimensions of morbidity discussed below.

At least five major dimensions of morbidity can be identified (Johansson, 1991): its incidence; its prevalence; its duration; its severity; and the co-occurrence of more than one morbid condition. With the exception of the incidence, which dimension by definition mortality statistics also inherently exhibit, all other dimensions are exclusively morbidity phenomena. Case-fatality rates and data on multiple causes of death, which are essentially diseases-oriented statistics, may be considered as close substitutes for the severity and the co-occurrence dimensions in mortality, but they do not convey the same information about the extent of morbidity as do pure morbidity statistics.

By contrasting the fundamental difference between mortality and morbidity, it becomes less difficult to see that the uni-directional relationship between mortality compression and morbidity compression envisaged by Fries is untenable and misleading. In the rest of this sub-section it is argued that the compression of mortality and the compression of morbidity are concerned with two entirely different phenomena along different demographic and time horizons.

First, mortality compression is in essence a population level concept. It involves a heavy concentration of *population* ages at death in a narrow age range. Individual ages at death data are aggregated and observations are made on the population distribution. Individual data contribute to the overall population statistics but would make no sense on their own. Comparing the ages of death of different individuals can neither prove mortality compression is occurring nor disprove it. Is a death at age 80 years more compressed than a death at age 70 years?

In comparison, morbidity compression is easier to conceptualise on an individual level. Essentially, it is a shortening in the *average* duration of morbidity for a typical individual. Individual observations not only contribute to the population statistics, but they also make good sense in their own right. Comparing individual observations helps to gauge the general direction of change. A one year episode of morbid condition is shorter than two years.

Moreover, morbidity data obtained through many health / disability surveys are essentially micro level datasets.

Second, in viewing the respective phenomena, mortality compression can occur in either a cross-sectional, synthetic sense or a longitudinal, cohort sense. A cross-sectional view, though technically less preferable as argued in the earlier chapters, is made easier because of the general data availability more recently. Viewing mortality in a longitudinal context creates demands for cohort exposure data which may be difficult to obtain. In contrast, morbidity compression in terms of shortened morbidity duration is, by definition, a strictly longitudinal observation. It follows that the compression of morbidity is longitudinal in its orientation. Given the theory's close attention to the age at death as the proxy for the end point of morbidity duration, the occurrence of morbidity compression can only be envisaged from cohort experiences lasting more than a few calendar years.

In short, the compression of mortality is a macro level concept and can be charted by both cross-sectional and longitudinal observations. Compression of morbidity, on the other hand, is based on micro level experience and can only be validated by data on cohort longitudinal experiences.

Due to the known difficulties in collecting morbidity data, cohort morbidity datasets are rare. With the exception of some small scale surveys, studies on morbidity are mostly done on the incidence and the prevalence rates, with little reference to the underlying pattern of duration. As a result, we are left with the paradox that some arguments on the compression of morbidity are formulated purely on the basis of prevalence rates, partly in response to evidence supporting raising prevalence as a part of the pandemic theory. Disagreements such as these help to place the pandemic and the compression theories diametrically opposite to each other. This is not necessarily a correct reflection of the two theories in their original intents, but it is a crucial point for a reconciliation of the different theories of morbidity, and this is picked up again later in this chapter.

6.2.2 Relevance of the Compression of Mortality Hypothesis to the Compression of Morbidity

A compressed mortality profile, represented by a rectangularised survival curve, is a pre-condition to Fries' compression of morbidity theory. The reasons why a rectangularised survival curve is necessary in order that the average duration of morbidity can be compressed is not well elaborated by Fries. This is because Fries makes no attempt to measure the duration of morbidity. Instead he uses a combination of two observations as parts of reference, (i) the onset of morbidity and (ii) death, to approximate the two end-points of an average morbidity episode and to infer population patterns. By introducing an upper limit to which the onset of morbidity can possibly be delayed, the combined patterns of compressed mortality and delayed onset of morbidity will produce shorter average durations of morbidity. It thus appears that the pre-condition of mortality compression is made for purely pragmatic (data) reason.

If a shortened average duration is the desired outcome in population health, it can be achieved through more than one means. Referring to the model of mortality and morbidity in Figure 6.1, the compression of morbidity can be envisaged as a narrowing of the space between the end point of survival (or mortality) and morbidity curves, depicting a shortened expectation of life in a state with morbidity. Compression of morbidity thus viewed can be achieved in the absence of a rectangularised survival curve. The most important determinant is the behaviour of the morbidity curve in response to changes in mortality, not a rectangularised survival curve.

In fact, it has been argued that it is legitimate to expect morbidity compression to work without mortality being compressed (Robine et al, 1997). By dealing directly with the duration dimension of morbidity the upper constraint of a rectangularised survival curve becomes redundant and can be relaxed.

6.2.3 The Value of the Compression of Mortality Hypothesis

The preceding discussions help to set apart conceptually the compression of mortality from the compression of morbidity. The conceptual disassociation of the two, however, does not suggest that the importance of the compression of mortality hypothesis and its research are to be discounted. To provide a just assessment of the value of Fries' compression of mortality hypothesis, it is necessary to reiterate the point made at the beginning of Chapter One, that mortality dynamics are the most readily measurable and arguably the strongest determinant of population behaviours. Over the last decade differences in the tempo of mortality reductions across population sub-groups and over time profoundly alter the demographic composition of a population. The most important aspect of these compositional changes is the changing population age structure. To illustrate this point, we can note that mortality reductions at the earlier stage of the epidemiologic transition were concentrated at infant and early childhood, and produced a younger age structure. Health concerns at the time were justifiably focused mainly on fighting communicable diseases of which the impacts on the younger members of the society were the greatest.

At older ages, it is the level of mortality, not fertility or even migration, which define the entire universe for whom health status is being studied. Mortality reductions at older ages, observed in the second part of the twentieth century, were directly responsible for the increased heterogeneity in the population, even within demographic sub-groups. As death becomes an increasingly weak natural selector, more people from the same birth cohort survive to older age groups and bring with them the diversity in their susceptibility to illness. Further developments of mortality at older ages will have significant impacts on morbidity patterns.

Despite some heavy criticisms it has received, the compression of mortality hypothesis poses a workable framework for the analysis of past, present and future mortality trends. It offers a convenient paradigm for framing contemporary mortality and related issues and synthesising future research questions. By further demystifying the relationship between mortality compression and morbidity compression as forged by Fries, the understanding of issues around future mortality trends and their implications can benefit from more focused analyses.

6.3 Re-examining the Leading Theories of Morbidity Trends

The multi-dimensionality of morbidity makes attempts to theorise its trends and patterns difficult. All identifiable dimensions of morbidity underwent substantial changes during the epidemiologic transition as a result of the shift in the disease profile, and thereby profoundly altered the relationship between mortality and morbidity. Views originating from one or two of the hypotheses about morbidity transitions such as pandemic or compression, tend to generalise the changes as an uni-directional process, but fail to encapsulate the underlying dynamic mechanisms. In this section it is argued that the three supposedly competing theories are not necessarily diametrically opposite to one another, but are potentially reconcilable to an extent that an integrated framework for analysing morbidity changes can be constructed. The following section of the chapter is thus the present author's theoretical elaboration centring on the proposition that these theories are reconcilable.

6.3.1 A Critique of the Leading Theories

The three leading theories on morbidity trends were introduced in Chapter Five. It is generally held that the pandemic of morbidity theory by Gruenberg (1977) and Kramer (1980) and the compression of morbidity theory by Fries (1980) are at the two extremes of the debate. Manton's dynamic equilibrium theory (1982) stands somewhere in-between. It seems that at the heart of the pandemic versus compression argument is the notion of a rising prevalence and reducing duration of chronic morbidity. From a population health standpoint, however, relationships between the morbidity dimensions of incidence, prevalence and duration are far more complicated through micro-macro interactions and confusions over cross-sectional / longitudinal observations. Positing a simple inverse linear relationship between prevalence and duration is unwarranted.

A rising population prevalence rate of chronic morbidity is central to the pandemic theory. Due to a general lack of adequate incidence data on chronic diseases which would suggest significant changes in their incidence rates, it is inferred by the proponents of the pandemic theory that the rise in prevalence is attributable to an increase in the average duration of morbidity. Such inference is somewhat flawed because prevalence,

whether point prevalence or period prevalence, is a population level cross-sectional phenomenon; whereas measuring duration requires longitudinal micro level data and is difficult to deduce from cross-sectional observations. Moreover, factors other than increases in duration may bring about a rise in the prevalence rate. The improved survivorship, especially at older ages, has a direct effect on population heterogeneity. The resulting increases in the number of people who are in high risk category as a proportion of the total population would tip the balance in favour of a rising prevalence. Even if the incidence and the duration are both declining, compositional shifts in the population's collective susceptibility to chronic diseases can more than offset these declines. As a group, the older population may be less robust than its predecessors; individually, however, each may have fewer years of morbidity than what would have been the case under the old epidemiological regime. Whether the average duration has increased or decreased cannot be deduced from a simple cross-sectional snap shot.

The compression of morbidity argues for a shortened average duration of morbidity. As indicated in the earlier discussions, morbidity duration can be observed for individuals. In addressing the issue of chronic morbidity duration, a considerable amount of the evidence presented by Fries is built on clinical data of the individuals. Conceptually population duration patterns constitute the average of aggregated individual durations, but how these are aggregated to a population level is unclear, especially when they are measured cross-sectionally. For example, the time interval between two cross-sectional points may be too short to capture the majority of episodes of sickness developed prior to the beginning of the interval. Or the interval may be too long, so that most people with chronic conditions exit during the interval either through recovery or death, and those who exhibit morbid condition at the end of the interval are mostly new cases. In both examples the incidence rates would be seriously underestimated. Furthermore, morbidity duration is essentially an exclusively cohort experience. Aggregating patterns of multiple cohorts into a single cross-sectional snap shot will significantly distort the picture, especially when dealing with lengthy episodes typical of most chronic diseases.

In presenting his arguments Fries has opted for the soft option of using population morbidity incidence and mortality data to represent the two end-points of average morbidity duration, and hence avoids the data difficulties in analysing the duration patterns directly.

Though methodologically simpler, this approach is harmful for two main reasons. First, delayed onset of chronic morbidity as a part of Fries' compression formula would mean declines in the incidence rate. Reductions in the average duration, the centre piece of the morbidity compression theory, combined with declines in the incidence rate naturally lead to reductions in the prevalence rate, which directly contradicts what is argued by the pandemic theory. Second, doubts remains over the logic in Fries' reasonings. Macro level hypothetical cross-sectional measures (morbidity incidence and mortality) are used to approximate micro level longitudinal patterns (average duration of morbidity) and then extrapolated back to the macro level to theorise the compression of morbidity. Most questionable is the basis for the approximation of the average duration. The two sets of data, morbidity incidence and mortality, relate to two fundamentally different groups of people; those who developed morbid conditions are not necessarily those who died from whatever causes. Combining the age at onset of chronic conditions of one group and the age at death of another to approximate the average duration of an "average" individual would most certainly give rise to some unfounded assumptions on the patterns of morbidity duration. In a sense this problem is analogous in a duration context to the classical "ecological fallacy".

By recognising that a longitudinal reduction in duration and a cross-sectional increase in prevalence are not mutually exclusive phenomena, but can co-exist at some point of the longitudinal process, it seems many of the competing arguments on the pandemic and the compression theories are mistaken. Most of the attempts to settle the dispute in the past use cross-sectional data, which incorporate an inherent bias against compression because of the factor of increasing population heterogeneity.

The theory of dynamic equilibrium can be viewed as an attempt to link the pandemic and the compression theories. Many strong similarities between the dynamic equilibrium and the other two theories exist and have summarised in Chapter Five. Drawing on the dynamic relationships between various dimensions of morbidity and that with mortality as its principal explanatory variable, Manton's dynamic equilibrium theory is capable of explaining the mechanisms behind most observed major changes. In presenting his arguments, Manton (1982) is careful not to directly dispute claims from the other two theories. The analytical and explanatory power possessed by the dynamic equilibrium theory helps therefore to position it as an intermediate interpreter, bridging the

retrospectively focused, descriptive style of the pandemic theory and the prospectively focused, predictive style of the compression theory.

6.3.2 Towards an Integrated Framework

Changes in population health evolve slowly over time more or less in accordance with the transition paradigms reviewed in Chapter Two. Patterns and trends in population health observed during the transitional process would be perceived differently, for example, from cross-sectional and longitudinal perspectives. A lesson drawn from the previous discussion is that a single-dimensional approach is insufficient to describe the dynamic process of changes in population health (see also Section 2.4). Care is needed in translating individual level information to population observations. A high prevalence of morbidity resulting from the replacement of early death by increased exposure to morbidity, may suggest a general worsening in the population health status. The health of the individuals is not, however, necessarily reflected in population trends. Following the epidemiologic transition, individuals can expect lower risks of morbidity at every age compared to their pre-transitional counterparts. Early intervention and better disease management lead to improved survival and quality of life. Moreover, a rising prevalence of chronic morbidity cannot be interpreted simply as a deterioration in the welfare of individuals. There are, of course, other factors at play, including the population age structure and its heterogeneity.

A more general explanation of the macro-micro interaction may reside with the notion of "marginal survivors" (Poterba and Summers, 1987), referring to those who are saved by advances in the curative power of modern medical technology. Disputes over health of the older population arise from a rapidly increasing number, and perhaps also from an increasing proportion in the population, of these "marginal survivors" and their effects on the population health status. In general, the presumed higher level of frailty among the "marginal survivors" pushes up the average susceptibility to chronic diseases in the population. The older population becomes more heterogeneous and less robust as the number of "marginal survivors" increases. A high prevalence of chronic diseases among the older population accompanied by an improved health of individuals in these age

groups compared to their older counterparts at the same age is an indication of population heterogeneity at work. Aggregated measures such as the prevalence rate are not sophisticated enough to capture these changes in population health characteristics.

To construct a methodology for the analytical approach to be used later, it is hypothesised that all three leading theories on morbidity trends reflect certain elements of reality as seen at different stages of the evolution of population health. They do, however, differ in their evaluating perspectives and theoretical emphases. The pandemic theory is formulated from a retrospective perspective, aiming at describing *what has happened*; the dynamic equilibrium theory focuses on the current period, and explains *why and how it happens*; and the compression theory looks to the future, and predicts *what would happen* if certain conditions are met, albeit that these may not be congruent with what is observed by the other two. The past, present and future perspective, and the descriptive, explanatory and predictive emphases separate, but do not mark the three theoretical perspectives antagonistic to one another. In combination they can be a useful framework for directing future research efforts. The hypothesised time orientation of the three theories seems consonant, moreover, with what is implied in a comment by Robine and colleagues, that "[o]ne can imagine that the different theories correspond to different phases in the health transition" (Robine et al, 1997:77).

Operationalising the theoretical framework developed in this chapter for an empirical study on non-Maori is the focus of the next chapter (Seven). The attention will turn to analytical approaches, methodological discussions and issues around data.

CHAPTER SEVEN
A METHODOLOGICAL FRAMEWORK FOR THE ANALYSIS OF NON-MAORI HEALTH PATTERNS AND TRENDS

The last chapter (Six) outlined a theoretical framework for the analysis of past, present and future mortality and morbidity patterns and trends. Not all aspects of the theoretical issues raised can be properly addressed given their broad scope and heavy demands on data. This chapter integrates the theoretical framework with the practical intention of operationalising the theories for an empirical case-study on non-Maori in New Zealand.

The present chapter explains the general analytical approach adopted for the empirical analyses of non-Maori in Parts Four to Six of this thesis. A significant part of this chapter is devoted to the discussion of health expectancy as a population health status measure and its recent developments. Later in the chapter, practical constraints imposed by data availability in New Zealand and their quality are examined.

7.1 A Combined Approach to the Analysis of Population Health Patterns and Trends

It was demonstrated in the last chapter (Six) that a time orientation in looking at mortality and morbidity, whether cross-sectionally or longitudinally, is a critical factor in harmonising and employing various leading theories on mortality and morbidity. A synthetic approach based on cross-sectional data is often the most convenient starting point, because data are readily available for the most part. Using synthetic measures also has the advantage of being able to employ the latest information cutting across both the young and the old. However, as explained in the following sub-section, cross-sectional summary measures obtained by aggregating the experiences of different cohorts can be misleading. Furthermore, the incidence and the duration dimensions of morbidity can hardly be measured, but at best approximated from cross-sectional data sets.

A cohort approach using longitudinal data is technically superior to the synthetic one for the reason that the experiences of the members of the population, in terms of exposure to mortality and morbidity risks, are accumulated and enumerated. Successive cohorts,

rather than successive years, become the focus. Two inherent difficulties often render this approach problematic. Cohort mortality and morbidity experiences take time to unfold. Cohort analyses are then often confronted with incomplete cohort patterns. A more severe obstacle, however, is related to obtaining the relevant data. Provided that the mechanisms for collecting death statistics (for example, central registration of vital statistics) is of reasonable quality, then data issue is less of a problem in studying cohort mortality. But population level longitudinal morbidity data hardly exist in most countries. On a sub-national level, smaller scale longitudinal surveys are few and rarely last longer than a life-cycle stage. More common are repeated cross-sectional sample surveys. In so far as respondents are not individually identifiable from the results of these repeated sample surveys, changes in the various morbidity dimensions can only be indirectly estimated.

The selection of a particular approach, cohort or synthetic, involves a trade-off between technical superiority and data availability. The empirical component of this thesis adopts both approaches where data support, in order to capitalise on their unique strengths. The analytical framework for each approach is explained in the rest of this section.

7.1.1 Synthetic Analysis

Historically, the demographic study of mortality is dominated by an emphasis on synthetic trends, as a direct result of the readily availability and more up-to-date status of the data. In the absence of systematic inter-generational mortality differentials, as is likely to be the case during a pre-transitional era, synthetic trends would be an adequate reflection of mostly random fluctuations in levels of mortality. The emergence of systematic inter-generational mortality declines accompanying the epidemiologic transition has thrown synthetic summary measures of mortality into doubt, because the mortality experiences of the present younger population is an overly optimistic reflection of what happened to the older population at the corresponding younger ages many years ago. Likewise, the earlier mortality experiences of the old are almost certainly over-pessimistic if inferred to the current younger members. More problematic is whether or not the experiences of current older generations, who may be or may not be the hardy

survivors of high levels of mortality and morbidity at younger ages than is the case for current younger cohorts, will be reflected in the behaviour of current younger cohorts when they reach older ages. The resulting cross-sectional summary measures do not represent any particular cohort, but relate to a hypothetical population.

Even acknowledging the potential pitfalls of cross-sectional measures of a population in transition, a synthetic approach is selected with the intention of realising potential values in the available information to the fullest possible extent. Appropriate use of cross-sectional data can assist in forming an overview. Detailed analysis of synthetic mortality trends in Chapter Eight helps to breakdown a century long mortality transition into major periods of changes. Owing to the long time series mortality data, emerging issues from an historical review of mortality trends can be given their appropriate historical context.

A general lack of appropriate morbidity data means that synthetic assessments of morbidity patterns is the only feasible approach (see Chapter Nine). Trends may be inferred from a series of cross-sectional measures, though the interpretations of these trends should proceed with great caution. This is particularly the case in dealing with data relating to the utilisation of health services, because changes in factors other than the underlying morbidity may be responsible for the emerging trends.

Comparing changes in the patterns of cause of death and the patterns of cause of ill-health can help in assessing alternative theories about mortality and morbidity dynamics. Methodological problems, especially in morbidity data collection and their subsequent measurements place heavy constraints, however, on the practicalities of this type of comparison. Limited comparisons with broader cause groups are attempted in the following chapters.

7.1.2 Cohort Analysis

The support for a cohort approach for the analysis of mortality and morbidity is strong (Manton and Vaupel, 1995; Manton et al, 1997b; Reynolds et al, 1998), though it is recognised that these efforts will be hampered by the lack of appropriate data. In

discussing the notion of population heterogeneity in Chapter Three, it was noted that analysis by cohort is possibly the closest available technique for disentangling many of "heterogeneity's ruses" (Vaupel and Yashin, 1985). Cohort approach to the analysis of mortality compression has also been strongly argued for in Chapter Four.

Earlier population level cohort mortality analysis for New Zealand was undertaken by Donovan (1969), for the non-Maori population, and by Pool (1982; 1985), for both principal New Zealand populations. Non-Maori life tables were computed directly from data available as far back as the 1870s. For Maori, Pool had to integrate directly computed life tables for later years and those obtained by using indirect estimation techniques. With the availability of recent mortality data, more complete cohorts can now be included in non-Maori cohort mortality analysis in Chapter Ten. More importantly, the analysis of the compression of mortality, which has been restricted to a cross-sectional assessments in many other studies, can be carried out for non-Maori cohorts in New Zealand (Chapter Twelve) using the cohort life table series.

As explained latter in the present chapter, available morbidity data sets generally do not support a cohort approach to their analysis. Historical hospital admission and discharge data may support a limited analysis by cohort, but great caution is required with this approach because factors relating to diagnosis, access to and duration of stay in hospital tend to operate in multiple directions and would affect different cohort at different stages of their life-cycle in different ways.

7.2 Life Table Techniques and Health Expectancy

The life table is a classical and highly respected technique in demography and allied subjects, such as population biology, actuarial science, and epidemiology (Namboodiri and Suchindran, 1987). It is a "human document ... of man's efforts to prolong the existence to which he instinctively clings, but to which a limit is set, partly by innate biological factors and partly by environmental influences" (Dublin et al, 1949:80). The history of the life table dates back to the seventeenth century Britain, where, in his 1662 publication, John Graunt used church burial records to study the mortality of London.

The life table thus comes down to us from centuries' of development. Its statistical properties and power are well known and link back into well developed areas of mathematical and statistical theory.

Life table techniques are an integral part of the present study. This methodology has been a central tool to demographers in the analysis of many demographic areas, such as mortality, fertility, marriage and labour force participation, to name but a few. In this thesis it is used to study mortality, morbidity and their interactions.

The advantages of the life table over other methods of measurement are indisputable. The most important is that the life table population is not affected by the age composition of the population which it models. The absence of age compositional effects makes comparisons between populations and over time possible and without the need for a standard population. As will be shown in Chapter Eleven, this property also ensures that the life table is the most suitable tool for investigating population issues such as the compression of mortality.

The life table is a powerful tool in that mortality rates at different ages are combined into a single statistical model. Life expectancy is arguably the most well known summary index extracted from the life table. Life expectancy essentially translates a population level phenomenon into an individual context, by giving the number of future years that can be expected for a person at different ages if he/she experiences at all ages, the same age specific mortality of the given year. A more versatile function in the life table is the survivorship function, often illustrated as the survival curve, which describes the number of people from the original hypothetical birth cohort as they survive through to older ages.

The analytical power possessed by the life table allows the exploration of many other contemporary issues in mortality and morbidity. Its capacity for modelling and analysing the issue of mortality compression is one such example. The continuing improvements in life expectancy and the accompanying expansion of the survival curve raises question about the quality of additional years of life gained, as exemplified by the three leading theories on morbidity. The essence of this debate can be modelled by survival curves, disaggregated to

illustrate the impact of morbidity on them, as was shown in the model of mortality and morbidity in Figure 6.1. Health expectancy is thus derived to describe the combined changes in mortality and morbidity. The rest of this section is devoted to a thorough discussion on the concept of health expectancy, the methodological issues and further development in this area for the purpose of this thesis.

7.2.1 The Concept of Health Expectancy

The life table was originally designed for the analysis of mortality data (Dublin, Lotka and Spiegelman, 1949; Shryock and Siegel, 1976). But recent significant methodological innovations, combined with the arrival of more powerful computer technology and the availability of data of better quality, have extended the scope of life table techniques into many different areas, and have also enhanced its popularity across many disciplines. For example, mortality data can be combined with other demographic data in a life table to model the combined effects of mortality and changes in one or more socio-economic, biological and/of biomedical characteristics.

The application of life table techniques in the field of morbidity analysis was somewhat limited, but the situation has changed dramatically since the notion of health expectancy was developed in the early 1970s. The concept of health expectancy is relatively simple. It is used as a generic term for all the population indicators that estimate the average length of time, expressed as expected number of future years, that a person can expect to live in a defined state of health. Health expectancy is based in life table techniques, but represents a conceptual and methodological extension. Taking the level of mortality embedded in the life table as its base, health expectancy then incorporates the morbidity or disability component for partitioning future years of life into different health states.

The principle of the concept of health expectancy has been illustrated earlier in Figure 6.1. The area below the survival curve denotes at each age the number of survivors from the original birth cohort. For the purpose of calculating health expectancy, these survivors are further divided according to their state of health so that space between the two curves represents the number of survivors at different ages meeting some pre-

defined health or independence (quality of health) criterion[1]. Questions regarding the future evolution of population health posed in Chapter Five are concerned with the precise behaviour of the space between the two curves. As mortality continues to decline will morbidity decline in tandem or follow a different trend?

A particularly appealing aspect of the construction of health expectancy is that their computation can be regarded as an empirically driven non-parametric modelling process. Instead of relying on some pre-determined parameters which have been derived from higher level theoretical assumptions, the calculations of health expectancy are entirely driven by empirical data. The absence of the need for pre-defined parameters ensures that the level of assumptions employed will be kept to a minimum.

7.2.2 International and New Zealand Developments in Health Expectancy

An international network of researchers and health administrators (Réseau Espérance de Vie en Santé, REVES) was set up in 1989, dedicated to the development of health expectancy as an indicator of population health status and as a policy tool for health planning. Similar developments soon followed in the United Nations, and more recently the OECD and the European Union. Since 1993, the OECD has included health expectancy in its official health statistics. The World *Health Report 1997* (WHO, 1997) emphasised health expectancy as a key indicator of population health, although the necessary data are not available for most developing countries. Health expectancy has been acknowledged as an essential indicator in the WHO *Health For All* programme, and the *Jakarta Declaration* (1997) identifies the ultimate goal of health promotion as being " ... to increase health expectancy, and to narrow the gap in health expectancy between countries and groups" . Health expectancy estimates are now available for at least 23 of the 29 OECD countries (Romieu and Robine 1997). The United States, Canada, the United Kingdom, France, the Netherlands and Australia have reliable time series extending over a decade or more.

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[1] The criterion employed is typically a level of disability for the main reasons that disability represents a central construct within the concept of health related quality of life, and it lends itself to more standardised measurement than other dimensions of quality of life.

The rapidly growing interest in health expectancy in both research community and the policy arena reflects an increased emphasis on the quality of life (Saito et al, 1999). Ideally, health expectancy should allow one to address two different issues (Pool and Cheung, 1999): to look at health in its wider context — that is, quality of life, in relation to survivorship — and not just mortality; and to provide an evidence-base for policy. Both issues are becoming critical in countries with low levels of mortality and the force of mortality falls mainly on the oldest age groups.

A critical review of the range of techniques developed in this general area, the data available in New Zealand on which they might be based, and thus of their possible applications in the New Zealand context have been published by the present author and colleagues (Johnstone et al, 1998). That review recommended that urgent work is required on the development of health expectancy measures in New Zealand.

The concept of health expectancy was first applied in New Zealand by Graham and Davis (1990) to the non-institutionalised adult population aged 15-64 years using disability prevalence estimates derived from the 1980/81 Social Indicator Survey. These authors have since updated these estimates, based on the disability items included in the 1992/93 Household Health Survey (Graham and Davis, 1998).

The first health expectancy tables for the total New Zealand population were constructed by the present author in collaboration with a colleague from the Ministry of Health (Cheung and Tobias, 1998), using disability prevalence rates from the 1996 Household Disability Survey and the 1997 Disability Survey of Residential Facilities. These surveys provide population disability prevalence rates based on well validated disability questionnaires, and on sampling frames that included both institutionalised and non-institutionalised populations of all age groups, (see Section 7.4.3 and Health Funding Authority and the Ministry of Health (1998) for details of the surveys). Due to the differences in survey design, in population coverage and in methods of calculation, this and the earlier studies by Graham and Davis are not comparable, so that a health expectancy time series does not yet exist in New Zealand.

The study by Cheung and Tobias (1998) and further analyses conducted by the present author (Cheung, 1999b) are preferred because of their total population focus and the more up-to-date data they employed. Findings from these earlier work by the present author are used as the base of the empirical study latter in this thesis.

The lack of a time series data on health expectancy in New Zealand is a major constraint to the present study. The analysis of past trends may yield valuable insights into the drivers of changes. For example, study by Wilkins et al (1994) on Canadian data suggest that changes in health expectancy thus far have been dominated by changes in mortality, and far less so by changes in morbidity, though this is likely to be a function of level of morbidity chosen for the analysis. Future outlooks on morbidity based on cross-sectional health expectancy data investigated in Chapter Fourteen are necessarily tentative, aiming at insights not predictions.

7.2.3 Methods for Calculating Health Expectancy

The idea of health expectancy was originated by Sanders (1964), who proposed a modified life table approach combining the probability of survival and "the subsidiary probabilities of those surviving on the basis of their functional effectiveness" (p.1067). However, he fell short of offering an operationable measures of functional adequacy or effectiveness. This idea was later followed up by Sullivan (1971). He used the observed age specific prevalence rate of disability to partition the person-years exposure, the ${}_nL_x$ function of the life table, into different states of health. Methods for estimating health expectancy soon proliferated, and quickly evolved into three main streams: Sullivan's observed prevalence method; multi-decrement life table methods; and multi-state life table methods, in their ascending order of level of sophistication and demand for data[2]. A thorough review of these methods and recent developments is provided by Saito et al (1999).

Sullivan's observed prevalence method enjoys the greatest popularity, reflecting the current data environment and the data intensive nature of the other two methods (Jagger, 1999;

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[2] There are also other methods suggested to be used in partnership with the life table, including the Grade of Membership model by Manton and colleagues and microsimulation methods (see Saito, 1999).

Nayar, 1999). Compared to the multi-decrement life table and multi-state life table methods, estimations using Sullivan's observed prevalence method are more straightforward. Many potential issues of data quality are also avoided by the simpler Sullivan's method.

The statistical basis of Sullivan's observed prevalence method has been investigated by many researchers (Barendregt et al, 1994; 1995; Bebbington, 1992; Brouard and Robine, 1992; Rogers et al, 1990; Mathers, 1991; Mathers and Robine, 1997; Van de Water et al, 1995). The main concern about this method is the use of prevalence rates, a *stock* measure, rather than the incidence rates, a *flow* measure (Brouard and Robine, 1992). The inherent bias in the prevalence rates in favour of lower health expectancy, as argued by some, is attributed to its dependence on earlier health conditions of each cohort (Mathers, 1996; Mathers and Robine, 1997). The combination of prevalence of disability, determined by health history and prior mortality experience, and incidence of mortality in Sullivan's method sets it apart from a traditional pure cross-sectional life table measures.

Studies by Mathers and Robine (1997; Robine and Mathers, 1993) conclude that under realistic scenarios of moderate long term trends in morbidity incidence and mortality rates, Sullivan's observed prevalence method is adequate for monitoring trends in health expectancy. This method is generally recommended for its simplicity, relative accuracy and ease of interpretation. The general consensus reached by the REVES network is exemplified by the prediction that "for years to come, Sullivan's method will be the most common method used worldwide" (Van de Water et al, 1995:330). For these reasons, this method is also chosen for the present study.

7.2.4 An Innovative Extension of Health Expectancy: Hospitalisation Expectancy

In view of the lack of population-level morbidity data in New Zealand, an extension of the general technique of health expectancy was designed specially for this thesis, combining level of mortality with data on public hospital utilisation. For each age group, age specific rates of hospital discharges are combined with the age group specific average length of hospital stay to calculate the period prevalence of hospital utilisation in the population. This set of period prevalence rates is then incorporated into the life table

using Sullivan's observed prevalence life table method (Sullivan, 1971), the same as that used in the calculation of health expectancies. This allows the disaggregation of life expectancy into time either in contact with or outside public hospitals. Since the former is expected to be only a fraction of life expectancy at each age (see Liang et al, 1996), the focus is on the time spent at public hospitals instead of trying to capture states of positive health which is the normal focus of the general family of health expectancy methods. The resultant measure is termed "hospitalisation expectancy", and is defined as the number of days of the remaining life expectancy expected to be in contact with public hospitals. The number of days is selected as the unit of measurement because the results tend to be superficially suppressed to insignificance when expressed in number of years.

The hospitalisation expectancy has a number of major practical advantages. It uses generally available data with sufficiently large numbers, even at a sub-national level, based on population, not survey samples. This feature has some far-reaching implications for policy research as will be noted below. Of equal importance and appeal is the fact that the methodology of hospitalisation expectancy is very close to, and thus draws its justification from that for health expectancies. Because hospitalisation expectancies use data on both incidence (discharge rates) and duration (bed-stay) to measure length of ill-health, it can be argued that estimates thus derived are more direct approximations and more comparable with the life table duration of exposure-to-risk function than estimates derived from Sullivan's method based solely on prevalence rates. Further discussion of the advantages and disadvantages of this method are held over until Chapter Nine.

The feasibility of hospitalisation expectancy type study was first explored by New Zealand researchers among whom the present author played the instrumental role (Pool and Cheung, 1999; and also personal communication with Martin Tobias at the Ministry of Health). The results of that study are presented in Chapter Nine of this thesis. Subsequent work by the present author with colleagues (Pool et al, 1999) has successfully brought the analysis to a regional level, though those results are not reported in this thesis because sub-national variations is not part of its focus.

Regional analysis of hospitalisation expectancy is supported by sufficiently large numbers in hospital utilisation data. At an international level, most developed countries will have

the required data for the period since Second World War, and developing countries may well have them for major cities where population-level surveys may not exist. Supported by the general availability of data, the technique of hospitalisation expectancy makes possible analyses of variations between populations and over time in population health status and questions relating to the social and economic determinants of health (Pool et al, 1999) for populations which otherwise might not have the data.

7.3 Study Scope

Most demographic studies conducted in New Zealand include an ethnicity dimension. As noted earlier, a broader ethnic classification into two major ethnic groups, the indigenous Maori population and Pakeha (non-Maori, of predominantly European origin, but also include other minor ethnic groups such as Asians and Pacific Islanders) is commonly used. Without disaggregating by ethnicity, patterns and trends of the predominant non-Maori population will effectively hide those of Maori. The definition of ethnicity, however, is particularly pertinent in the New Zealand context as explained in the next section. A diversity in definitions of ethnicity are in use which can influence the population number concerned.

The empirical study reported in Parts Four to Six is conducted exclusively on non-Maori. The reasons for selecting the non-Maori population as the centre of this study are mainly four-fold.

1. The non-Maori health transition is at a more advanced stage than that for Maori, and is similar to those for populations in many developed countries.
2. The non-Maori population has an older age structure. The momenta of mortality and morbidity changes have shifted significantly to older ages.
3. The overwhelming majority in the total population are non-Maori. Other than a larger size, the larger population base, particularly at older ages, helps to alleviate many analytical problems often arise from small numbers.
4. Data on non-Maori span over a century with reasonable quality. This allows analysis by birth cohorts.

Each of these points is further elaborated in the following paragraphs (For detailed studies on Maori mortality, and to a less extent morbidity, trends, see Pool, 1991; 1994).

In reviewing transitions in population health in Chapter Two, it was pointed out that two distinct patterns of transition co-exist in New Zealand (Heenan, 1990; Pool, 1993; 1994). The non-Maori epidemiologic transition fits with Omran's Classical Model, whereas the Maori follows the transitional variant of the delayed model. The non-Maori transition started well over a century ago and by quite early the twentieth century had reached the third stage of Omran's epidemiologic transition and beyond (see Section 2.4). Both mortality and fertility levels for non-Maori are now low, and further changes are expected to be gradual. In comparison, Maori mortality and fertility had dropped rapidly in the last half century, but their levels are still somewhat higher compared to those of non-Maori. The two transitions of non-Maori and Maori differ not only in their timing and velocity, but also in their key determinants relating to the prevailing socio-economic and policy environments.

The historical trends of non-Maori and their current patterns exhibit many strong similarities with changes in other developed countries. For example, in 1995-97 life expectancy at birth for non-Maori of 75.3 years for males and 80.6 for females are just above the OECD average, and over 8 years higher than the corresponding figures for Maori. The issues of rising prevalence of morbidity in association with recent dramatic declines in mortality at older ages are topical to non-Maori as to many other developed countries.

A fundamental demographic attribute shared by non-Maori and populations in other developed countries is an older age structure. That said, in terms of ageing non-Maori fall into the later and lower end of the spectrum of ageing across developed countries, being the least advanced of the later ageing group (Pool, 1999b). At the 1996 census, the average age of the non-Maori population was at around 36 years, some 10 years older than that for Maori. Focusing on the older population, 13 percent of non-Maori were aged 65 years or over compared to 3 percent for Maori. As noted in the opening chapter, survivorship has continued to improve into advanced ages, affecting in the main non-Maori, although Maori are also following this trend. The issue of the longer-term

"numerical" ageing of the non-Maori population is propelled by the combined survivorship improvements and the large post-war baby-boomer cohorts approaching retirement ages. It should be noted that demographic waves created by baby boomer cohorts are a phenomenon unique to non-Maori and some other English-speaking developed countries, and are particularly awkward for non-Maori because of its intense fertility fluctuations since 1939 and the length of its baby-boom (1943-1973) plus the attainment in the boom of bi-model peaks for birth cohort size. The non-Maori population therefore provides a stronger platform for studying population health patterns and trends and their implications than does Maori. Using what we know of the gaps between the two health transitions in New Zealand, inferences to the Maori population can, however, be drawn from studies on non-Maori.

As noted earlier in Chapter One, focusing on the non-Maori population also has major analytical advantages. Non-Maori are the overwhelming majority in the New Zealand total population, accounted for just over 85 percent of the total population at the 1996 Census. Given the older age structure of non-Maori, this percentage figure increases with age, to 96 percent of all people aged 65 years or above, and 98 percent at ages 85 years or above. Since deaths are highly age selective, it is not surprising that the proportion of over 90 percent of all deaths in 1996 coded as non-Maori was higher than their corresponding share for the total population, despite the lower death rates of non-Maori than Maori. At older ages of 65 years or above, 95 percent of all deaths were non-Maori. The relatively larger numbers, both in the numerator and the denominator, support analysis done at advanced age groups. Life table estimates can be made with certain degree of confidence.

Furthermore, the collection of non-Maori demographic data in New Zealand has a long history, back to the 1870s. Compared internationally, data for non-Maori are relatively reliable and sufficiently robust to support time series analysis. With compulsory schooling since 1870s universal literacy and compulsory birth registration since early year of colonisation, non-Maori age reporting in registration statistics is reasonably accurate. In a critical investigation by Kannisto (1994) of mortality data at older ages in 28 mainly developed countries, he ranks the quality of data by four categories: 1) good quality; 2) acceptable quality; 3) conditionally acceptable quality; and 4) weak quality. Non-Maori mortality data were classified as acceptable quality, second from the top

group along with Australia. Maori mortality data, on the other hand, were considered as of weak quality.

The available non-Maori historical data also facilitate analyses by cohort. For those born between the 1870s and the turn of the twentieth century, their complete or near complete history of exposure to the risk of mortality can be compiled. While vital registration for Maori was introduced in 1913, reliable data for Maori started to be collected only from the 1930s or 1940s. Prior to that, Maori life table values were estimated or adjusted by using indirect estimation techniques as done by Pool (1985; 1991).

7.4 Data Sources and Quality

The last section comparing issues that arise from data of non-Maori and Maori demonstrated some practical dilemmas often encountered because of data availability and quality. Not surprisingly, practical considerations arising from data concerns can have a major bearing on scientific study design, ranging from dictating the methodological approach to influencing the overall research agenda. For the present study not all data required are available. Even with the available data sets, the level of details and format of reporting are not necessarily satisfactory.

Most noticeably, New Zealand has a dearth of morbidity data by comparison with some other developed countries (Johnstone et al, 1998; Katzenellenbogen and Cheung, 1998). In New Zealand, health or related surveys at a national level are limited to once-off application. To date we have no repeated population level surveys such as those conducted in countries like Canada, Australia and the United States, which would give comparable synthetic longitudinal information at two or more points in time (except for the National Health Survey which the second survey is currently in the field). A more common approach in New Zealand is to extract and/or to consolidate information from different individual questions from different surveys conducted over certain time period, such as was done by Graham and Davis (1990; 1998) on health expectancy noted earlier. These authors have drawn on information from selected questions from the 1980/81 Social Indicator Survey and the 1992/93 Household Health Survey. This approach of

combining selected information from different surveys is not followed in the present study because the level of comparability between individual questions from these surveys is generally low. With the exception of the 1996 Household Disability Survey and the 1997 Disability Survey of Residential Facilities, almost no other available national survey were collected for the sole purpose of obtaining information on morbidity and related dimensions of health. Thus, instead of maximising information in the entire survey for deriving summary population measures, the use of these other surveys relies on responses from individual questions as a proxy for morbidity.

Four types of data are used in the present study. They are all collected with a national coverage so that the numbers are sufficient to support most population level analyses.

1. Population data from the quinquennial New Zealand Census of Population and Dwellings;
2. Death statistics from vital registration spanning back to 1881, and cause of death data going back that far, but recently from the National Minimum Data Set (NMDS);
3. Population disability prevalence data from the 1996 Household Disability Survey and the 1997 Disability Survey of Residential Facilities (hereafter referred to as the 1996/97 Disability Surveys); and
4. Hospital admission and discharge data from the National Minimum Data Set (NMDS) going back to 1950.

In the rest of this section, the quality aspects of these data sets are discussed separately, covering procedures followed in data collection, coding and validation, level of reporting, consistency over time, and comparability with other sources. In assessing data reliability, particular attention is given to how information on ethnicity is collected.

7.4.1 Population Data from the Census

The census is a reliable long term source of population data, taken in New Zealand for non-Maori since 1851. Relatively reliable and complete enumerations of non-Maori started to be collected from the 1870s, and from the 1930s for Maori. Since 1881, a population census has been taken quinquennially in March/April in the years ending in '1'

and '6', with the sole exceptions of 1931 and 1941, and 1946 when the census was taken instead in September 1945.

Standard Statistics New Zealand validation procedures are used to ensure that the census data are of a high quality. This relates to coding and data entry, and also includes logic checks of the data. Following the last census in 1996, the first Post-Census Enumeration Survey (PES) was carried out by Statistics New Zealand. An undercount of 1.2 percent revealed in the results of the PES was low by international standard. The undercount was higher for males, for people aged 15 to 29 years and for Maori and Pacific Islanders.

Typical issues confronting time series analysis of census information are non-response to certain questions, changes in definition and/or coding practice, and occasional omissions of certain questions from the census. For the purpose of drawing aggregate population counts as the denominators of population rate calculations, most of these issues are avoided, bar from one most noticeable exception: changes in the ethnicity definitions over the last few decades.

Prior to the 1976 census, Maori ethnicity was defined in New Zealand censuses as "persons of half or more Maori blood". In the 1976 census an additional question was added which asked respondents whether they claimed Maori descent, even if they gave a negative response to the question on degree of Maori blood. The net effect of this change appeared to be an over-reporting of people claiming half or more Maori blood (Pool and Pole, 1987, see also Gould, 1992). The question on Maori descent was dropped in the 1981 census, but was picked up again as a separate ancestry and descent question in 1991 and 1996 respectively. Meanwhile, a self-identified, cultural affiliation definition of ethnicity and an hierarchical procedure of ethnicity coding was introduced in the 1986 census, which was also used in the next two subsequent censuses. Under the new system respondents were asked to tick as many circles as needed to show to which ethnic group(s) they belong. If New Zealand Maori is one the groups reported the person is assigned to New Zealand Maori, otherwise a series of procedure is observed to assign to other ethnic groups. Further problems arising from this change is the issue of category jumping, where individuals may change ethnic identity before data collection executes (Kilgour and Keefe, 1992). For the purpose of this thesis, all persons other than those

who identified Maori as one of the ethnic group(s) which they feel belonging to are grouped as non-Maori.

The series of changes in census ethnicity definition and coding practices rendered longitudinal analysis of Maori ethnic data during the change over periods difficult (Pool, 1991). The implications of this extend beyond the census data, affecting comparability between the census and other data sources. The effects on non-Maori population data, however, were greatly mitigated by its much larger population size.

It should be pointed out that the value of the census is not limited to providing the denominator for most population rate calculations, but information provided by individual respondents also allow the analysis of proxy variables known to be correlated with health status. This latter application of census information typically found in studies concerning socio-economic and environmental effects on personal health, and lies outside the scope of the present study.

7.4.2 Mortality Statistics and Cause of Death Data

New Zealand mortality data are considered of very high quality by international standard (Kannisto, 1994; Pool, 1985), and have been collected over a very long period of time. Mortality data are collected through the system of vital registration[3] which started for non-Maori as early as 1851, and 1913 for Maori. Registration of non-Maori deaths have been relatively reliable since the 1870s, although infant deaths may have been under-reported until late in the nineteenth century (Sceats and Pool, 1985). Improvements in Maori death registration were achieved in the late 1940s, but for infant deaths perhaps not until about 1960. Cause of death data from the 1950s, for both non-Maori and Maori, are regarded as reliable, although there are some questions about the accuracy of death certificates (Johnstone et al, 1998). The accuracy of cause of death diagnoses improved over time. The percentage of ill-defined deaths for non-Maori was very low in

[3] In New Zealand registration of vital events are compulsory, a procedure backed up by notification, whereby householders, institutions, etc are obliged by law to notify the occurrence of birth and death. If registration does not follow soon thereafter, penalties are introduced. Reporting is made using a standard form giving certain information.

1950, at 1.1 percent, and was further reduced to less than 0.5 percent by the middle 1970s.

Death registration is backed up by a system of notification, while cause of death is certified normally by a medical practitioner, pathologist and/or coroner. Currently three New Zealand government ministries are involved in the collection, processing and publication of mortality data. The registration and death certificate and/or post-mortem report are collected annually by the Ministry of Internal Affairs. The registration forms are sent to Statistics New Zealand to be included as part of the vital statistics data set. The death certificates, which indicate if a post-mortem is to be carried out, are sent to the New Zealand Health Information Services at the Ministry of Health for cause of death coding, using the International Code of Diseases (ICD) coding system[4]. The cause of death data sets are kept and published by the Ministry of Health as part of the NMDS.

A number of potential problems exist in the analysis of mortality statistics and cause of death data. Age misreporting, particularly at very old ages of 85 years and over, may be a source of concern. Problems arise when age recording relies on second hand reporting and is not able to be verified by birth certificates. This is particularly true for older Maori as birth registrations for Maori prior to 1948 were unsatisfactory, as well perhaps, for a small numbers of immigrants included in the non-Maori population coming from societies in which registration was incomplete. The study by Kannisto (1994) referred to above indicates that non-Maori mortality data at older ages is of generally good quality. The issue of age mis-reporting among non-Maori is of minor concern in comparison with that for Maori. Furthermore, by using five year age groups as is done in this thesis, some of the perturbations created by age-misreporting can be dampened down.

While the accuracy of ethnicity reporting is often a problem area, the effects of this on the present study of non-Maori mortality were significantly limited by the much greater proportion non-Maori deaths in the overall number of deaths. The inherent difficulties with ethnicity classification are that the ethnicity of the deceased is often determined by

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[4] Coding of the underlying cause of death is carried out by the New Zealand Health Information Services following international guidelines and is based on the death certificate and, if available, the post-mortem report. If there is a difference between the two sources the information on the post-mortem report is used.

an observer, usually the funeral director. The family of the deceased is rarely consulted (see Kilgour and Keefe, 1992). Often ethnic identification becomes based on skin colour, a questionable identification technique. Furthermore, non-responses to the ethnicity question on death registration form are subsequently coded as Other, or non-Maori, and this can lead to considerable under-reporting for Maori (Johnstone et al, 1998).

Prior to September 1995 a biological definition of ethnicity was used on the death registration form, asking about the "degree of Maori or Pacific Islander blood" of the deceased's parents. A new form was introduced on 1 September 1995, with the question on ethnicity being the same as that asked in the 1996 census. This change improved the comparability between census and death data, but it also introduced a definitional discrepancy in the mortality historical time series. Again this is an issue which affects Maori data more than those on non-Maori.

A further complication is the accuracy and the consistency of cause of death reporting. The accuracy of cause of death data depends on the quality of the certification of cause. From 1950, when several causes were reported the underlying cause was tabulated. This, in turn, is affected by a number of factors, including whether or not the death was certified by a physician, changes in diagnostic techniques and patterns, and changes in the classification of causes of death (Pool, 1985). Recent mortality declines at older ages and increases in the incidence of chronic multi-causal mortality makes diagnosis of the exact cause of death more difficult. Nevertheless, cumulation of cause of death, as used here, tends to dampen the effects of this problem (Caselli, 1991). At the population level, this is a useful strategy as disease groups rather than site specific afflictions are typically used as population health measures.

In Parts Four to Six of this thesis, non-Maori cause of death data are analysed at an ICD chapter level, and are presented using further aggregations of ICD chapters. At the ICD chapter levels, effects on time series comparisons from changes in the ICD versions are kept at a minimum. Adjustments are made to data in the earlier years for major changes arising from ICD definitional shifts, such as the regrouping of deaths due to cerebrovascular causes (strokes) from "Diseases of the Nervous System and Sense

Organs" to "Diseases of the Circulatory System" in the late 1960s. This reflects the underlying patterns as they would be recorded under ICD version 9 used in 1996.

7.4.3 1996/97 Disability Surveys

The 1996/97 Disability Surveys were the first national surveys on disability undertaken in New Zealand. They were based on well validated questionnaires[5] and sampling frames that included both institutionalised and non-institutionalised populations of all age groups, and were devised from the census and a disability question there (see below). The aim of the surveys were to measure the prevalence of disability among the New Zealand population and to provide information on the nature, duration, severity of disability and their needs for equipments and/or support services. The functional concept of disability was used, defined as any limitation in activity resulting from a long term morbid condition or health problem. The limitation had to be for a minimum of six months (or expected to last for that time) and could not be eliminated through the use of simple corrective devices such as glasses. Respondents were asked whether they experienced any difficulty performing any of the itemised activities because of a long term condition or health problem. For children under 15 years (reported via proxy, typically by parents or care givers) a broader definition was used which also included specific chronic conditions and support needs (Statistics New Zealand, 1997, see the joint report by Health Funding Authority and Ministry of Health (1998) for details of the surveys).

In June 1996, a Household Disability Survey of non-institutionalised population of all age group was carried out. To supplement the household survey and to include those living in health related long term residential facility, a Disability Survey of Residential Facilities was conducted in February/March 1997. The two surveys combined to provide population level data on disability and are collectively referred to as the 1996/97 Disability Surveys.

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[5] The questionnaires, separate for household and residential surveys and for adults and children under 15, are validated and used by Statistics Canada in the 1986 Health and Activity Limitation Survey of the Canadian population.

For the 1996 Household Disability Survey, for the purpose of establishing a sample frame of people with long term disability, two questions on disability had been added to the 1996 Census. A Screening Questionnaire was administered to a sample of 20,848 people, including those who answered "Yes" to the two census questions as well as those who answered "No". This was done to assess the magnitude of false negative in the census questions. A sub-sample of 4,100 who were identified as having a disability were asked to complete a detailed questionnaire. The sampling stratification was based on the four former Regional Health Authorities areas to support most sub-national analyses.

The sampling frame of the 1997 Disability Survey of Residential Facilities included all people over the age of 15 years living in health related long term residential facilities. A sample of 130 such facilities were selected, stratified by size and residential type, and 122 agreed to take part. A screening questionnaire was administered to residents to determine their disability status, among them a further short questionnaire was administered to a sample of 1,016 people who were identified as having a disability.

The response rate for the 1996 Household Disability Survey was 86 percent. For the 1997 Disability Survey of Residential Facilities 94 percent of the facilities sampled eventually took part. Within the participating facilities, 91 percent response rate was achieved. Standard data validation procedures including verification of coding and data entry were employed by Statistics New Zealand. Preliminary processing and analysis of the data by Statistics New Zealand was carried out in collaboration with a Health and Disability sector working group, which has developed a severity scoring system (see Health Funding Authority and Ministry of Health, 1998).

Ethnicity in both surveys was defined and coded using the Statistics New Zealand standard classification, same as that used in the 1996 Census. This ensured complete comparability between survey and census data. The 1996 Household Disability Survey was linked to the 1996 Census which enabled the analysis of socio-economic variables relating to disability (Katzenellenbogen and Cheung, 1998). A wide range of physical, intellectual and mental disability were covered in the survey questionnaires. However, the surveys were not successful in collecting data on causes of disability, since translating

from disability to specific diseases of injuries is often difficult and survey respondents might not be able to attribute their functional limitations to specific causes.

7.4.4 Hospital Admissions and Discharges

Data on hospital admissions and discharges are arguably the most complete, in terms of its national coverage, and one of the few long term morbidity information sources in New Zealand. Published records of public hospital admissions and discharges have been available since 1950, and data for private hospitals were first published in 1974.

All admission and discharge data, including those for mental health reasons, are collected and collated by the New Zealand Health Information Services (NZHIS) at the Ministry of Health. Discharge include deaths in the hospital. These data are part of the NMDS, and are published as three separate volumes pertaining to public hospital, private hospitals and mental health data. The time series public hospital volume was used for the present study. Discharges from private hospitals are predominantly for elective and relatively minor procedures, and constitute a small proportion of all discharges. Details of reporting and reliability of private hospital data are noticeably poorer than that for the public hospitals (Johnstone et al, 1998).

Public hospital admission and discharge data are collected on a continuous basis as the NZHIS requires error free data within 28 days of discharge. The ICD provides detailed codes for all diseases. The government funding agreement between the NZHIS and the Crown Health Enterprises (CHE), which manage public hospitals, stipulates that diagnosis and subsequent coding must be 95 percent correct. This target is not currently being achieved. As at 1997/98, it is estimated that there is an approximately 9 percent error rate in the ICD coding (Johnstone et al, 1998). Co-morbid conditions, especially those require cross-speciality coding, are particularly problematic. Nonetheless the accuracy of ICD coding had improved over the years, a combined result of improved internal data validation by the NZHIS, external audits of hospital data, and increased NZHIS database capacity of accepting up to 25 associated causes (increased from 4 previously). It is worth noting, however, that these data are published by their principal

cause of hospitalisation. The subsequently analysis on patterns of cause of hospitalisation in Chapter Nine is thus limited to the immediate causes.

There is little consistency in the collection of ethnicity information. Currently, information on the ethnicity of the patient is obtained through a pre-admission form or by interviewing the patient during or after admission. There is not a standard format, but research by Sceats (1987) and Kilgour and Keefe (1992) found that none of the admissions forms included in their sample used a biological definition of ethnicity, and neither did any allow for multiple response. The same study also revealed a range of questionable methods used by admission clerk to assign ethnicity, such as judging by patient's skin colour or by surname, making a guess or simply leaving a blank space on admission form. The situation, however, is improving as the general awareness on this issue of the public and the health sector increases. For example, there has been some indication from the Ministry of Health that it is looking at putting in place a standard admission form for hospitals.

Several other problems also exist with using hospital admission and discharge data to measure changes over time. Frequent revisions of the ICD versions make time series comparisons difficult. This problem can be largely overcome by aggregating diseases type at ICD chapter level, as is done in the present study, following the same data processing procedures for cause of death data described above. A variety of reporting formats were used in the publications of hospital admission and discharge data over the last four and half decades. The discontinuities in tabulated publications and the variables included, particularly pertaining to age group and ethnicity, make comparisons difficult. Combinations of measures, of using broader age groups of more than five years, and of interpolating between calendar years are adopted in the present study to accommodate these problems. However, the most important dilemma affecting time series comparisons of hospital admission and discharge data are changes in hospital admission and discharge policy, improvements in diagnostic procedures and reporting practice, and changes in other environmental factors affecting relative access to hospital services. These changes need to be taken into account in interpreting time series trends of hospital utilisation.

7.5 Conclusion

This thesis draws on the complementary strengths of the synthetic and the cohort approaches to the analysis of population health in the empirical study of non-Maori. Supported by the long history of non-Maori mortality data, synthetic and cohort explanatory analyses of non-Maori mortality can be carried out (see Chapters Eleven and Twelve), using Fries' (1980) compression of mortality hypothesis as its starting point. The same cannot, however, be said of morbidity data. Indeed, a major gap in health knowledge in New Zealand as identified by Johnstone et al (1988) is related to limited data on the population prevalence of different ill-health conditions. Health expectancy type analysis, which integrates patterns of morbidity with that for mortality and provides a powerful analytical and policy tool, is currently restricted to applications with data from once-off surveys, but do not yield comparable observations at two or more point in time. Hospitalisation expectancy, an extension from the health expectancy techniques, can go some way to solve this problem (see Chapter Nine).

In the next two chapters (Eight and Nine), detailed descriptive analyses of non-Maori mortality and morbidity patterns and trends using cross-sectional data will set the scene for exploratory work later in the thesis. They are followed by a cohort mortality study in Chapter Ten which is also a first step towards explanatory analyses in Part Five.

PART FOUR:

AN EMPIRICAL ANALYSIS OF TRENDS IN NON-MAORI MORTALITY AND MORBIDITY

CHAPTER EIGHT
A REVIEW OF THE NON-MAORI MORTALITY TRANSITION

Historically, non-Maori mortality rates were low by international standard (see below). In 1996, non-Maori life expectancies at birth of 75.3 years for males and 80.6 for females were in the highest grouping in the world, at around the average for populations in developed countries. This currently high level of life expectancy at birth was attained through more than a century long of mortality declines, which is the subject matter of this chapter.

The secular decline in mortality over the last century primarily reflects a shift in the causes of death, from infectious diseases to chronic degenerative diseases, and the subsequent redistribution of deaths from the young to the old. The essence of this shift in causes of death in most developed countries up to the first three-quarters of the twentieth century is encapsulated by Omran's epidemiologic transition theory (Omran, 1971; 1982) reviewed in Chapter Two. More recent developments, signified by somewhat unanticipated declines in selected chronic degenerative deaths at older ages such as ischaemic heart disease, permit various extensions to Omran's models (see Chapter Two).

The mortality transition of the non-Maori population corresponds to the three-stage classical model outlined in Chapter Two. The Maori population, on the other hand, follows Omran's transitional model with a later onset of mortality decline but at a much rapid pace. As two of Omran's transition models co-exist in the same setting, Pool points out that "New Zealand is an ideal demographic laboratory for an analysis of the key factors producing [demographic] change, and the mechanisms of interaction between them" (Pool, 1993:420).

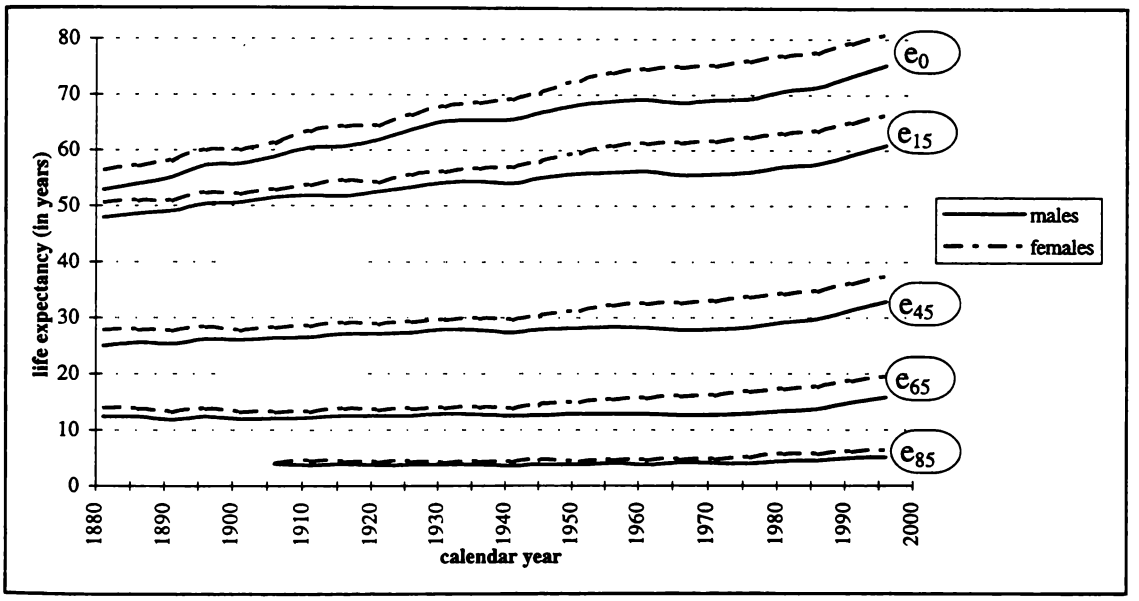
This chapter focuses on the non-Maori mortality trends from 1881 to 1996, in a period corresponding to the middle and later stages of Omran's epidemiologic transition and its proposed extensions. The purpose of this chapter is to set an historical context for the substantive issues dealt with in the subsequent chapters.

The analysis draws heavily on earlier work by Pool (1982; 1985; 1991; 1993; 1994), who has examined in detail the major epidemiological trends in New Zealand for both Maori and non-Maori (see also Cheung 1999a). Cross-comparisons he made (1994) with other countries permit one to identify the key determinants of these trends and the prevailing social and policy environments in New Zealand at the time.

8.1 An Historical Overview

Historical mortality trends can be summarised by life expectancies at selected ages as is shown in Figure 8.1. The corresponding numerical values are tabulated in Appendix One.

Figure 8.1 Life expectancies at selected ages, non-Maori population, by gender, 1881-1996



Sources: Life expectancies before 1950, life tables computed by the author from published raw data by Statistics New Zealand.
Life expectancies after 1950, extracted from completed life tables constructed by Statistics New Zealand.

In general, life expectancies have increased over the last century and beyond as a result of mortality declines, which occurred, however, at different age groups during different periods. As would be expected, the increases in life expectancy were most impressive when mortality declines were concentrated at younger ages. Overall, life expectancy at birth increased by more than 40 percent between 1881 and 1996, from 52.9 years for males and 56.4 years for females to 75.3 years and 80.6 years respectively. Starting from

the middle of the twentieth century to the early 1970s the increases in female life expectancy had out-paced those for males. Gender differentials in mortality declines during this period opened up an increasingly wider gender gap in life expectancy.

Hidden by these seemingly smooth continuous life expectancy trends were, however, some often conflicting forces of mortality change. Figures 8.2a and 8.2b, showing the life table function of probability of survival between key life-cycle stages, are provided to illustrate the underlying trends in mortality. Although the overall trend is one of increases in the probability of survival, period fluctuations and reversals are also visible from these figures. The rest of the analysis is devoted to the understanding of the changes in force of mortality and will refer heavily to these figures.

Also included in this review are analyses of patterns and trends in causes of death These permit us to understand the changing patterns of risk of exposure to communicable and non-communicable diseases at different time periods and by different birth cohorts. Broader cause of death groupings by five major groups are used for this purpose. They are: i) infectious diseases (including tuberculosis), ii) respiratory diseases, iii) degenerative diseases, iv) accidents and v) all other causes. The first two groups constitute communicable causes, and are often referred to as such in the following paragraphs. The category degenerative diseases is made up of cancers and cardiovascular diseases (including cerebrovascular diseases). Superimposed on the graphs are trend lines for cancers and cardiovascular causes for the more recent decades. But even in this period their rates are included in the category degenerative diseases. The last group, other causes, consists mainly of deaths due to other chronic conditions, and, for the earlier periods, also include maternal and infant deaths.

As will be recalled from Chapter Seven, broader cause of death groupings are used to accumulate and so to dampen the effects of perturbations typically associated with smaller numbers of deaths when data are sub-divided by finer cause groups. Some of the potential issues around accuracy of cause of death certifications in the earlier years data can also be resolved through this accumulation process.

Figure 8.2a Probability of surviving n years from age x to age x+n (npx), non-Maori male population, 1881-1996

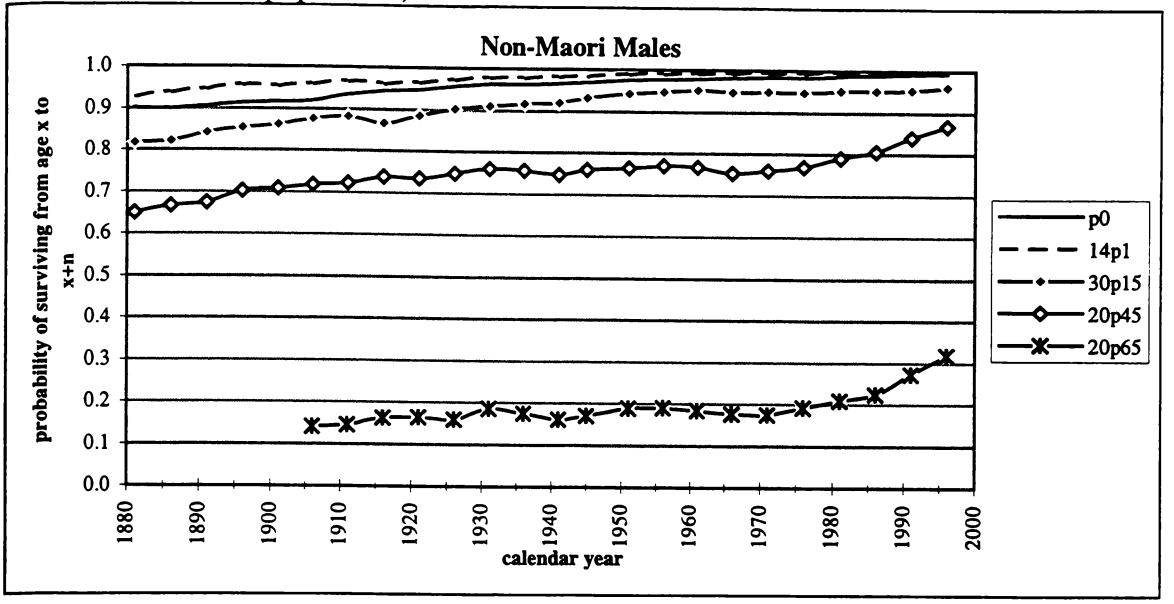
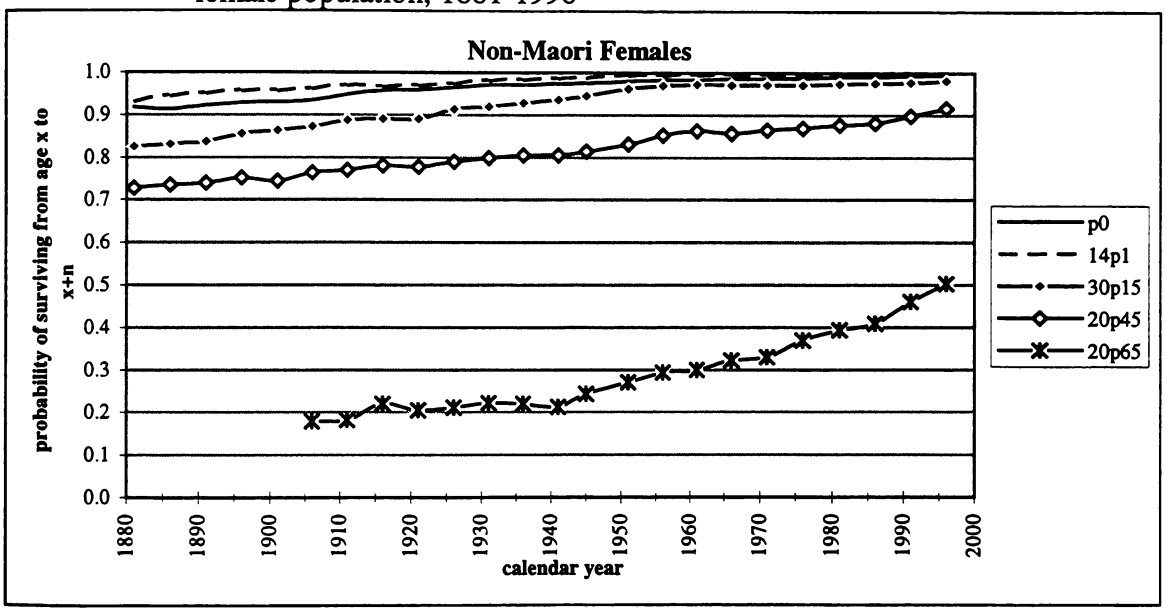


Figure 8.2b Probability of surviving n years from age x to age x+n ($_nP_x$), non-Maori female population, 1881-1996



Sources: Computed by the author from published raw data and life tables constructed by Statistics New Zealand.

Trends in death rate by major cause groups, age standardised[1] to the combined non-Maori male and female populations in 1996, are presented in Figures 8.3a and 8.3b. In addition, from 1951 trends in the two components of degenerative diseases group, cancers and cardiovascular diseases, are also shown separately.

[1] Cause specific death rates analysed in this thesis are age standardised for the entire age range, including the most advanced ages. It is worth noticing that most other epidemiologic studies do not age standardise when dealing with the open-end age group, and their results are therefore more likely to be disproportionately affected by age structural changes at older ages.

Figure 8.3a Age standardised death rates by major causes of death, non-Maori males, 1876-1996

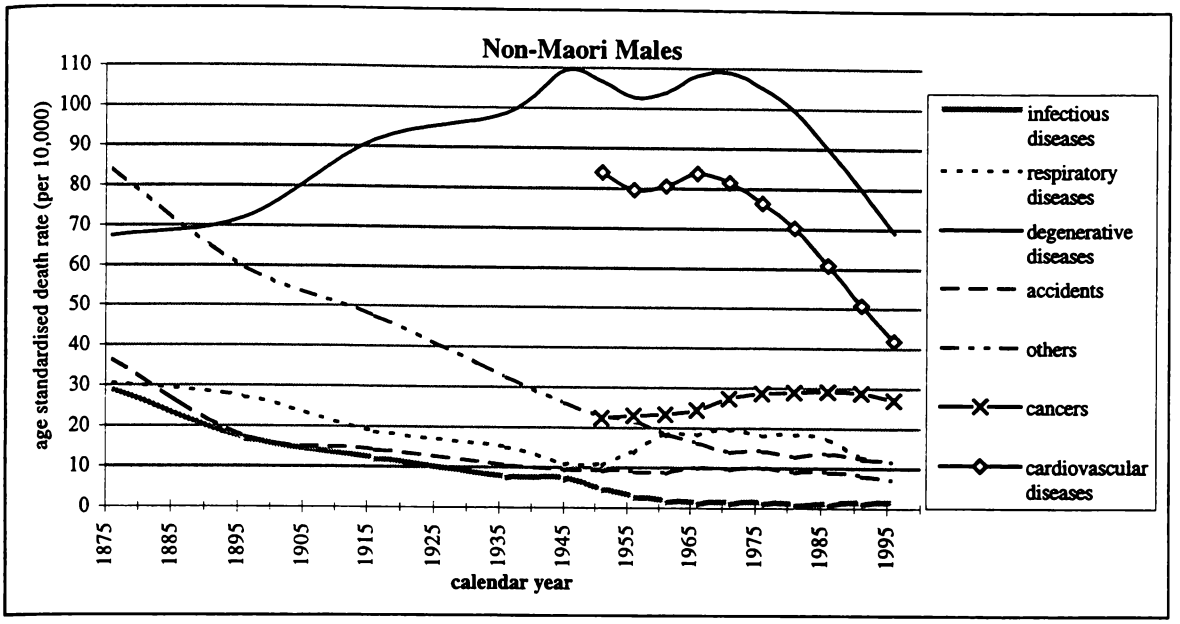
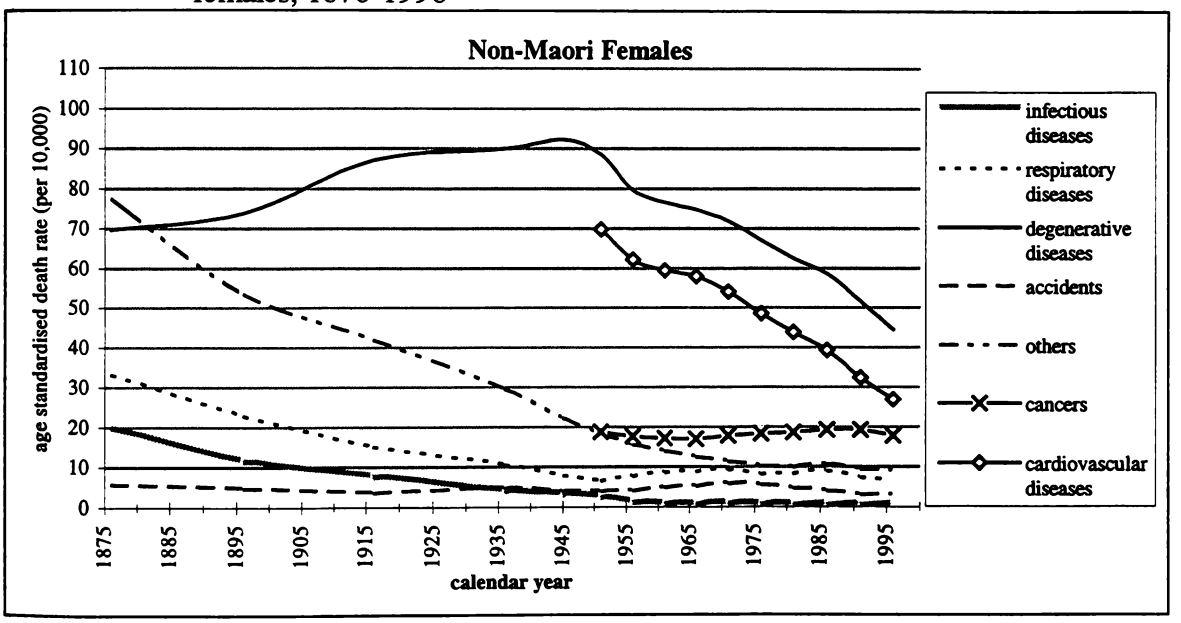


Figure 8.3b Age standardised death rates by major causes of death, non-Maori females, 1876-1996



Note: i) "cancers" and "cardiovascular diseases" are components of the group "degenerative diseases" for the entire period but are also graphed separately for recent decades

ii) "others" include other chronic diseases (ICD chapters 3-6, 9, 10, 12 and 13), maternal and infant deaths (ICD chapters 11, 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Rates before 1950, estimated by the author based on death rates for major groups of cause compiled by I. Pool

Rates after 1950, computed by the author from cause of death data published by the Ministry of Health and population data published by Statistics New Zealand

Figures 8.3a and 8.3b highlights the end stage of the long-term historical shift in the causes of death, from infectious to chronic degenerative diseases. Mortality rates for most cause groups except degenerative diseases have demonstrated significant declines in their historical trends. Until the middle of the twentieth century death rates for degenerative diseases were increasing. The more recent sharp reversal in its trend has been driven primarily by reductions in cardiovascular deaths. By 1996 male death rates for degenerative diseases fell to the level of the late nineteenth century, and female rates went under their corresponding mark since the early 1970s.

The recent declines in the degenerative disease death rates have coincided with a rise in the respiratory disease death rates. The latter is almost entirely attributable to increases in pneumonia and chronic bronchitis deaths at ages 65 years and over. Patterns in causes of death by different time periods are analysed in greater detail in the following sections.

What is interesting, however, is that even in the earliest years covered in these graphs non-communicable causes of death predominate. This situation is very different from that of the Maori transition where even as recently as 1945 communicable causes were more important (Pool, 1994).

For the purpose of this review, changes in non-Maori mortality rates and life expectancies are analysed over five major periods outlined in Table 8.1. The divisions broadly correspond to the periods when certain forces of mortality change were particularly dominant. Each of these five periods will be investigated separately below.

Table 8.1 Major periods of changes in life expectancy between 1881 and 1996

Period	Dominate features
Prior to 1931	phases of "maximum short-run increases in life expectancy" (Pool, 1992)
1931-1941	conflicting forces producing little net movement in life expectancy at birth
1941-1956	steady improvements in survivorship at most age groups
1956-1971	a near stagnation in female life expectancy and a deterioration in males
1971-1996	significant increases in life expectancies at older ages

Source: Prepared by the author

8.2 Prior to 1931: The Period of Phases of Maximum Short-run Increases in Life Expectancy

8.2.1 Changes in Levels of Mortality

Similar to many European populations, systematic declines in non-Maori mortality rates and accompanying accelerated increases in life expectancy started to be observed around the middle of the nineteenth century. That said, however, non-Maori mortality rates were already low compared to those of their contemporary European counterparts. Life expectancy at birth of 52.9 years for non-Maori males and 56.4 years for non-Maori females recorded in 1881 was probably the highest anywhere at that time (Pool, 1982; 1985). This lower mortality and higher life expectancy for non-Maori, however, did not hold at all ages, and certainly was not shared by Maori. Pool (1982) has shown that in 1876 non-Maori life expectancies at ages 30 years and over were lower than those for Norway, which shared some strong demographic and geographical similarities with the non-Maori population in New Zealand.

During the early twentieth century, however, there was a significant improvement in non-Maori life expectancy similar to, but perhaps not as rapid as, those which occurred in most European countries. By the end of the period, non-Maori had lost their top position in the international ranking of life expectancy at birth.

Non-Maori life expectancy at birth increased by more than 20 percent from its 1881 level to 65.3 years for males and 67.8 years for females in 1931. The increase was greater for males than females, although male life expectancies at all ages were consistently lower than those for females. The influence of "crisis mortality" was diminishing, but the effects of pandemic diseases were still evident in their interruptions in a general trend of increases in life expectancy. The period up to 1931 is therefore characterised by regular interruptions in a significant climb in life expectancy at birth.

Three major phases of maximum short-run increases in life expectancy at birth occurred during this period. For both males and females, the first major phase lasted until 1896 by

which year life expectancy at birth had increased from 52.9 years for males and 56.4 years for females in 1881 to 57.4 years and 59.9 years respectively.

The increases in life expectancy were interrupted around 1901 by some minor reversals in the trend of improving survivorship at ages one to 14 years and ages 50 years and over. As is shown in Figures 8.2a and 8.2b, the probability of surviving through childhood ($_{14}p_1$) dropped marginally between 1896 and 1901. Similarly, the probability of surviving 20 years between ages 45 and 65 years ($_{20}p_{45}$) stayed flat for males compared to the earlier years and actually trended downwards for females. These changes combined to produce a marginal decline in females life expectancy at birth. It was the only decline in life expectancy at birth for either genders throughout this entire early period.

A second phase of short-run maximum increases started from 1901. Declines in infant mortality had continued over this period of time at an accelerating pace, in this case, it seems propelled by real changes in public health (Pool, 1982). For males, mortality decreases were halted in 1916 by a number of epidemics, including poliomyelitis and measles, and the early wave of the 1917-1919 influenza epidemic. The 1918 flu, the great influenza epidemic, was arguably the most significant outbreak of infectious disease in the twentieth century New Zealand[2] (Pool, 1985). Most affected were adults at the reproductive ages of 15 to 45 years, particularly males.

Beyond this, a trough in 1916 in the probability of surviving between ages 15 and age 45 years ($_{30}p_{15}$) for males is also clearly visible from Figure 8.2a, although the corresponding drop in male life expectancy at age 15 years was only marginal as a result of some improvements in survivorship at ages 45 years and over. This trough in male survivorship trends at young adult ages, however, needs further elaboration. The effects of World War One were likely to have contributed to the decreases in survivorship at these ages[3].

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2 The main wave of the this epidemic, October through to December 1918, fallen in the middle of inter-censal period. The effects were therefore not reflected in mortality statistics of the three years around either the 1916 or the 1921 censuses, based on which life tables are computed.

3 Between 1914 and 1919 a dislocation of young male population took place on an unprecedented scale as a result of the World War One. However, the departure and return of New Zealand troops of expeditionary force, including those who died on foreign battle fields, were excluded from the census and vital statistics or other published sources, and therefore cannot be included in the present study.

It was estimated that 17,000 New Zealanders died on foreign battle fields out of 552,000 male population enumerated at the 1916 census. Loss of life was particularly severe at the key young male adult ages. The true survivorship between ages 15 and 45 would therefore expected to be even lower in 1916.

The effects of the epidemics on female mortality were less marked compared to that for males. The second phase for females continued later, but was interrupted in 1921 by some widespread worsening of mortality rates at adulthood as is shown in Figure 8.2b, particularly at ages 35 years and over.

By 1921, male survivorship at young adult ages had recuperated from the epidemics and had regained much of the lost ground in survivorship at younger ages. This recovery sustained an increase in male life expectancy at birth of more than one year between 1916 and 1921, despite some worsening of mortality rates at late adult ages. Between 1921 and 1931 sharp reductions in mortality rates occurred before the ages of 65 years for males and 85 years for females, most affecting the probability of surviving between ages 45 and 65 years ($_{20}p_{45}$). The steady climbs in survivorship added three and half years to life expectancy at birth in the space of a decade. This rate of increases in life expectancy at birth even surpassed that observed earlier in the period. The last phase of significant increases in life expectancy at birth for both males and females continued to the end of the period, and slightly further beyond for females.

8.2.2 Shifts in the Momentum of Mortality Reductions

In accordance to Omran's epidemiologic transition, most of the rapid improvements in non-Maori life expectancy at birth occurring during this early period were sustained by declines in infant and childhood mortality. However, as argued below, the argument is less one-sided when the contributions of mortality reductions at the reproductive ages to this significant increases in life expectancy at birth are also considered.

Increases in life expectancy at birth during this period and the proportion of these increases attributable to mortality reductions at infancy (less than one), at childhood (one

to 14), at reproductive ages (15 to 44) and at mid to late adulthood (45 to 64) are estimated in Table 8.2. Methods of estimation are explained in Appendix Two.

Table 8.2 Increases in life expectancy at birth and the percentage of increases attributable to mortality reductions at different ages, by gender, 1881-1931

Age group	Males	Females
e_0 increases	12.4 years	11.4 years
< 1 (p_0)	32%	31%
1-14 ($_{14}p_1$)	23%	26%
15-44 ($_{30}p_{15}$)	26%	31%
45-64 ($_{20}p_{45}$)	15%	12%
65+ ($_{\infty}p_{65}$)	3%	1%

Source: Computed by the author from published raw data by Statistics New Zealand

The combined contributions of reductions in infant and childhood mortality accounted for more than half of the increases in life expectancy at birth. Before the turn of the twentieth century, however, the impact of infant mortality reductions was less marked and was overwhelmed by the effects of mortality reductions at childhood or even young adulthood (which were the leading contributors to increases in life expectancy at the time). The low contributions from infant mortality reductions is likely to be explained by some serious under-estimation of the infant mortality rates in the early years as noted in Chapter Seven. The study by Sceats and Pool (1985) estimated that there were significant under-registrations of perinatal and infant mortality before 1900. Survivorship in the first year of life was likely to be over-estimated in the life tables for the early years and thus the true percentage contribution from infant mortality reductions would be greater than estimated here. Reductions in infant mortality accelerated in the second phase between 1896 and 1916, and had taken a leading role in the increases of life expectancy at birth, contributing more than half of the increases during the second phase. During the third phase between 1916 and 1931, the impact of mortality reductions at infancy and childhood tended to even out, to around 30 percent of the increases in life expectancy at birth for each age group, reflecting a progressively diminishing scope for further decreases at these ages after some accelerated reductions.

The role of mortality reductions at reproductive ages during this early period was equally significant. It contributed around 30 percent of the increases in life expectancy at birth. Its net effect for the entire period surpasses that at childhood, and in the case of females,

rivalling that at infancy. The increased percentage contribution at the reproductive ages in the second half of this period (principally during the third phase between 1916 and 1931) reflects a shift in the momentum of mortality decline from the young to the old. The shift occurred because, after accelerated declines in infant and childhood mortality had slowed down, scope for future mortality reductions at these ages diminished progressively and was more difficult to achieve. In comparison, the impact on life expectancy at birth from mortality reductions at older ages during this period was relatively less significant.

What is important is that the changes in the childhood death rates and female mortality at reproductive ages came at a time when medical factors played little roles except for children after one. Indeed, this is a very interesting period in the non-Maori demographic and epidemiologic transitions as we can document fertility and mortality interactions.

1. Most rapid increases occurred in life expectancy at birth (e_0), infant (p_0) and childhood ($_1p_{14}$) survivorship.
2. The above changes were parallelled by sharp increases in female survivorship between reproductive ages ($_{30}p_{15}$).
3. The combined effects of mortality decreases caused very rapid decline in fertility, which was facilitated by changes in the patterns of age at marriage (Sceats and Pool, 1985). Between 1887 and 1901 the total fertility rate (TFR) reduced by half, from around 7.0 to 3.5. The 1901 level was even lower than those recorded during the baby boom.
4. The rapid fertility decline would have had the effects of lowering further infant and childhood mortality, by reducing the proportion of high risk babies born (to younger mothers, given changes in nuptiality at that time), by reducing incidence of infection and cross-infection among siblings, and by increasing per capita food and amenities available to family members (Pool, 1985:226). Reduced frequency of childbearing also helped to lower mortality due to maternal causes and causes at reproductive ages for women.

For adult males, as will be shown below, reductions in accidents, probably related to work, played an important role.

8.2.3 Changes in Patterns of Cause of Death

The shift in causes of death characterising the middle stage of the epidemiologic transition is demonstrated by non-Maori during this period. The age standardised death rates for degenerative diseases increased substantially over the period while rates for all other major causes reduced by a half or more. The percentage share of the age standardised death rates of all causes, broken down by the five major causes of death, are provided in Table 8.3 to explain this dramatic changes in the pattern of causes of death.

Table 8.3 Percentage distribution of age standardised death rates by major causes of death, non-Maori population, by gender, 1881 and 1931

	Males		Females	
Causes of death	1881	1931	1881	1931
Infectious diseases	12%	4%	10%	3%
Respiratory diseases	12%	9%	16%	8%
Degenerative diseases	27%	60%	34%	65%
Accidents	15%	6%	3%	3%
Others	34%	20%	38%	21%
Total	100%	100%	100%	100%

Note: "others" include other chronic diseases (ICD chapters 3-6, 9, 10, 12 and 13), maternal and infant deaths (ICD chapters 11, 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Estimated by the author based on death rates for major cause groups compiled by I. Pool

At the beginning of the period, deaths due to communicable causes (both infectious and respiratory diseases) were one of the most important single force, accounting for a quarter of the age standardised death rate for all causes. By the end of the period, however, the impact of communicable causes had greatly subsided to less than 15 percent of the age standardised death rate for all causes. Similarly, the percentage share of causes in the "other" category had also fallen sharply, partly a result of improved coding of causes of death. Over the period, degenerative diseases had been firmly established as the leading causes of death. By the end of the period, deaths due to degenerative diseases accounted for almost two-thirds of the age standardised death rate for all causes, more than all other causes combined.

The age standardised death rates for infectious diseases underwent the largest percentage reduction, by nearly 80 percent over the period (see also Figures 8.3a and 8.3b).

Younger age groups tended to benefit more from the declining lethality of infectious diseases than did older age groups. Children before five years of age gained the most. Decreases in deaths due to diarrhoea contributed to over half of the declines in the infectious death rates of children under five years and older adults aged 65 years and over. In comparison, the impacts of reductions in the death rate due to tuberculosis at childhood were only minor, contributing less than 10 percent of the declines in their age specific rates. Reductions in tuberculosis deaths were more important among adults, especially before the age of 65 years, contributing roughly half of the declines in their age specific infectious disease death rates.

The death rates for respiratory diseases, the other major component of communicable causes, had also fallen sharply. The age standardised respiratory disease death rates had been halved during the period, from over 30 per 10,000 to around 15 per 10,000 for males and less than 10 per 10,000 for females. The youngest and the oldest age groups were the biggest beneficiaries. Again, children under five years had the largest reduction in age specific rates of all age groups.

The age standardised death rates for degenerative diseases increased throughout the period. The increases were certainly not shared at all ages. Age specific rates at ages younger than 45 years had reduced steadily. At ages 45 to 65 years, the degenerative death rates increased by around 20 percent for both genders to just under 70 per 10,000 by the end of the period. The biggest increase, however, occurred among males aged 65 years and over, doubled from just over 300 per 10,000 to 650 per 10,000. The corresponding increases for females at the same ages was also marked, from 300 per 10,000 to 450 per 10,000.

Another major group requiring specific attention is deaths due to accidents. While the female age standardised death rate for accidents stayed largely unchanged throughout the period, at five per 10,000 or less, the rate for males had dropped substantially from over 30 per 10,000 to around 10 per 10,000. The fall in the male accident death rates occurred at all age groups, and were more noticeable at adult ages. Up until the 1920s, males at working ages were exposed to the occupational hazards of the primary and secondary sectors of the economy. The shift-shares in industrial labour force in the

following decades with more males moving into the tertiary sector contributed to the declines in work-related accident deaths. In the late nineteenth century, significant gender-imbalance among the older population, as a result of high level of gender selectivity in migration, led to the situation of many males living alone as elderly bachelors or widowers and were at high risk of accident mortality. The sex ratio at late adult ages has since decreased radically, and thus helped to lower the male accident death rates at older ages (Pool, 1982; 1985).

By 1931, the major trends of the first two stages of the epidemiologic transition had been achieved. From 1931 on, however, other complex changes, including mortality deterioration, would occur. A net result of which was to increase the complexity of cohort heterogeneity to which reference will be made later in this thesis.

8.3 1931-1941: The Period of Conflicting Forces of Mortality Change

8.3.1 Changes in Levels of Mortality

Little movement in life expectancy was recorded between 1931 and 1941. Concealed within the flat life expectancy curve in Figure 8.1, however, were some considerable mortality changes in various age groups visible from Figures 8.2a and 8.2b. While the probability of survival through to mid adult ages continued to improve, a worsening in survivorship at mid to older adult ages occurred, though this was slight among females.

For males, life expectancy at birth has barely moved during this period, from 65.3 years in 1931 to 65.6 years in 1941. At all other ages, however, a decline in life expectancy was recorded. The drop in life expectancy was more acute at adult ages where the impact of a worsening in survivorship at mid to older adult ages was more immediate. The same impact was partially off-set by the continuing but slow improvements in survivorship at childhood and young adult ages, producing a small net decline in life expectancy at these ages.

For females, there was a modest increase of 1.3 years in life expectancy at birth during this period, from 67.8 years in 1931 to 69.1 years in 1941. Small increases in life expectancy were also recorded at younger ages. Unlike males, the worsening of mortality rates at older ages was much less marked, and its impact on the life expectancy was felt at a later age of 50 years and onwards.

Two major conflicting forces of mortality changes occurred during this period. The first was continuation from the previous period of mortality reductions at younger ages. This force had weakened considerably from the previous period, with the possible exception of female mortality at the early reproductive ages. The second force of mortality change, mainly among males, was the worsening of mortality at mid- to older adult ages.

The first of the two forces of mortality change noted above could have been expected as a continuation from the previous trend. The same cannot be said of the second. There was no previous trend or earlier indications which might have suggested possible increases in mortality rates at older ages. Given that mortality fluctuations did not occur at younger ages and that the starting ages of these fluctuations were a few years apart for males and females, this force was unlikely be driven by external environmental changes during the period. If it had been, it would otherwise have shown the same effects among other population sub-groups. The explanation of this change may lie in cohort differences in exposure to the risks of mortality, a point investigated further in Chapter Thirteen.

8.3.2 Shifts in the Momentum of Mortality Reductions

Increases in life expectancy at birth and the proportion of these increases attributable to mortality reductions at five main life-cycle stages are estimated in Table 8.4, similar in form to Table 8.2 (see Appendix Two for method of estimation). Given the small margin of change in life expectancy at birth during this period large variations in percentage values by age are to be expected. The focus should, therefore, be instead on comparisons between life-cycle stages rather than on the absolute magnitudes.

Table 8.4 Increases in life expectancy at birth and the percentage of increases attributable to mortality reductions at different ages, by gender, 1931-1941

Age group	Males	Females
e_0 increases	0.3 years	1.3 years
< 1 (p_0)	70%	16%
1-14 ($_{14}p_1$)	87%	19%
15-44 ($_{30}p_{15}$)	104%	60%
45-64 ($_{20}p_{45}$)	-64%	12%
65-84 ($_{20}p_{65}$)	-91%	-9%
85+ ($_{\infty}p_{85}$)	-6%	2%

Source: Computed by the author from published raw data by Statistics New Zealand

Male mortality profiles demonstrated the interactions of the two forces of mortality change. Contributions to the increases in male life expectancy at birth were clearly divided at age 45 years, with positive contributions at younger ages and negative contributions at older ages. For females, the increases in life expectancy at birth were predominantly driven by survivorship improvements at the reproductive ages.

Comparing Tables 8.2 and 8.4, mortality reductions at reproductive ages had gradually taken over from infancy and childhood as the most dominant age group in terms of their relative influence on the value of life expectancy at birth. This marked the shift towards older ages in the underlying momentum of future mortality reductions.

The importance of the reproductive ages in this period, particularly to females, came from a different cause from that pertaining in the preceding period. Earlier for women they had been linked to fertility declines. But by 1931 fertility were very low, and instead we started to observe the effects of a low fertility and systematic ageing in the force of mortality from children to middle ages to old age.

8.3.3 Changes in Patterns of Cause of Death

The overall pattern of causes of death showed only minor changes during this period. Death rates generally continued to fall for most causes, and the previously rapid increases in the degenerative disease death rates decelerated. Consequently, the percentage distributions of the age standardised death rate by major causes of death had

changed little from the end of the last period (shown in Table 8.3), and are thus not reproduced in a table format here. The percentage share made up by degenerative diseases had increased slightly at the expense of all other causes, to account for two-thirds of the age standardised death rate for all causes.

Female mortality advantages over males were established for all major cause groups during this period. The age standardised death rates for infectious diseases stayed unchanged for both genders, at 7 per 10,000 for males and 4 per 10,000 for females. Female age specific death rates for infectious diseases reduced marginally at all age groups. For males, small reductions at younger ages were offset by minor increases at middle to late adult ages. As a result, the impacts of infectious diseases on males became less lethal in the first few years of life than at older ages, reversing the earlier pattern.

The death rates for respiratory diseases continued to fall, although the magnitudes of reduction were only marginal. Most of these reductions were realised from reduced death rates at ages 65 years and over because of their relatively higher levels compared to other age groups.

Compared to the last period, increases in the age standardised degenerative disease death rates had eased. The easing was mostly sustained by declines in age specific rates at older ages of 65 years and over, though rates at younger adult ages before 65 years were still increasing gradually.

8.4 1941-1956: The Period of Steady Mortality Declines

8.4.1 Changes in Levels of Mortality

Between 1941 and 1956, non-Maori enjoyed a period of uninterrupted mortality decline at all ages, rivalling the major phases of maximum short-run life expectancy increases in the earlier period. Life expectancy at birth increased by 3.3 years for males and 4.8 years for females, to 68.9 years and 73.9 years respectively in 1956.

During this period there was a resurgence in the declines in infant and childhood mortality, especially for young males, doubling the rate of decline from the last period. More impressive, however, was the significant improvements in survivorship at mid to older adult ages. This was particularly true for females, as reflected in the sharp increases in female probability of survival between these age groups in Figure 8.2b.

Compared to female, male survivorship improvements at mid to older adult ages were very modest and fluctuating, mostly recuperating from the losses of the previous period. Some temporary increases in mortality rates occurred at ages 60 to 75 years. Notably, most of these males experiencing mortality increases came from the same birth cohort who had also experienced a larger scale mortality worsening during the previous period.

The gender differentials in survivorship improvements at the adult ages resulted in faster rates of increases in female life expectancy at every age. This happened despite young males having attained during this period a greater survivorship improvement at infancy and childhood than had females. Starting from 1941 the gender gap in life expectancy widened, and this persists to this day, as was shown in Figure 8.1.

8.4.2 Shifts in the Momentum of Mortality Reductions

Increases in life expectancy at birth during this period and the proportion of these increases attributable to mortality reductions at five main life-cycle stages are estimated in Table 8.5. Positive contributions were made at all key life-cycle stage to the increases in life expectancy at birth. The considerably smaller share in mortality reductions for males at ages 65 to 85 years reflects the fluctuations and minor reversals in the trends of their age specific mortality rates. Mortality declines at the reproductive ages, which were significant in both genders, accounted for the largest proportion of the increases in life expectancy at birth.

Table 8.5 Increases in life expectancy at birth and the percentage of increases attributable to mortality reductions at different ages, by gender, 1941-1956

Age group	Males	Females
e_0 increases	3.3 years	4.8 years
< 1 (p_0)	22%	12%
1-14 ($_{14}p_1$)	17%	11%
15-44 ($_{30}p_{15}$)	33%	28%
45-64 ($_{20}p_{45}$)	15%	20%
65-84 ($_{20}p_{65}$)	7%	22%
85+ ($_{\infty}p_{85}$)	6%	7%

Source: Computed by the author from published raw data and completed life tables constructed by Statistics New Zealand

For males, the increases in life expectancy at birth continued to be sustained, in large part, by reductions in mortality at younger ages. While for females, a shift to older ages in the momentum of mortality reductions and thus the sources of further increases in life expectancy was more evident.

8.4.3 Changes in Patterns of Cause of Death

A prominent feature in the causes of death pattern in this period was the reversal of the previously increasing trend of the degenerative disease death rates, which had peaked at the beginning of the period (see Figures 8.3a and 8.3b). For males, the decline in the degenerative disease death rates was fuelled by substantial reductions in the age specific rates at ages 70 years and over where the risk was the greatest. At younger adult ages before 70 years, however, there were widespread minor increases in the age specific death rates, especially at ages 55 to 65 years. In contrast, the declines in the female degenerative disease death rates was substantial across all adult age groups.

Another key feature visible from Figures 8.3a and 8.3b was the increases in the respiratory disease death rates, which defied the general decreasing trend exhibited by all other major cause groups. The rise in the respiratory disease death rates was mostly attributable to increases in the pneumonia and chronic bronchitis death rates at late adult ages of 65 years and over. Age specific rates at ages before 50 years continued to decline, most impressively at infancy.

The overall profile of cause of death, expressed as a percentage breakdown of the age standardised death rate by major causes in Table 8.6, had shown only slight movements during the period. The impact of infectious diseases in the overall mortality pattern continued to diminish. Despite the reversal from its previous increasing trend, degenerative disease mortality accounted for an increasingly large proportion of the age standardised death rate for all causes. This was because reductions in other causes, mainly other chronic diseases, were greater than those for degenerative diseases. The percentage share of respiratory diseases had also increased in accordance with the increases in the respiratory death rates.

Table 8.6 Percentage distribution of age standardised death rates by major causes of death, non-Maori population, by gender, 1941 and 1956

	Males		Females	
Causes of death	1941	1956	1941	1956
Infectious diseases	4%	2%	2%	1%
Respiratory diseases	7%	9%	6%	7%
Degenerative diseases	67%	69%	71%	73%
Accidents	6%	6%	3%	4%
Others	16%	14%	17%	14%
Total	100%	100%	100%	100%

Note: "others" include other chronic diseases (ICD chapters 3-6, 9, 10, 12 and 13), maternal and infant deaths (ICD chapters 11, 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Estimated by the author based on death rates for major cause groups compiled by I. Pool; and computed from cause of death data published by the Ministry of Health and population data published by Statistics New Zealand

In his work Pool (1982; 1994) had looked on this period as one in which health programmes, including the effects of providing public access to the available anti-biotic and other chemo-therapies curative methods, had played a major role. But the analysis here suggests that, while this may have been partially correct, other forces were also important. Notably among these were the recuperation following deterioration in the previous period, plus the overall momentum of the upward shift in age at death of the epidemiologic transition.

8.5 1956-1971: The Period of Stagnation and Deterioration in Life Expectancy Trends

8.5.1 Changes in Levels of Mortality

Following a period of rapid life expectancy increases at most ages, in the period 1956-1971 non-Maori mortality trends entered a period of stagnation and marked deterioration in some male mortality rates, in which each male cohort reaching a range of middle to late adult ages had lower survivorship probabilities than its predecessors. There were some strong similarities between this period and the second earlier *period of conflicting forces of mortality change*. Compared to that earlier period, improvements in infant and childhood survivorship during this period were even slower and the fluctuations in adult mortality trends were more widely spread across most ages.

There were few net movements in life expectancy at birth during the period. Male life expectancy at birth first fluctuated to its lowest point during this period to 68.7 years in 1966, before recovering in 1971 to 69.1 years. The net result was only a 0.2 year increase in life expectancy at birth from the 1956 level. However, life expectancies at other ages actually reduced. The fall in male life expectancy was sharper at younger ages, but less so at older ages.

In comparison, movements in female life expectancy at birth were larger in magnitude and more even over the years, increased by 1.3 years during the period to 75.2 years in 1971. It is worth noting that by 1971, for the first time in non-Maori demographic history, female life expectancy at birth equalled and exceeded that at age one. This cross-over is only achieved in populations with the very lowest mortality rates. Upon surviving the first year of life the chance of living into late adulthood was considerably enhanced. The same cross-over did not occur for non-Maori males until 1981.

Between 1956 and 1971 infant and childhood survivorship improvements exerted only a weak influence on life expectancy at birth. While the declines in the infant mortality rate maintained its momentum, the trend in the childhood mortality rate flattened out. This was because the childhood mortality rate had already dropped to a minimum, as is shown

in Figures 8.2a and 8.2b which give the probability of surviving between ages one and 14 years. Further significant reductions in childhood mortality became almost impossible to achieve.

The drop in male life expectancies and the almost flat trends in female life expectancies at adult ages were propelled by several key forces. The first was a rather unexpected increase in the young adult mortality rates in the second half of this period. Some significant reductions in survivorship at ages 15 to 30 years were recorded around 1966. Young males were more affected than young females. Similar declines also occurred for males at ages 30 to 40 years, though the changes were only short-term and had mostly reversed by 1971.

The second key force of mortality change was a deterioration in survivorship at mid adult ages as is shown in Figures 8.2a and 8.2b. This again was more marked among males. Mortality deterioration among adult males generally started at around 1956, while the starting point for females was some five years later. Mortality levels in 1966 were the worst for the entire period. By 1971, the worsening trend in survivorship at these ages had been arrested, and females had seen their survivorship at mid adult ages recuperate back to the levels in 1961.

A third force of mortality change was an extended survivorship deterioration at ages 70 years and over, and only occurred among males. Mortality deterioration at these ages lasted to 1971 and made a greater impact on male life expectancy at birth than that at ages 40 to 70 years, as is shown below. In contrast, female survivorship at these ages made some significant improvements.

Mortality deterioration among young adults was somewhat unexpected, and, to a lesser extent, so was that occurred at mid adult ages. The deterioration at ages 70 years and over had, however, its antecedents revealed by relatively minor mortality fluctuations at younger ages some 20 years earlier. Many of the older males in cohorts undergoing deterioration were from the same birth cohorts which had also experienced fluctuations in mortality rates at mid to older adult ages over the last two periods. Thus a mortality trend by birth cohort was emerging.

8.5.2 Shifts in the Momentum of Mortality Reductions

The shift-share in contributions to changes in life expectancy at birth by key life-cycle stages is shown in Table 8.7. Again, the magnitude of change in life expectancy at birth was small, so that the focus should be more on the general direction and comparisons between life-cycle stages.

Table 8.7 Increases in life expectancy at birth and the percentage of increases attributable to mortality reductions at different ages, by gender, 1956-1971

Age group	Males	Females
e_0 increases	0.2 years	1.3 years
< 1 (p_0)	169%	23%
1-14 ($_{14}p_1$)	59%	9%
15-44 ($_{30}p_{15}$)	27%	5%
45-64 ($_{20}p_{45}$)	-73%	18%
65-84 ($_{20}p_{65}$)	-87%	41%
85+ ($_{\infty}p_{85}$)	5%	4%

Source: Computed by the author from completed life tables constructed by Statistics New Zealand

The deterioration in male mortality rates was highlighted by its negative contributions to the change in life expectancy at birth. As a result, the gender gap in life expectancy had widened at every age. A shift towards older ages in the relative impact of changes in age specific mortality rates on life expectancy at birth was observable for both males and females. What were different from the previous periods, was that the reproductive ages of 15 to 44 years accounted for the least proportion of the change in life expectancy at birth. In contrast, ages 45 years and over assumed a more important role. The older of the two broad age groups included in Table 8.7, ages 65 to 84 years, was more dominant than the younger 45 to 64 years broad age group. This shift was more pronounced among females.

8.5.3 Changes in Patterns of Cause of Death

The deteriorations in male mortality were mostly driven by increases in the respiratory and degenerative disease death rates, while death rates for other chronic diseases continued to decline. Similar increases in the female respiratory disease death rates was

primarily responsible, though to a much lesser extent, for the stagnation in their mortality trends during the period.

A continuation from the trends of the last period, was that the respiratory disease death rates had increased at most ages. The most noticeable change was at the older ages of 70 years and over for males and 75 years and over for females. These increases were more marked in the first half of the period, but tended to ease in the second half.

Increases in the male degenerative disease death rates constituted the second contributing factor to their mortality deterioration. Over this and the next period, the category degenerative diseases is analysed by disaggregating it into its two major components: cancers and cardiovascular diseases. This is because there are marked differences in their trends (see Figures 8.3a and 8.3b). The increases in the degenerative disease death rates in the first half of the period were mostly driven by increases in age specific cardiovascular disease death rates at ages 40 to 70 years, and, to a lesser extent, by increases in age specific cancer death rates at ages 75 years and over. The increases in the male cardiovascular disease death rates at middle to late adult ages were in sharp contrast to declines in the cardiovascular death rates at the oldest ages of 80 years and over.

The deterioration of male survivorship in the second half of the period was primarily attributable to the accelerated worsening of the adult cancer death rates, especially at ages 65 years and over. On the other hand, the male adult cardiovascular disease death rates stabilised and began to decline. Interestingly, younger cohorts in the group with poorer cancer death rates in the second half of the period had also already suffered higher mortality rates from cardiovascular diseases earlier in the period.

Female cancer age specific death rate had fluctuated mostly within a narrow range to yield marginal net movements in this period. Despite some minor increases at ages 30 to 50 years, systematic declines in the female cardiovascular disease death rates at older ages had even produced a clear downward trend in their age standardised death rate for cardiovascular diseases (see Figure 8.3b).

The overall profile of causes of death in Table 8.8 showed an increasingly compressed pattern of causes of death into a restricted number of major causes, namely, cancers and cardiovascular deaths. For males, these degenerative diseases continued to account for progressively larger proportion of the age standardised death rate for all causes. For females, the percentage share of degenerative diseases had levelled out, reflecting the general decline in lethality of these diseases at older ages.

Table 8.8 Percentage distribution of age standardised death rates by major causes of death, non-Maori population, by gender, 1956 and 1971

	Males		Females	
Causes of death	1956	1971	1956	1971
Infectious diseases	2%	1%	1%	1%
Respiratory diseases	9%	13%	7%	9%
Degenerative diseases	68%	71%	73%	73%
Cancers	*15%*	*18%*	*16%*	*18%*
Cardiovascular diseases	*53%*	*53%*	*57%*	*55%*
Accidents	6%	6%	4%	6%
Others	14%	9%	14%	11%
Total	100%	100%	100%	100%

Note: i) "cancers" and "cardiovascular diseases" are components of the group "degenerative diseases"
ii) "others" include other chronic diseases (ICD chapters 3-6, 9, 10, 12 and 13), maternal and infant deaths (ICD chapters 11, 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Computed by the author from cause of death data published by the Ministry of Health and population data published by Statistics New Zealand

8.6 1971-1996: The Period of Significant Life Expectancy Increases at Older Ages

8.6.1 Changes in Levels of Mortality

The period between 1971 and 1996 is a period of unprecedented rapid declines in non-Maori mortality rates at older adult ages and accompanying increases in life expectancies. For example, after nearly a century of very little change, male life expectancy at age 65 years increased by a quarter (3.2 years) over the relatively short span of 25 years, from 12.6 years in 1971 to 15.8 years in 1996. The sudden upward trends of life expectancies at selected ages are clearly visible in Figure 8.1. The upturn in female life expectancy

during this period, which had started much earlier around 1940, was less steep than that for males. Gains in life expectancy at birth were made mostly from mortality declines at older ages, and, not surprisingly, these trends moved in tandem with life expectancy shifts at older ages.

Declines in the infant and childhood mortality rates continued from the previous periods. The declines among young males were faster than those for females, although females still maintained their advantages in infant and childhood survivorship. By 1981, male life expectancy at birth exceeded that at age one. This cross-over in male life expectancies was some 10 years later than that occurring for females.

The worsening of survivorship at adolescents ages and for young adults aged 15 to 29 years starting in the middle of the previous period continued into the early part of the more recent period. Despite some minor reversals of this trend later in the most recent period, the probability of surviving the 10 years from age 15 to 24 years for both males and females was still lower in 1996 than it had been in 1961. Again young males were more affected by these mortality fluctuations than were females.

Some minor mortality fluctuations also occurred for males at ages 30 to 40 years in the early part of this period. These mortality fluctuations appeared to be the flow-on effects from mortality deterioration experienced by the same cohort at their adolescence and young adulthood during the latter part of the previous period. By contrast, adolescents and young adult cohort of the most recent period who had experienced a moderate worsening in mortality rates early in the period did not show any signs of adverse effects in their survivorships near the end of the period when they were at ages 30 to 40 years. In fact, survivorship at mid adult ages for this birth cohort improved progressively, albeit that the improvements were small.

Mortality movements at younger ages were overshadowed by systematic declines in mortality rates at ages 40 years and over. By 1976, males had recuperated most of the losses from the adult mortality deterioration they had experienced in the previous period. At ages 45 to 64 years, increases in male survivorship were particularly rapid, at 0.6 percent a year, more than double that for female. The sharp gender contrast in

survivorship improvements at ages 45 to 64 years can be seen by the two different gradients of the upward climbing probability curves in Figures 8.2a and 8.2b.

Increases in survivorship at ages 65 to 84 years were equally significant, and more impressive in the case of females. Survivorship increases for males were in direct contrast to the fluctuating but mostly flat movements in the earlier years, while increases for females started much earlier and had accelerated during this period.

At ages below 80 years males made larger improvements in survivorship than females. This helped to close the gender gap in life expectancy at younger ages. In contrast, the larger improvements for females at the more advanced ages was more remarkable because female mortality rates at these ages were already significantly lower than those for males.

The dominant force of mortality changes during this period was evidently the remarkable declines in mortality rates at mid to older adult ages. This trend was a sharp reversal from the trend that had lasted through the previous 40 years from 1931. The worsening of mortality rates among adolescents and young adults is also of interest, although there is so far no evidence of adverse consequence in their mortality experience later in life.

8.6.2 Shifts in the Momentum of Mortality Reductions

Contributions at key life-cycle stages to the rapid increases in life expectancy at birth are shown in Table 8.9. Following a trend started earlier in the previous periods, mortality reductions at childhood and reproductive ages had little overall impact on the life expectancy at birth, since survivorship at these ages were already high. Infant mortality still maintains a considerable influence although the percentage share for it too are expected to diminish over time.

The shift towards older ages having a relatively greater impact on the overall mortality profile became more evident during this period. Mortality changes at ages 45 years and over accounted for more than 60 percent of the overall increases in life expectancy at

birth. For females, the shift was faster and occurred at even older ages. Future mortality changes at the more advanced ages will have increasingly significant impacts on the overall mortality experience of the population.

Table 8.9 Increases in life expectancy at birth and the percentage of increases attributable to mortality reductions at different ages, by gender, 1971-1996

Age group	Males	Females
e_0 increases	6.2 years	5.4 years
< 1 (p_0)	13%	12%
1-14 ($_{14}p_1$)	4%	4%
15-44 ($_{30}p_{15}$)	7%	7%
45-64 ($_{20}p_{45}$)	32%	23%
65-84 ($_{20}p_{65}$)	32%	39%
85+ ($_{\infty}p_{85}$)	12%	15%

Source: Computed by the author from completed life tables constructed by Statistics New Zealand

8.6.3 Changes in Patterns of Cause of Death

The unprecedented rapid declines in non-Maori mortality at older ages occurring during this period were principally driven by declines in the chronic degenerative disease death rates. Most noticeable was the impressive decline in the cardiovascular disease death rates throughout the entire period. This was followed by declines in the respiratory disease death rates in the second half of the period.

The persistent declines in the cardiovascular disease death rates between 1971 and 1996 had cut the age standardised rate for cardiovascular diseases by half for both genders, from 82 per 10,000 for males and 54 per 10,000 for females to 42 per 10,000 and 27 per 10,000 respectively. Reductions in age specific rates were consistently substantial at all adult ages, particularly at ages 45 years and over for males and at ages 50 years and over for females, a life-cycle stage at which the risk of cardiovascular deaths increases exponentially with age.

The cancer death rates changed little during the period. The age standardised cancer death rates increased marginally until 1986 for males, and until 1991 for females. Declines in the cancer death rate were observed only very recently, with some noticeable

decreases at most adult ages in the last inter-censal period between 1991 and 1996. For males at ages 85 years and over, however, increases in age specific cancer death rates persisted to the end of the current period.

The respiratory disease death rates also declined during the period. Most of the declines were achieved in the second half of the period when declines in age specific respiratory disease death rates at infancy and older ages accelerated in both males and females. After staying at approximately the same level for almost half a century, the accident death rates also decreased during this period. Most of these decreases again occurred at infancy and older ages of 65 years and over.

The percentage breakdown of the age standardised death rate by major causes in Table 8.10 reveals a stable but highly concentrated cause of death distribution, which was a direct result of substantial reductions in the cardiovascular disease death rates. The combined rate of cancers and cardiovascular diseases seems to have stabilised since the middle 1940s to around 70 percent of the age standardised death rate for all causes. Within this two groups, however, some shift-share effects — from cardiovascular diseases to cancers — were happening (Pool, 1994).

Table 8.10 Percentage distribution of age standardised death rates by major causes of death, non-Maori population, by gender, 1971 and 1996

	Males		Females	
causes of death	1971	1996	1971	1996
infectious diseases	1%	1%	1%	1%
respiratory diseases	13%	11%	9%	10%
degenerative diseases	71%	70%	73%	71%
cancers	*18%*	*27%*	*18%*	*28%*
cardiovascular diseases	*53%*	*42%*	*55%*	*43%*
accidents	6%	7%	6%	5%
others	9%	11%	11%	14%
Total	100%	100%	100%	100%

Note: i) "cancers" and "cardiovascular diseases" are components of the group "degenerative diseases"
ii) "others" include other chronic diseases (ICD chapters 3-6, 9, 10, 12 and 13), maternal and infant deaths (ICD chapters 11, 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Computed by the author from cause of death data published by the Ministry of Health and population data published by Statistics New Zealand

8.7 Discussion

The evolutionary theories of senescence presented in Chapter Three suggest that the non-Maori mortality transition analysed in this chapter represents a major departure from a much longer historical trend of human survival. The high level of life expectancy now enjoyed by non-Maori is unprecedented in human history, and was achieved over an extremely short time span on the evolutionary time scale (Carnes and Olshansky, 1993). The historical significance of this mortality transition is therefore unmistakable.

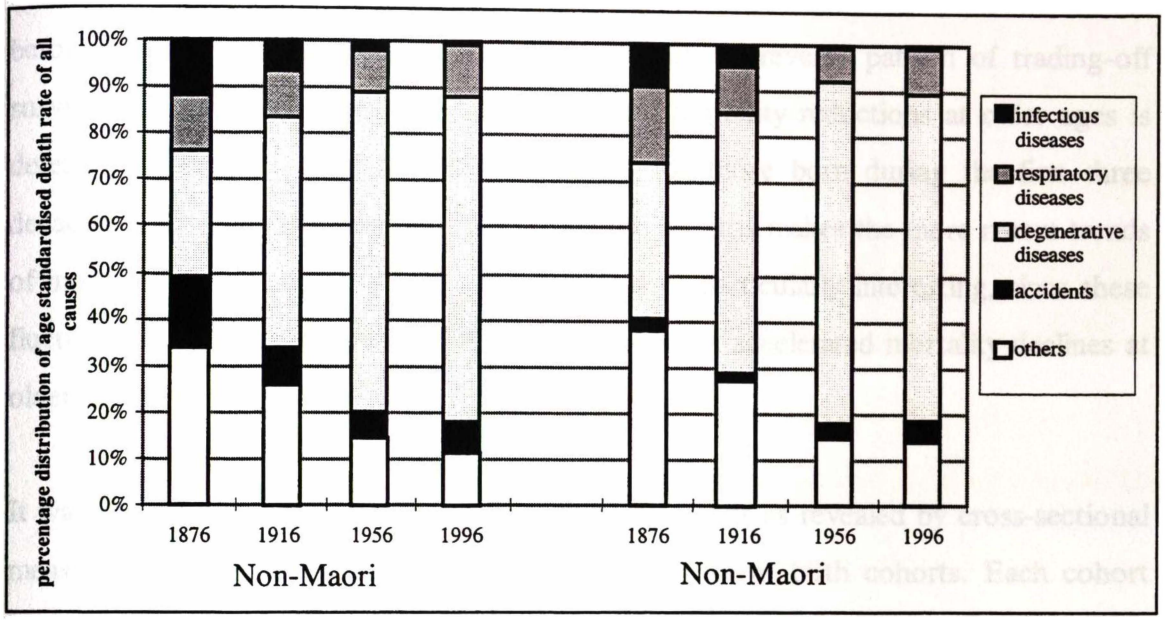
An important issue emerging from the analysis of non-Maori historical mortality trends is the shift in mortality patterns, generally from the young to the old. Not only has age at death shifted upwards, but the force of mortality had also shifted, carrying with it the momentum of future mortality reductions. As a result, life expectancy at birth, a summary measure of the overall mortality profile of a population which is often used as indicator of population health, is becoming increasingly dependent on mortality movements at older ages as demonstrated by the series of Tables 8.2, 8.4, 8.5, 8.7 and 8.9.

A more immediate issue is, however, whether or not recent trends in non-Maori mortality can be expected to last indefinitely. In particular, the significant mortality declines at older ages over the most recent period cannot be sustained at their current pace for very long. One can hypothesise that these rapid changes will decelerate in some form which will profoundly affect the size and the composition of the elderly non-Maori population and, above all, its health profile. This issue will be investigated in several subsequent chapters of this thesis. Chapter Eleven is devoted to the investigation of the shift in age at death in synthetic life tables for the period 1971 to 1996. The same question is investigated from a cohort perspective in Chapter Twelve. In that same chapter (Twelve) findings from both synthetic and cohort studies are contrasted and their implications for future mortality developments are discussed.

Another trend unveiled from the analyses in this chapter is the concentration of cause of death, as summarised by Figure 8.4. In the second half of the twentieth century, the major part of mortality came from chronic degenerative diseases. Deaths due to cancers

and cardiovascular diseases accounted for roughly 70 percent of all deaths, even after controlling for the age distribution of the population. The impact of communicable diseases had receded significantly from a quarter of all deaths a century ago to just over 10 percent in 1996.

Figure 8.4 Percentage distribution of age standardised death rate by major causes of death, non-Maori population, by gender, 1876, 1916, 1956 and 1996



Note: "others" include other chronic diseases (ICD chapters 3-6, 9, 10, 12 and 13), maternal and infant deaths (ICD chapters 11, 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Computed by the author based on death rates for major cause groups compiled by I. Pool, and from cause of death data published by the Ministry of Health and population data published by Statistics New Zealand

A pattern of substitution between causes of death is emerging. Declines in the lethality of communicable and other chronic diseases have widened the scope for other leading causes of death. However, despite this increased potential, the risk of dying from cardiovascular disease has reduced significantly and the cancer death rate has stayed constant over the more recent years. This seems to suggest that were it not for widened risk due to the declining lethality of other competing causes, the cancers and cardiovascular disease death rates would have fallen further. The trend of substitution between competing risks of death suggests that further reductions in the risk of a particular cause of death could be achieved at a cost of increased risk of other causes. If reducing death rates and increasing life expectancy are the primary health policy

concerns, a more holistic approach, rather than tackling selected diseases, would therefore seem more appropriate.

The dominant forces of mortality change identified in this chapter have helped to uncover what appear to be cohort effects in mortality patterns, particularly for non-Maori males. A pattern of trade-off between strong gains in survivorship at younger ages and worsening mortality at mid to late adult ages is evident among older male cohorts born before or around the turn of the twentieth century. A reverse pattern of trading-off survivorship gains at younger ages for significant mortality reductions at older ages is detected in the male cohort mortality patterns for those born during the first three decades of the twentieth century. These trade-off patterns makes the more recent trends of mortality fluctuations at young reproductive ages particularly interesting, since these fluctuations may signal a trade-off for these cohorts an accelerated mortality declines at older ages.

It was argued in the earlier chapters that mortality trends as revealed by cross-sectional measures mask different mortality experience of different birth cohorts. Each cohort carries along its unique experience of past exposure to the risk of mortality, which manifests itself temporally in association with all other cohorts when measured in a simple cross-sectional snap shot. Major changes in mortality trends as measured by cross-sectional observations are the aggregated outcomes of accumulated prior risk exposure by different cohorts. Synthetic trends are, therefore, unnecessarily complicated by multiple cohort mortality patterns at selected ages, but the emerging trends strictly do not apply to any particular cohort.

Major mortality transitions are expected to occur over a long time horizon, generally longer than that implied by synthetic period trends. The antecedents of these major transitions were often revealed in the history of exposure to risk of death of different birth cohorts. A mortality analysis of non-Maori birth cohorts is therefore the most logical approach to a fuller understanding of this population's mortality transition. This analysis is undertaken in Chapter Ten of this thesis.

The broad mortality trends described in this chapter will have been related in complex ways to changes in patterns of morbidity. In Chapter Nine the question of morbidity is analysed in detail for the more recent period, representing the end of the epidemiologic transition. Then a less robust and detailed analysis is taken back to the end of the Second World War, covering the two more recent periods identified in the present chapter, one of a slowing of declines in mortality, the other of a return to improvements in survival.

CHAPTER NINE
NON-MAORI MORBIDITY PATTERNS AND TRENDS IN RELATION TO MORTALITY

Chapter Eight laid out the epidemiologic transition pathway followed by a non-Maori population in attaining its current low level of mortality. This chapter turns to the accompanying issue of morbidity, and shows how the morbidity patterns and trends are related to changing levels of mortality. In this chapter in-depth attention is given to the analysis of morbidity patterns at around 1996, for which date there is a robust data set. This helps to establish a baseline for inferring likely future developments in the later chapters of this thesis. But in doing this, this chapter also looks back over time in a more superficial way. Morbidity analyses can be carried out only for recent decades, simply because the required data are either not available or in inadequate format (see Section 7.4).

Beyond this, compared to the analysis of mortality, morbidity analyses are often burdened with many conceptual and definitional difficulties, as a direct consequence of the multi-dimensional characteristics outlined in Chapter Six. The methodological and data issues in both mortality and morbidity analysis have been dealt with in Chapter Seven. In most circumstances, it is the data available which determine the morbidity research agendas and the quality of measurements.

For the purpose of a population level analysis of non-Maori morbidity patterns and trends two major morbidity data sources are used in the present study. These are:

1. population level disability prevalence rates from the 1996/97 Disability Surveys
2. public hospital admissions and discharges data from the National Minimum Data Set (NMDS) by New Zealand Health Information Services at the Ministry of Health, for the years 1950 to 1997. Public hospital discharge data include deaths in hospital.

These two data sets measure different dimensions of morbidity: (i) data from the 1996/97 Disability Surveys measure functional limitations (often as results of injuries and/or diseases), while hospital discharge data relate to causes of sickness and hospitalisation; (ii) the Surveys data rely on self reporting, while hospital data are based on clinical assessments. Each of the two data sets has its unique strengths, but both are limited to

prevalence measures of selected aspects of ill-health and to the more recent periods (see Sections 7.4.3 and 7.4.4).

In this chapter, morbidity patterns as measured by the relevant data sets are analysed in relation to the corresponding level of mortality where possible. Giving full considerations to the shortcomings of historical hospital discharge data (see Section 7.4.4), a main objective of this chapter is, to the extent data allow, to contrast trends in hospital utilisation with mortality trends over the last half century (which covers two recent major periods of mortality changes, 1956-1971 and 1971-1996, as identified in Chapter Eight), and thus to analyse interactions between mortality and morbidity.

9.1 Current Patterns of Disability of non-Maori in Relation to Mortality

The 1996/97 Disability Surveys, the combined 1996 Household Disability Survey and 1997 Disability Survey of Residential Facilities, provide New Zealand-wide estimates of population-based disability prevalence rates. The two surveys were designed to allow pooling of data collected and hence the calculations of population-based disability prevalence rates, including persons both living in health and disability-related institutions or living in the community. (See Section 7.4.3 for a detailed description of the surveys.)

To link these data to the provision of disability support services and the eligibility for these services, a Health and Disability sector working group has developed a severity scoring system (Health Funding Authority and Ministry of Health, 1998). Severity definitions were based on reported need for assistance rather than the numbers of activities which the person could or could not perform. Therefore the scores incorporate not only functional limitations but also limitations in social roles expected of persons in various social contexts.

The disability severity levels were defined as follows:

- Level one represents those who have a disability (ie. those who report a function limitation in response to one or more of the items in the questionnaire) but who do not report requiring any assistance.

- Level two represents those who report requiring assistance to live independently, but who do not report requiring such assistance on a daily basis (ie. those who report requiring personal assistance or the use of an assistive device on an intermittent basis).

- Level three represents those who report requiring intensive assistance on at least a daily basis. Typically this involves assistance with the self-care domain. These people may or may not live in an institutional setting.

In the following analysis, levels two and three are sometimes combined and referred to as disability requiring assistance.

9.1.1 Population Level Disability Prevalence

In 1996/97 it is estimated that 193 per 1,000 non-Maori males (292,200) and 202 per 1,000 non-Maori females (324,400) have some form of disability. Among these people 105 per 1,000 males and 123 per 1,000 females require assistance with activities of daily living (ie. levels two and three combined). Furthermore, 25 per 1,000 males and 37 per 1,000 females require assistance on at least daily basis (ie. level three). As will be noted below, the relative meaning of these rates internationally is hard to assess because of differences in the measurements relating to conceptual, methodological and even cultural factors (see Section 9.1.2).

These crude rates mask significant variations by demographic groups. For example, the crude rates for non-Maori are not too different from that for Maori despite the existence of ethnic differentials in health status documented elsewhere. This is because Maori have a much younger population age structure compared to non-Maori, and disability prevalence rates increase exponentially with age. It is estimated that the rate of disability in the overall adult population increases by three percent with each additional year of life (Health Funding Authority and Ministry of Health, 1998:72)

Figures 9.1a and 9.1b present disability prevalence rates for each of the three severity levels by five year age groups[1]. The increases in the prevalence of disability over age become evident from around age 50 years onwards. The apparent discrepancy in rates for males between the age groups 10-14 and 15-19 years is a result of the use of different questionnaires and the broader definition of disability for children (Health Funding Authority and Ministry of Health, 1998).

Figure 9.1a Age specific disability prevalence rates by five year age groups, non-Maori males, by levels of disability, 1996/97

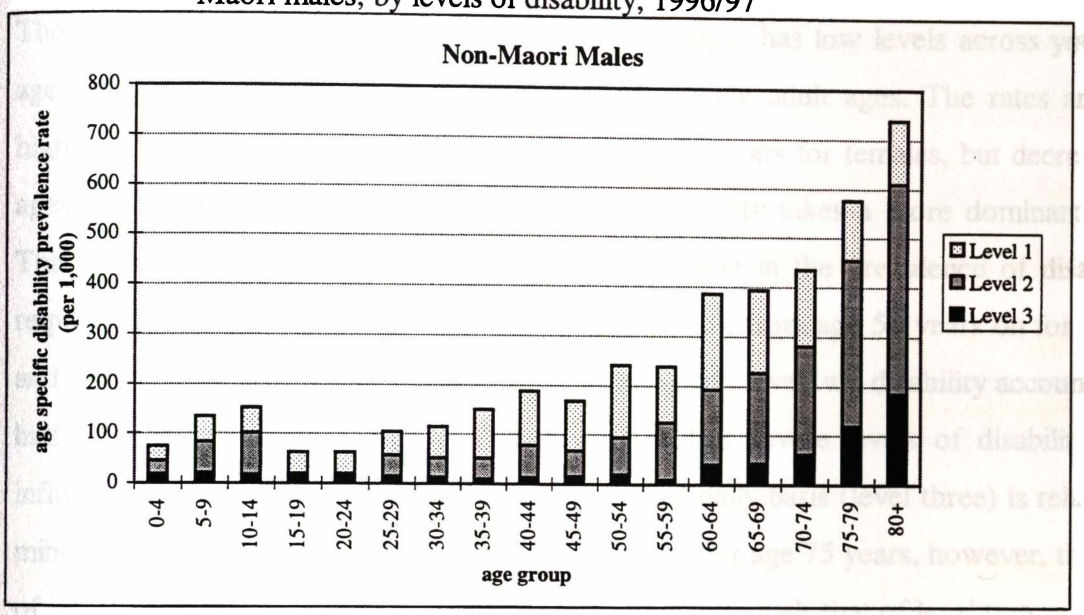
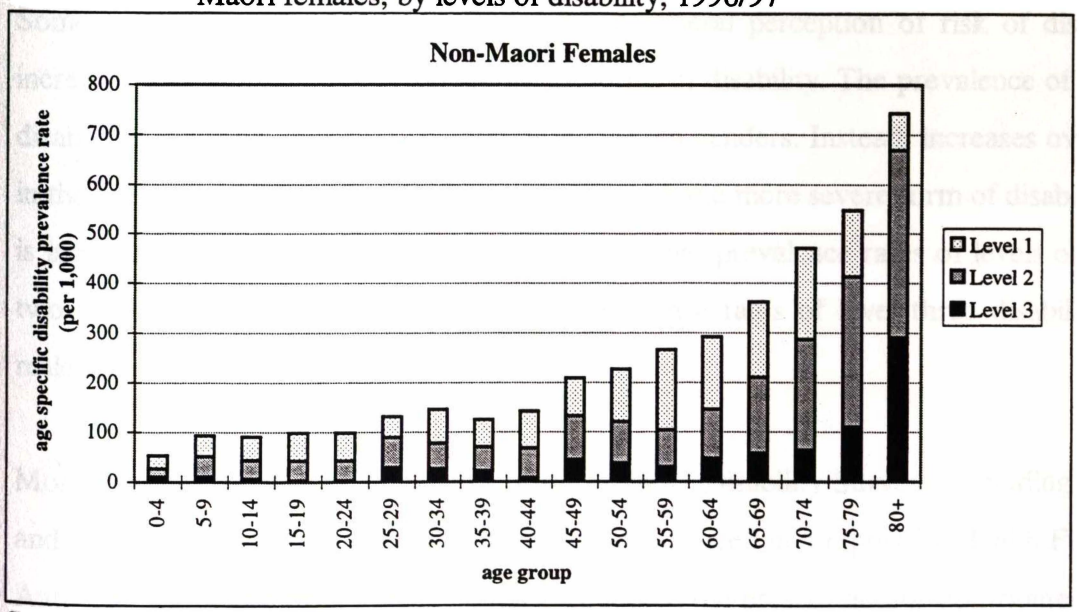


Figure 9.1b Age specific disability prevalence rates by five year age groups, non-Maori females, by levels of disability, 1996/97



Source. unpublished processed data by Statistics New Zealand

[1] Ages 15 to 24 years were grouped as single age group in the original data set. It is split here into two age groups 15-19 and 20-24 to conform with the five year age grouping used in the remainder of this thesis, assuming the same disability prevalence rates for both groups.

Non-Maori boys are over-represented among children with disability/chronic conditions, reflecting the biological vulnerability of boys to negative health and disability outcomes and other gender specific behavioural factors which tend to increase the probability of injury among boys (Health Funding Authority and Ministry of Health, 1998). It is interesting, however, that disability prevalence rates at ages 15 to 24 years are substantially lower for males than for females. This may reflect the general unwillingness among young males to report poor health.

The minor disability (level one), requiring no assistance, has low levels across younger ages, and starts to increase in magnitude at the middle adult ages. The rates are the highest at ages 60 to 75 years for males and 55 to 75 years for females, but decrease at ages 75 years and over as disability with greater severity takes a more dominant role. This latter point is reflected in the increases over age in the prevalence of disability requiring assistance on an intermittent basis (level two), from age 55 years on for males and age 60 years for females. At ages 70 years and over, level two disability accounts for half or more of all disabilities. Compared to the less severe levels of disability, the influence of disability requiring assistance on at least daily basis (level three) is relatively minor before age 75 years, especially among males. After age 75 years, however, the rate of increases in the prevalence rate of level three disability rivals that of level two.

Some general observations can be made. The general perception of risk of disability increases with age may not hold true for all forms of disability. The prevalence of minor disability has shown to reduce at older ages for both genders. Instead, increases over age in the prevalence of disability tend to start later with the more severe form of disability. It is interesting that while males have a generally higher prevalence rates of levels one and two disability than those for females, the prevalence rates of level three disability for males are lower than that for females.

More extended analysis of the results of the 1996/97 Disability Surveys, including social and ethnic comparisons of disability patterns, are included in a report by Health Funding Authority and Ministry of Health (1998). To which the present author contributed both instrumentally and significantly and which is drawn on here.

9.1.2 Disability in Relation to Levels of Mortality

The following analysis in most part draws on earlier work by the present author, part of which was in collaboration with colleague from the Ministry of Health (Cheung and Tobias, 1998). This collaborative work reporting on findings concerning the total population in New Zealand was peer-reviewed by local and international experts in the field and published in a report by the Health Funding Authority and Ministry of Health (1998). Further work using the 1996/97 Disability Surveys was subsequently carried out by the present author in a commissioned and peer-reviewed report to the Ministry of Health and is also drawn on below (Cheung, 1999b).

Age specific disability prevalence rates of all levels can be summarised and integrated with the current level of mortality in health expectancy calculations. The concept of health expectancy has already been introduced in Chapter Seven. Health expectancies reported in this section were calculated from age specific disability prevalence data from the 1996/97 Disability Surveys and the 1995-97 complete life tables for non-Maori. The construction of the abridged health expectancy life tables are explained in detail in Appendix Three, and the full results are given in Appendix Four. An actuarial technique known as graduation is used in the construction of the abridged health expectancy life tables to reduce random sampling errors associated with small samples. The standard errors of health expectancies are also included in Appendix Four.

A series of three disability-based health expectancies were constructed based on a hierarchy of increasingly severe disability prevalence rates (levels one to three), using Sullivan's observed prevalence life table method (Sullivan, 1971). They were:

1. Disability-Free Life Expectancy (DFLE) - life expectancy free of any self reported functional limitation, ie. no disability;
2. Independent Life Expectancy (ILE) - life expectancy free of any self reported functional limitations requiring assistance, ie. total of no disability and level one disability; and
3. Active Live Expectancy (ALE) - life expectancy free of self reported functional limitations requiring continuous daily assistance in the self-care domain, ie. total of no disability, levels one and two disability.

In the original work by the present author health expectancies were calculated separately for the total, Maori, and non-Maori populations, for males and females. It is worth reiterating that after comparing the different results, the present author and his collaborators recommended using independent life expectancy (ILE) and the ratio between ILE and life expectancy as the most robust and relevant indices for policy purposes. The concept of morbidity used to construct ILEs (disability requiring assistance, ie. dependency) is both robust and transparent, and has direct implications for the funding of health and disability support services.

Life expectancy and the three disability-based health expectancies at selected ages (at birth, 15, 45, 65 years), representing the starting points for key life-cycle stages, are summarised in Table 9.1. The full results by five-year age intervals are provided in Appendix Four. Health expectancies are positive measures, capturing health in various positive states. Their reciprocals, "expectation of life with disability" (levels one, two and three), "expectation of life with dependency" (levels two and three), and "expectation of life with dependency for continuous daily assistance" (level three) can be derived by subtraction.

Table 9.1 Life and health expectancies and ratio of health expectancy to life expectancy at selected ages, non-Maori population, by gender, 1996/97

Age	Life expectancy (in years)	Health expectancy (in years)			Ratio of health expectancy to life expectancy		
		DFLE	ILE	ALE	DFLE:LE	ILE:LE	ALE:LE
				Males			
0	75.3	58.7	65.6	73.1	0.78	0.87	0.97
15	61.0	45.8	52.3	59.0	0.75	0.86	0.97
45	32.8	20.8	25.4	31.2	0.63	0.77	0.95
65	15.8	7.6	9.9	14.3	0.48	0.63	0.91
				Females			
0	80.6	62.0	68.9	76.9	0.77	0.86	0.95
15	66.2	48.7	55.1	62.7	0.73	0.83	0.95
45	37.1	23.1	28.0	34.3	0.62	0.75	0.92
65	19.3	9.3	11.9	16.7	0.48	0.62	0.87

Source: Calculated based on the 1996/97 Disability Surveys and life tables constructed by Statistics New Zealand

According to the disability patterns observed in the 1996/97 Disability Surveys, the majority of future years of life at all ages would be expected to be spent in states of positive health or without disability. For example, more than three-quarters of life

expectancy at birth (58.7 years for males and 62.0 years for females) are expected to be disability-free. Another additional seven years are expected to be with mild disability but living independently not requiring assistance. Severe disability is restricted to less than five percent of life.

This pattern of health expectancy and in relation to life expectancy stays largely unchanged before age 15 years. The ratio of health expectancy to life expectancy declines between ages 15 and 45 years. After age 45 years, however, all three health expectancies decrease more rapidly than does life expectancy. About two-thirds of life expectancy at age 45 years would be spent free of functional limitations, and approximately a quarter would be in a state of dependency (the remainder being in a state with level one functional limitations but not requiring assistance).

After age 65 years, less than half the remaining life expectancy would be disability-free. Even so, almost two-thirds of this time would still be spent independently, and only about 10 percent of the time would require continuous assistance.

Female life expectancy is higher than male life expectancy at all ages, reflecting their lower level of mortality. Similarly, all female health expectancies are higher than the corresponding male health expectancies at all ages, although the differentials vary and are in all cases narrower than that for life expectancy. Thus females not only live longer than males at all ages, but also live longer both in healthy state and paradoxically with some form of disability. This point is illustrated by the health expectancy to life expectancy ratios. At all ages and for all disability-based indices, these ratios are lower for females than for males.

An analysis of the causal structure of the disability-based health expectancies was attempted by the author in a commissioned report to the Ministry of Health (Cheung, 1999b). However, the 1996/97 Disability Surveys were not completely successful in collecting data on self reported cause of disability since survey respondents were generally not able to attribute their functional limitations to specific disease causes, making it difficult to translate from disability to disease or injury at an International Code

of Diseases (ICD) chapter level (Martin Tobias, personal correspondence). As the results lack validity in a form that can be statistically confirmed they are not reported below.

It would be of interest to assess non-Maori's current standing in terms of disability prevalence and disability-based health expectancies relative to other populations from developed countries with whom non-Maori share comparable levels of mortality. However, despite intensive efforts by an international research group (for example, Réseau Espérance de Vie en Santé (REVES)) to develop methods at an international level and to standardise surveys, differences still persist between national surveys for disability measures and in definition of the variables collected, making direct international comparisons difficult (Romieu and Robine 1997). In addition to methodological differences (for example, study design, data definitions), cultural and socio-economic differences in the meaning of, and the willingness of respondents to report functional limitations also inhibits international comparisons.

A classification system of international health expectancies developed by Romieu and Robine (1997) using the WHO International Classification of Impairments, Disabilities and Handicaps (ICIDH) as a framework has shown a considerable range of variations between countries within each category. Meaningful comparisons between countries is further complicated by the different time periods for which health expectancies were calculated for different countries. Under the classification system developed by Romieu and Robine, ILEs reported here would be included in a category of health expectancies which they called "functional limitation-free life expectancy". Also included in this category are Australia (1993), with life/health expectancy at birth 75.0/58.4 years and 80.9/64.2 years for males and females respectively; and Netherlands (1986-88), with life/health expectancy at birth 73.5/64.1 years and 79.9/65.1 years for males and females respectively. Non-Maori health expectancies as calculated in 1996/97 seem to compare relatively favourably with these countries. However, since many of the methodological and related differences in compiling health expectancies in different countries cannot be adequately accounted for, further comparisons of non-Maori experience and that internationally is not justified at present (Romieu and Robine 1997).

9.1.3 Analysis of Survival and Disability Curves

Figures 9.2a and 9.2b are provided below to show the survival curves, and are disaggregated respectively into four components: no disability and disability of severity levels one to three. The disability curves, the l_x function for disability in the health expectancy life table calculations, were estimated indirectly from the L_x function for disability which was estimated directly using observed population prevalence rates (Sullivan, 1971). The indirectly estimated disability l_x curves do not necessarily decrease monotonously. These curves can be interpreted as movements between disability states by individuals as they age. For example, increases in level one disability curves at childhood can be understood as children recovering from mild disabilities.

The curve for DFLE, dividing between states of no disability and minor (level one) disability, resembles almost the classical horizontal "s" survival curve as one would find with a high mortality population. Thus the initial drop in the male DFLE curve is so large that it creates a space between it and the survival curve which is maintained until around age 30 years[2]. Interpreting this in the context of a hypothetical cohort, male disability in the first 30 years of life would be expected as mostly originating at infancy. The initial drop in the female DFLE is less significant than that for males.

For both genders, the exponential decline in the DFLE curve starts much younger at around age 30 years, some 30 years earlier than that in survival curve, though at a much less rapid pace. The DFLE curve starts to converge with other curves at around age 80 years, but tapers off to zero between ages 90 and 95 years, which is five to 10 years earlier than for the corresponding survival curve.

From around age 30 years, the space between the DFLE and the survival curves for females becomes greater than that for males. Thus gives females a longer expectation of life in states both with and without disability.

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[2] The initial drop may be partly an artefact of the actuarial graduation process, since unexpectedly high rates at ages 10-14 tend to exert upward pressure on the smoothed rates of the adjacent age groups. Analysis of the variance in the estimated disability curve (see Appendix Four) using the relative sampling errors constructed by Statistics New Zealand (1998) lends some support to the validity of the estimations. Nevertheless, the estimations need to be interpreted with caution.

Figure 9.2a Survival and disability curves of life and health expectancies, non-Maori males, 1996/97

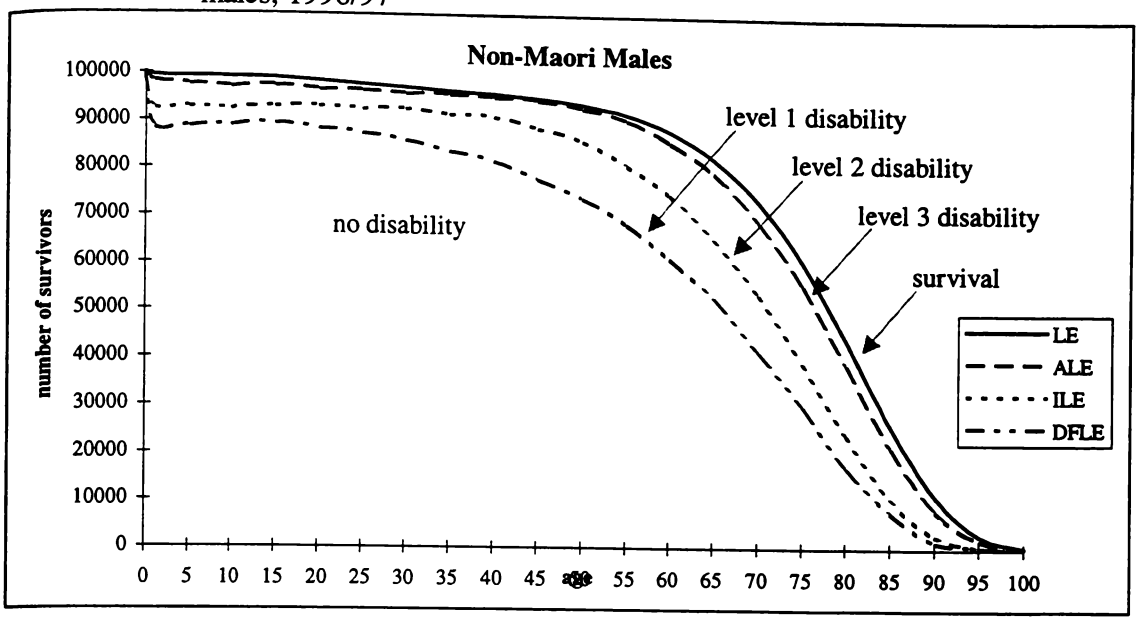
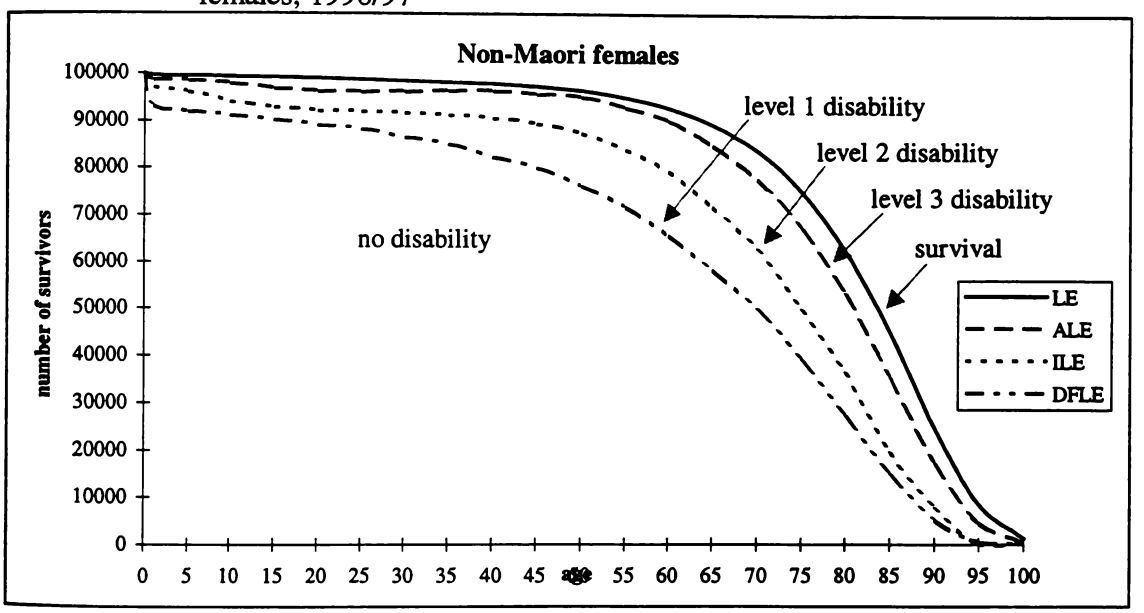


Figure 9.2b Survival and disability curves of life and health expectancies, non-Maori females, 1996/97



Source: Calculated based on the 1996/97 Disability Surveys and life tables constructed by Statistics New Zealand

The initial drop for the ILE curve is less marked than that for the DFLE curve. Minor fluctuations in the female ILE curve also occurred at younger ages. In the adult age ranges, however, the ILE curve for both genders almost mirrors the corresponding survival curve. The timing and the pace of exponential declines in the ILE curve are similar to those for the survival curve, thus producing almost a parallel shift. The substantial distance maintained between the ILE and the survival curves throughout adult life means that the proportion of the remaining years of life spent in a state with disability

requiring assistance increases with age, as the area under the ILE curve reduces faster than the area under the survival curve. This is in accordance with the declining ratio of ILE to life expectancy over age, shown in Table 9.1. In other words, while the impact of disability requiring assistance is felt at every age from birth onwards, it intensifies at the middle to older ages.

At older ages, the ILE curve converges with the DFLE curve and both taper off to zero at ages 90 to 95 years. In contrast, the distance between the ILE and the survival curves is maintained at the upper age ranges. Influences of mild disability quickly diminish at older ages and disability requiring assistance becomes a dominate force in determining the health status of older populations.

The ALE curve for both genders generally moves in tandem with the survival curve. At every age, the most severe form of disability is restricted to a minority of the population except at the very advanced ages. Not surprisingly, this points to a strong correlation between the risk of severe disability and the risk of mortality.

It is of great interest, however, that for both genders, particularly for males, the ALE and the survival curves tend to converge at ages 40 to 50 years. The space between the two curves at younger ages before 40 years is likely to be attributable to severe disabilities due to congenital malformations and other defects; while the space after age 50 years corresponds to the onset of degenerative diseases. The convergence at ages 40 and 50 years thus may be inferred as deaths of people with severe disabilities originating at birth and infancy. Unfortunately, the cross-sectional data set and inadequate information on causes of disability do not permit further investigation at this stage of this potentially very important issue.

In summary, for all levels of severity, disability originating at birth and infancy is largely responsible for the differentiation of the area under the survival curve before the age of 30 years. From then on, however, the risks of disability, mainly of a mild form, accelerate while mortality risks remain stable until late middle age. The relative impact of mild disability is the greatest at young to middle adult ages, and moderate disability is the

more dominant force at middle adult ages onward. In comparison, the impact of severe disability is less dominant than other levels except at the most advanced ages.

Patterns in functional limitations analysed above will be contrasted in the next section with patterns in hospital utilisation. As will be shown later in the chapter, characteristics demonstrated by the disability-based health expectancies, particularly the ALE, closely resemble those for the hospitalisation expectancy, and thus lend support to the validity of both sets of measures.

9.2 Patterns and Trends of Hospitalisation of Non-Maori

So far this chapter has looked at the one cross-sectional data set of morbidity. Now an attempt will be made to extend the analysis both to causes (as against functional limitations) and to take this back in time.

This section presents results of an analysis of public hospital admission and discharge data from the National Minimum Data Set (NMDS)[3]. Hospital discharge data include deaths in the hospital. The relevant data were obtained from published sources by the New Zealand Health Information Services of the Ministry of Health. Data on admission and discharges from private hospitals were less complete and generally not available in a format suitable for this analysis[4].

Information on hospitalisations from the NMDS constitute the most comprehensive centrally collected and collated morbidity data set on a national level. The quality of the various aspects of this data set and its limitations for time series analysis have been discussed in Chapter Seven. Hospitalisation represents the more extreme form of ill-health, and should be considered as such in gauging the general health status of the population. Hospital utilisation is influenced by an array of factors, including changes in access to hospital care, hospital admission and discharge policies and the effects of health

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3 Admissions and discharges for obstetrical purposes plus categories such as admissions for observations/test and the discharge with no further interventions are excluded from this analysis.

4 Private hospital admissions and discharges consists a small portion of the NMDS, and were generally for non-acute conditions.

care technology in general (for example, a change from invasive to micro-surgery, and from dependence on surgery and thus hospital admission to control using chemotherapies on an out-patient basis). The latter determine indirectly affects patterns of referral to hospital.

In one sense this can be seen as a substantive and even methodological limitation. But in another sense this is a measurement of reality: over time improved medical technology may reduce the risks of disability which limit daily living of the sufferers.

There is another problem. This thesis is on non-Maori, but historically the classification of ethnicity in the NMDS was poor in terms of accuracy and consistency, though considerable improvements were made in the more recent years (Johnstone et al, 1998, see also Chapter Seven on data sources and quality). Problems in the time series analysis are also compounded by the fact that the format for data published prior to 1990 sub-divided by gender, age group and ICD chapters are available only for the total population, but not for major ethnic groups. Thus analysis of non-Maori below is restricted to the 1990s. The formats of age groupings in the published data are also inconsistent over time, making age specific comparisons difficult.

9.2.1 Current Patterns of Hospitalisation in Relation to Mortality

This section presents patterns of hospital admissions and discharges of non-Maori for the three years around 1996. References are also made to the corresponding patterns for the total population of New Zealand (both Maori and non-Maori combined). By turning, however, to the total population one can compute what is almost a proxy for a non-Maori time series in hospitalisation[5]. This is because discharges for non-Maori accounted for the bulk of the overall hospitalisations, at over 85 percent in 1995-97, roughly about the proportion non-Maori are in the population. Moreover, the proportion of Maori has grown rapidly over recent decades, from six percent in 1951 to 14 percent in 1996.

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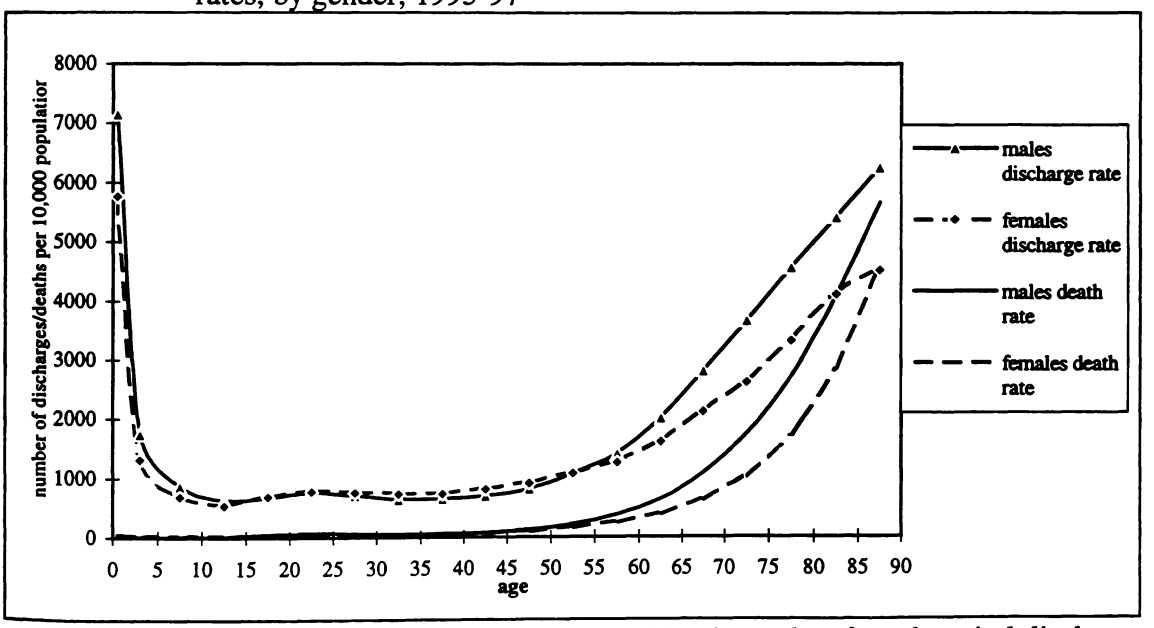
[5] Comparisons of total and non-Maori disability-based health expectancies show only narrow differences between the two sets (Cheung and Tobias, 1998). For example, ILE of the total population of 64.5 years for males and 67.9 years for females is roughly one year less than the corresponding non-Maori figures (65.6 years and 68.9 years respectively).

Between 1995 and 1997, on average, there are an annual number of 203,229 public hospital discharges for males and 196,571 discharges for females (excluding pregnancy and childbirth) who have their ethnicity coded as non-Maori. The corresponding figures in the total population including both Maori and non-Maori are 233,598 for males and 226,004 for females.

The non-Maori age standardised hospital discharge rates for all causes in 1996 are 1,401 per 10,000 for males and 1,205 per 10,000 for females. These rates are very similar to that for the total population (1,405 per 10,000 for males and 1,220 per 10,000 for females) due to the overwhelming dominance of non-Maori in the total population figure.

As is the case for mortality, the risk of hospitalisation is highly age selective. In Figure 9.3, non-Maori age specific hospital discharge rates in 1996 are overlaid with the corresponding age specific death rates (expressed as per 10,000 to be comparable with discharge rates).

Figure 9.3 Non-Maori age specific hospital discharge rates and age specific death rates, by gender, 1995-97



Sources: Hospital discharges rates: computed by the author from hospital discharge data published by Ministry of Health and Census population data published by Statistics New Zealand
Death rates: extracted from the complete life tables constructed by Statistics New Zealand

The pattern of age specific hospital discharge rates follows the classical "U" shaped age specific mortality curve but closer to one for a high mortality population. Thus the rate of hospitalisation is highest at birth while the risks of mortality are at almost a minimum beyond the neonatal period (when anyway levels are so low, relatively, that they barely show up on the graph). The risks of hospitalisation, also however, reduce dramatically after birth to reach the lowest point at ages 10 to 15 years. The small hump for males at young adult ages reflects accident related injuries.

At around age 45 years for males and age 50 for females, the risks of hospitalisation start to increase exponentially with age. For both genders, the acceleration in the risk of hospitalisation from around age 60 years coincides with an exponential acceleration in the risk of death. Compared to the age specific mortality curve, the pace of increase in the hospitalisation rates by age is slower but starts at a higher level. The two curves tend to converge at older ages. At the more advanced ages a sign of slowing down in female exponential increases of hospitalisation rate can be detected.

9.2.2 Current Patterns of Cause of Hospitalisation

Public hospital discharge data from the NMDS are published by the principal causes of hospitalisation. Data on associated causes are generally not available from these published sources. Causes of hospitalisation in 1995-97 are analysed using a modified grouping system as that used in the cause of death analysis in Chapter Eight. Discharge data are grouped into six cause groups: i) infectious and respiratory diseases, ii) cancers, iii) cardiovascular diseases, iv) accidents, v) diseases of the digestive system, vi) diseases of the genitourinary system, and vii) other causes. Discharges for infectious diseases are combined with those for respiratory diseases because of the small numbers for the former. Two additional groups are added: digestive and genitourinary diseases. Table 9.2 summarises for 1996 cause specific age standardised hospital discharge rates, and each category is also shown as a percentage of all causes. Patterns in age specific rates for the first six major causes are presented in Figures 9.4a and 9.4b.

Table 9.2 Non-Maori age standardised hospital discharge rates (per 10,000) by major causes of hospitalisation and as a percentage of all causes, non-Maori population, by gender, 1995-97

Causes of hospitalisation	Males		Females	
	Rate (per 10,000)	Percentage	Rate (per 10,000)	Percentage
Infectious and respiratory diseases	182	13%	152	13%
Cancers	124	9%	111	9%
Cardiovascular diseases	221	16%	136	11%
Accidents	222	16%	145	12%
Digestive diseases	153	11%	133	11%
Genitourinary diseases	70	5%	123	10%
Others	429	31%	405	34%
Total	1401	100%	1205	100%

Note: "others" include other chronic diseases (ICD chapters 3-6, 12 and 13), infant conditions (ICD chapters 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Computed by the author from hospital discharge data published by Ministry of Health and Census population data published by Statistics New Zealand

The distribution of causes of hospitalisation is significantly less compressed than are patterns of causes of mortality (see Table 8.10). Moreover, the combined infectious and respiratory diseases group is the third leading cause of hospitalisation for males and the leading cause for females. Discharges for respiratory diseases account for 80 percent of the combined sum of the two causes. Cardiovascular disease is the leading cause of death, but has ceded its top position for hospitalisations to accidents when both genders combined. More interestingly, cancers, being a second leading causes of death among non-Maori, account for only a small portion of the total hospital discharges, and are only the sixth leading cause for males and seventh for females. Discharges for digestive diseases and diseases of urinary tract and female reproductive systems are more prevalent than cancers, but are far less likely to be life-threatening.

Accidents constitute the leading cause of hospitalisation for both genders at most ages. The risk of hospitalisation because of an accident among females is concentrated at older ages when accidental falls resulting in hospitalisations are more prevalent. The acceleration in female accident hospitalisation rates at ages 70 years and over is most noticeable. For males, accident hospitalisation rates are wider spread over the age groups, due to the relatively high rates at young adult ages, though male accident hospitalisation rates are still higher at older ages than at younger ages.

Figure 9.4a Patterns of age specific hospital discharge rates by major causes of hospitalisation, non-Maori males, 1995-97

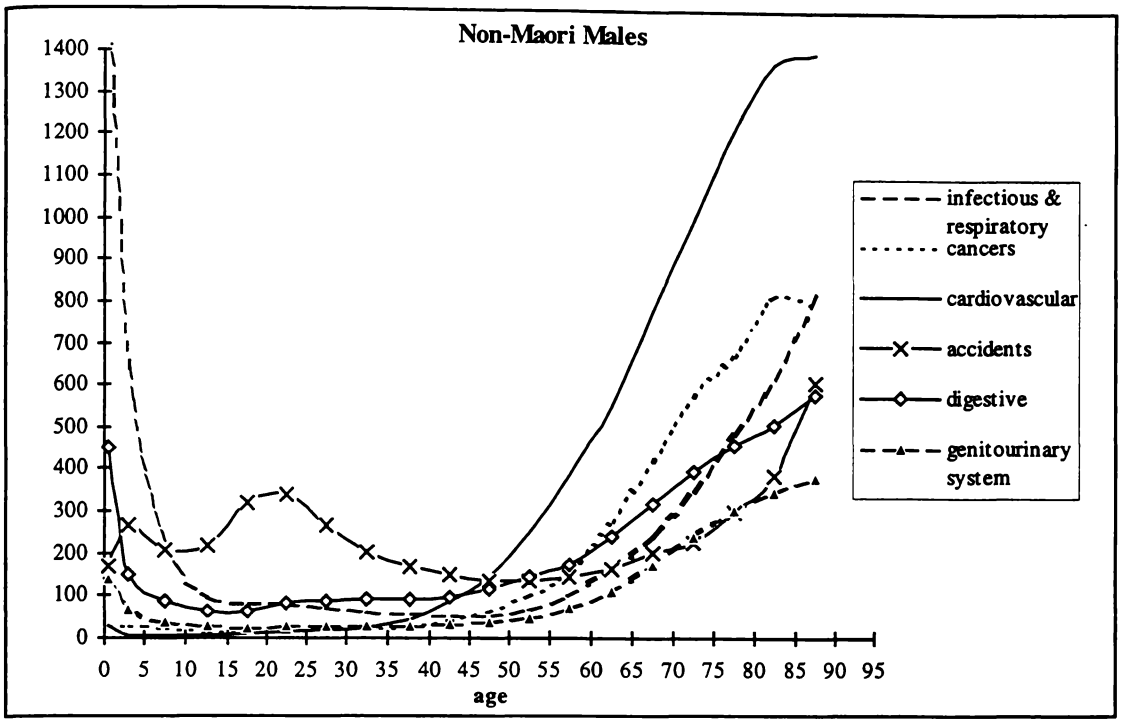
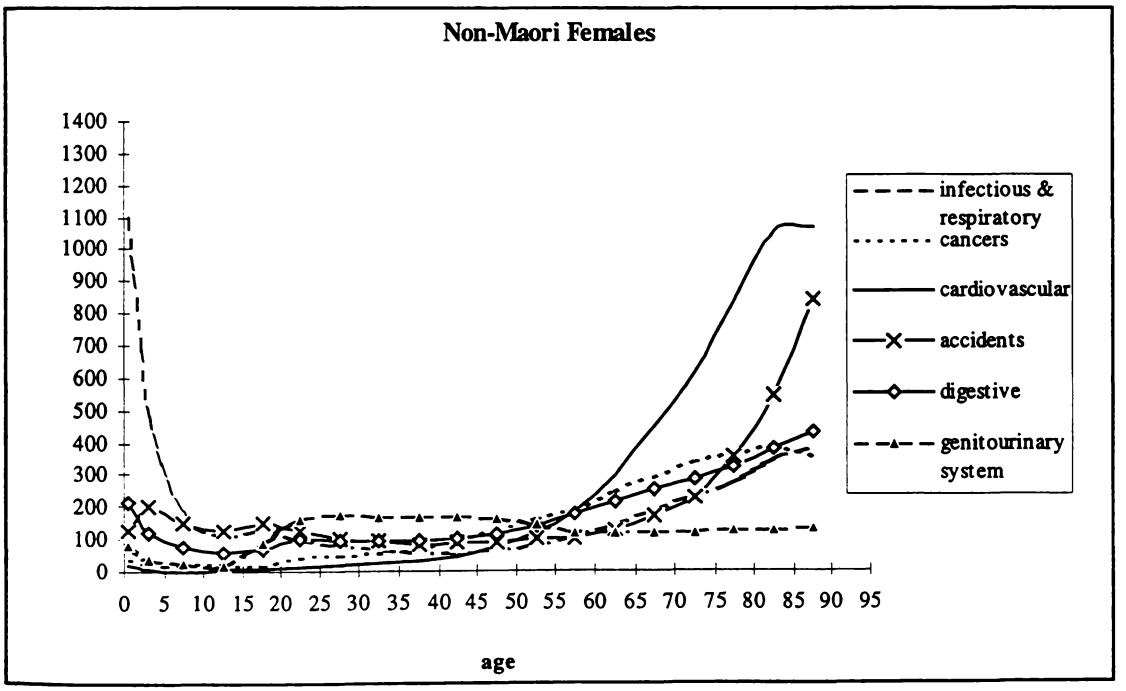


Figure 9.4b Patterns of age specific hospital discharge rates by major causes of hospitalisation, non-Maori females, 1995-97



Sources: Computed by the author from hospital discharge data published by the Ministry of Health and Census population data published by Statistics New Zealand

At late adult ages, cardiovascular disease is clearly the leading cause of hospitalisation. The accelerated increase in male age specific rates from age 40 years onward for both cardiovascular diseases and cancers features prominently in their cause pattern. In contrast, the corresponding acceleration in the female cardiovascular hospitalisation rates at older ages is at a less rapid pace. Increases in female cancer age specific hospitalisation rates is more gradual and follows a linear instead of an exponential path. It is worth noting that for both genders, age specific rates of hospitalisation for cancer and cardiovascular disease tend to level out or even decline at ages 80 years and over. This may be explained by increasing risks of other competing causes of hospitalisation, and/or that cancer and cardiovascular disease are an associated cause of hospitalisation.

Examples of competing risk of hospitalisation at these older ages included respiratory and digestive diseases. Compared with the corresponding patterns in cancer and cardiovascular disease, for both genders age specific hospitalisation rates for infectious and respiratory diseases and digestive diseases demonstrate no sign of slowing down at ages 80 years and over. Respiratory disease is also the most dominant causes of hospitalisation at infancy and early childhood.

The risk of hospitalisation for diseases of reproductive system among females is relatively high at reproductive ages, and is the leading cause of hospitalisation at these ages. The risk reduces gradually towards the end of the reproductive ages and stabilises after age 60 years. Males follow an opposite pattern with minimal risk of hospitalisation for diseases of the genitourinary system at early adult ages, but the risk increases steadily after age 55 years.

9.2.3 Current Patterns of Hospitalisation Expectancy

Since hospitalisation data mostly capture extreme forms of ill-health, it is not too surprising that the risk of hospitalisation shows a strong correlation with the risk of dying from the middle adult ages when most deaths occur. To examine better patterns of non-Maori hospitalisation in relation to the level of mortality, in the rest of this sub-section an experimental life table based analysis similar to the health expectancy technique is carried

out. Hospitalisation expectancy measures the expected number of hospital days that will occur while still surviving by combining the incidence (the discharge rate) and the duration (the average length of stay) of hospitalisation with levels of mortality (see Section 7.2.4). As will be shown below, for the general population hospital stays are expected to be merely a fraction of life expectancy, even at the very old ages (see Liang et al, 1996). The focus of hospitalisation expectancy is therefore on the period of time in an elevated level of ill-health and is measured by days instead of years.

Life table techniques are applied by researchers in studying the propensity of institutionalisation and use of health services (Liang et al, 1996; Manton et al, 1984). The application of health expectancy methodologies in studying patterns of hospital utilisation were few[6]. The analysis of hospitalisation expectancy is therefore highly experimental. A major reason for caution is the reliance on data on the average length of stay by age groups. Length of hospital stays are subjected to administrative manipulations to an even greater extent than admissions. Systematic declines in the average length of stay at every five year age group are clearly noticeable even over a short period of three years between 1995 and 1997. This systematic biasness over time would certainly affect time series comparisons in the next section.

The construction of the abridged hospitalisation expectancy life tables are explained in Appendix Three. Hospitalisation expectancies by five-year age intervals are presented in Figure 9.5, the corresponding numerical figures are provided in Appendix Five. As noted above, hospitalisations can be expected to occupy a small portion of future life expectancy at all ages. In 1996, hospitalisation expectancy at birth is 63 days for males and 69 days for females. These numbers represent just under a quarter of one percent of their respective life expectancy at birth. Higher hospitalisation expectancies for females than for males are seen at younger ages, but gradually disappear at late adult ages. At age 90 years, the expected number of hospital days for males of just under 23 days exceeds that for females of 21 days.

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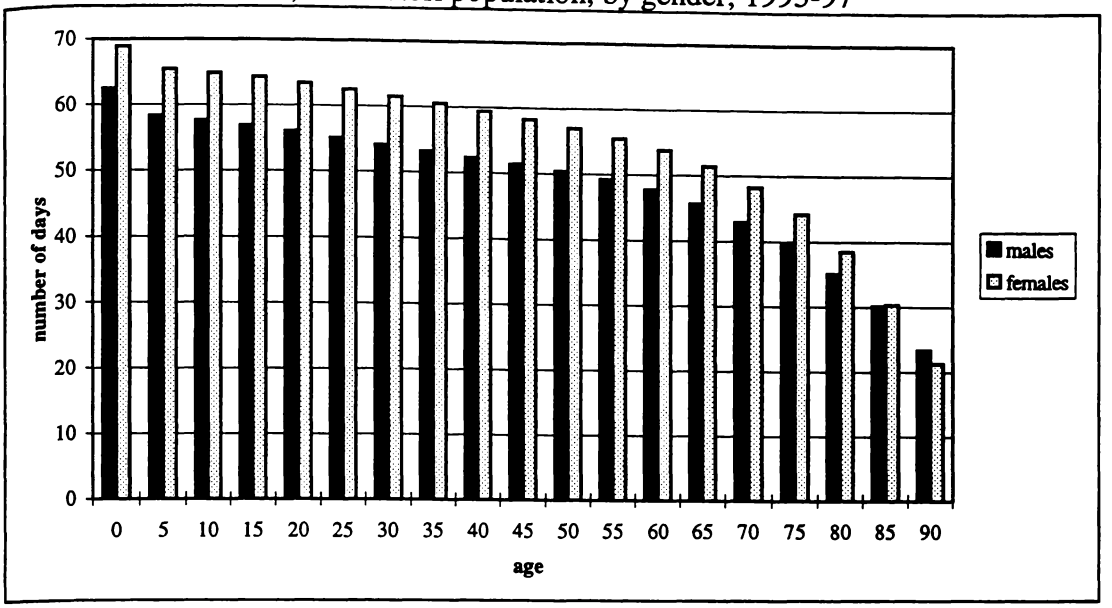
[6] For example, see Liang et al (1996) for a multi-state life table modelling of transitions between community, hospital stay and nursing home use.

Figure 9.5 Expected number of future days of hospitalisation by five year age interval, non-Maori population, by gender, 1995-97



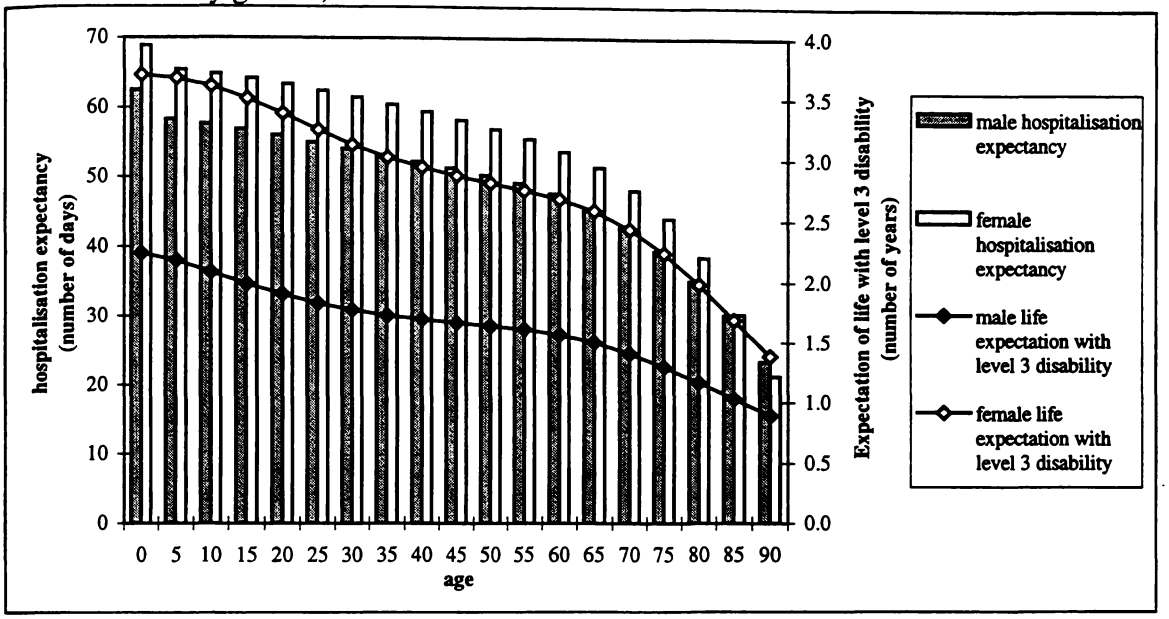
Sources: Calculated by the author based on public hospital discharge data and data on average length of stay published by the Ministry of Health, and complete life tables constructed by Statistics New Zealand

From the time of birth, hospitalisation expectancy reduces systematically with age. Most interestingly, however, the future number of days in which can be expected to be in contact with public hospitals declines exponentially with age after reaching the end of the reproductive ages. The age at which this exponential decline occurs is inversely correlated with exponential increases in both the risk of hospitalisation and the risk of death (see Figure 9.3). The explanation lies, of course, with corresponding mortality patterns. The accelerated risk of hospitalisation at late adult ages is strongly correlated with an acceleration in risk of mortality, and the combined impacts of these at a population level, when measured as future hospital stays, tend to off-set each other. This is the most likely explanation at older ages of 70 years and over when the largest reductions in hospitalisation expectancy over age coincides with the largest increases in age specific death rates.

A link to disability-based health expectancies analysed in Section 9.1 can be made. Being hospitalised involves a high degree of functional limitations and continuous care. Thus hospitalisation expectancy resembles the more extreme level of disability-based health expectancies, that is, Active Life Expectancy (ALE). To illustrate this point, in Figure 9.6 non-Maori hospitalisation expectancy in 1995-97, as presented in Figure 9.5, is overlaid with the reciprocal of ALE, measuring expectation of life with level three

disability. The indices are, of course, measured differently: days versus years. The two sets of indices are congruent in terms of both their characteristic shapes and the gradient of the curves.

Figure 9.6 Hospitalisation expectancy and reciprocal of ALE, non-Maori population, by gender, 1995-97



Sources: Calculated by the author based on the 1996/97 Disability Surveys, public hospital discharge data and data on average length of stay published by the Ministry of Health, and complete life tables constructed by Statistics New Zealand

In general, the findings of hospitalisation expectancy are consistent with the pattern recorded for ALE and its reciprocal, expectation of life with level three disability. The probability of developing morbid conditions requiring hospitalisation and/or continuous assistance is small at most ages. When measured at a population level, time expected to be spent in states of more severe forms of ill health is restricted to a fraction of future life expectancy.

So far this analysis has been cross-sectional. Using the same methodologies is now taken back over recent decades.

9.2.4 Recent Trends in Hospitalisation in Relation to Mortality

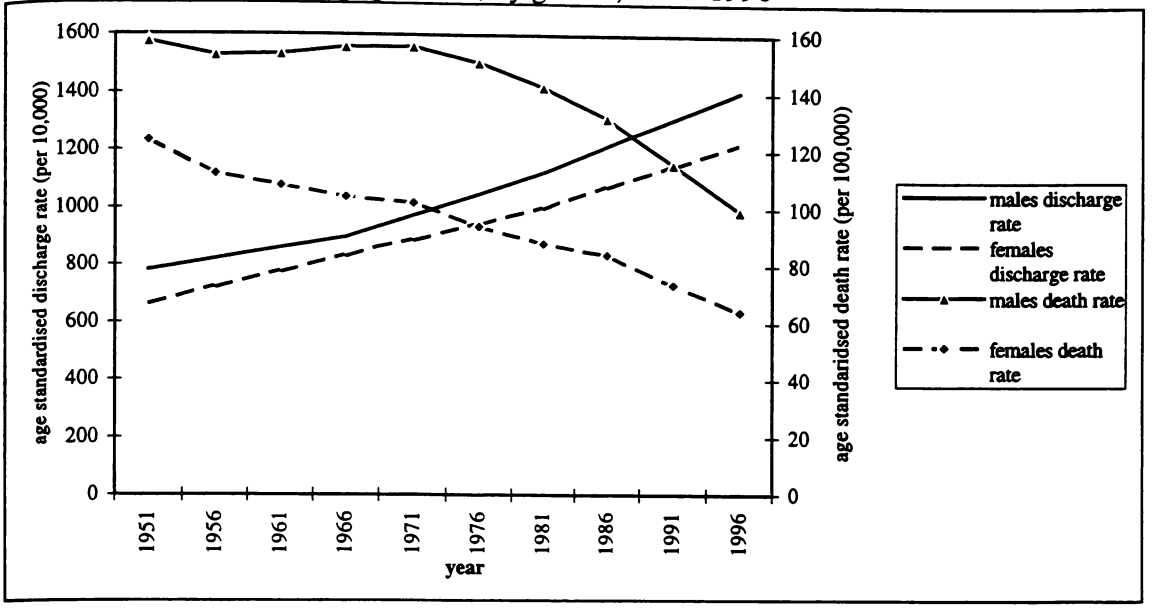
Hospital admission and discharge data have some serious weaknesses for longer term studies. The limitations imposed by this data set in time series analysis are outlined earlier in the section and also discussed in Chapter Seven. Three changes in the coding of causes of hospitalisation occurred during this period, from the ICD (International Code of Diseases) version six to ICD version nine.

Moreover, data prior to 1990 were published only for the total population, but not by ethnic group. The following analysis is on the recent trends in hospitalisation of the total population. As suggested in the previous section, non-Maori hospital discharge rates are very similar to those for the total population because of the pre-dominance of non-Maori in the total population. Trends in hospitalisation of the total population can therefore be regarded as an approximation of those for non-Maori.

Between 1951 and 1996, the hospitalisation rate increased steadily despite accelerated declines in mortality rates. The increase was observed at most age groups and for all causes. Trends between 1951 and 1996 in hospital discharge rates and death rates, both of which are age standardised to the combined non-Maori male and female populations in 1996, are provided in Figure 9.7. An almost linear increase in the age standardised discharge rates emerged. The increases between 1966 and 1996 were slightly more rapid than those in earlier years, and were in direct contrast to the downward trends in death rates.

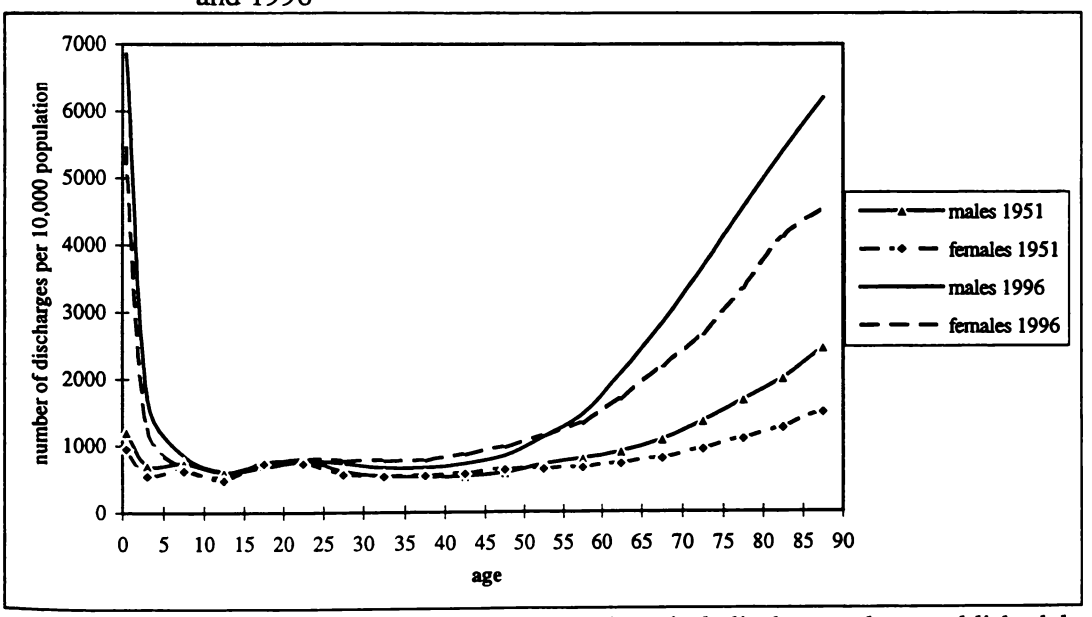
The great majority of these increases occurred at the youngest and the oldest ages. Figure 9.8 presents age specific discharges rates between 1951 and 1996, and is supplemented by Figure 9.9 which shows the changes by cause groups during the period.

Figure 9.7 Age standardised hospital discharge rates and age standardised death rates, total population, by gender, 1951-1996



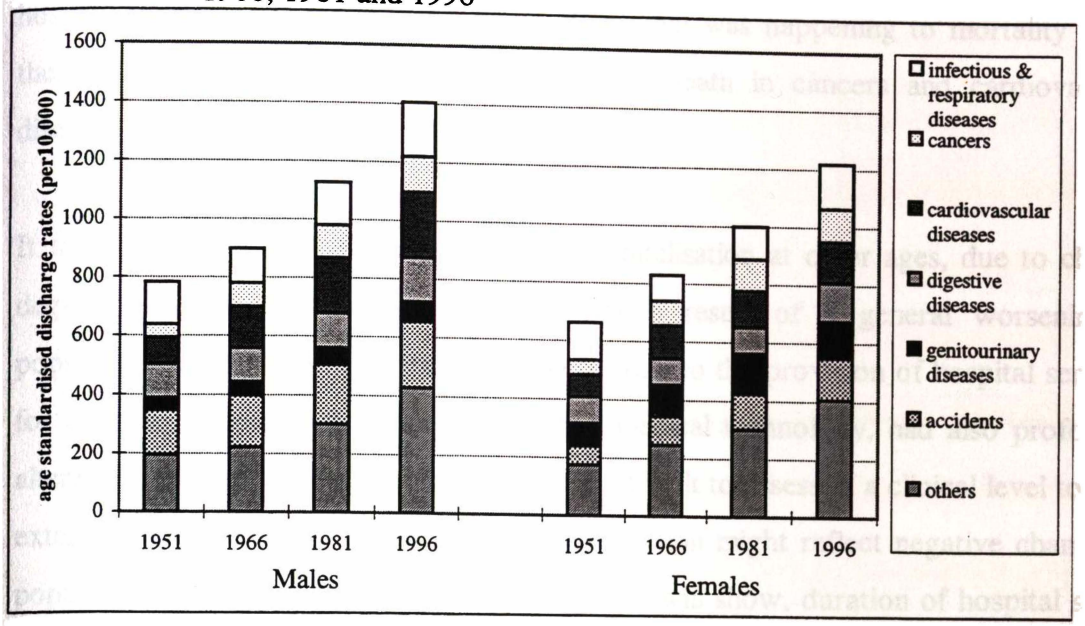
Sources: Computed by the author from hospital discharge data published by the Ministry of Health, vital statistics and population data published by Statistics New Zealand

Figure 9.8 Age specific hospital discharge rates, total population, by gender, 1951 and 1996



Sources: Computed by the author from hospital discharge data published by the Ministry of Health, vital statistics and population data published by Statistics New Zealand

Figure 9.9 Cause specific hospital discharge rates, total population, by gender, 1951, 1966, 1981 and 1996



Note: "others" include other chronic diseases (ICD chapters 3-6, 12 and 13), infant conditions (ICD chapters 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Computed by the author from hospital discharge data published by the Ministry of Health, vital statistics and population data published by Statistics New Zealand

The sharp rises in the infant and early childhood hospital discharge rates reflect significant medical technological advances and changes in hospital service provisions for neonates during the period. For example, the infant discharge rate for conditions originating in the perinatal period increased by nearly 10 times between 1951 and 1996, coinciding with rapid developments in perinatal care and continuing declines in the infant mortality rate (see also Sceats and Pool, 1985). Sharp rises in number of young children treated at hospitals with respiratory conditions accounted for a third of the large increases in age specific discharge rate. Increases in infectious diseases, digestive diseases and ill-defined conditions of early infancy also contributed significantly to the increases in the hospital discharge rates at early childhood.

Increases in the hospital discharge rates at middle to older adult ages were nearly as dramatic as those at infancy. General increases in discharges for chronic degenerative diseases, especially cardiovascular diseases, were responsible for this sharp rise. The increased number of discharges for cancers, respiratory diseases, digestive diseases, accidents and disorders of the musculoskeletal system (including arthritis) were the other major contributors. The general increase across most cause groups reinforced the

historical diversity in the distribution of causes of hospitalisation (see Figure 9.9). This hospitalisation trend was in direct contrast to what was happening to mortality where there was a heavy concentration of causes of death in cancers and cardiovascular diseases.

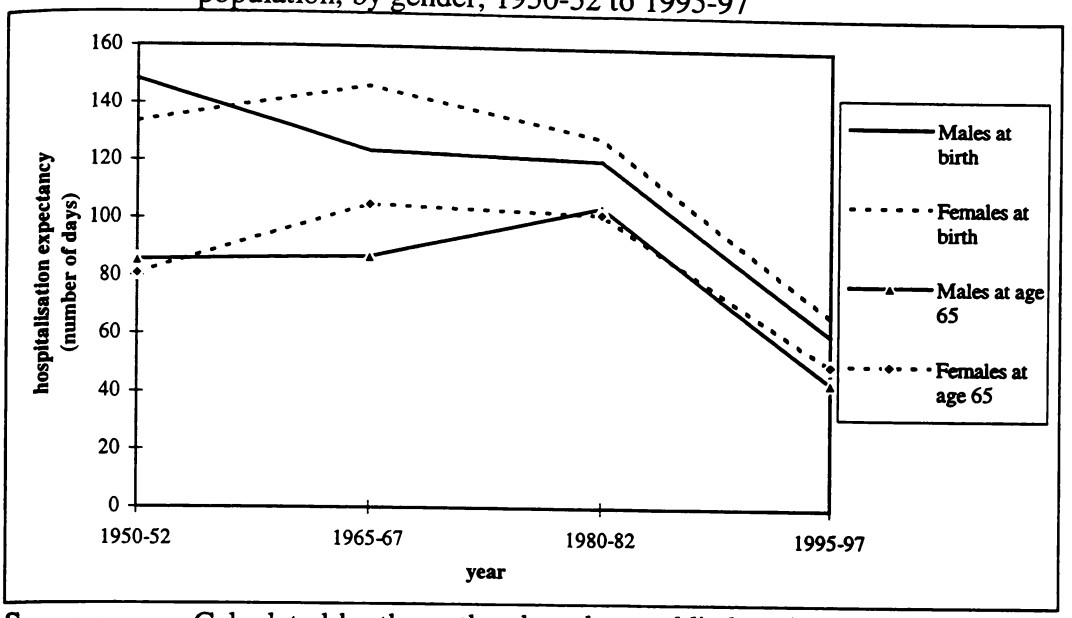
It might be argued that the sharp rises in hospitalisation at older ages, due to chronic degenerative diseases, mostly non-fatal, was a result of a general worsening of population health status. However, factors relating to the provision of hospital services, for example, access to hospital services and medical technology, had also profoundly altered the pattern of hospital utilisation. It is difficult to assess at a clinical level to what extent the increases in the rate of hospital utilisation might reflect negative changes in population health status. As the next sub-section will show, duration of hospital stay is also a factor.

9.2.5 Trends in Hospitalisation Expectancy

Historical hospitalisation expectancy analysis is affected by systematic changes in hospital stay as noted earlier. That said, data on the average length of stay for earlier years were particularly problematic and were generally not in very useful formats. Broader age bands of 0-4, 5-14, 15-44, 45-64 and 65+ were reported in the published sources and have had to be used in the following analysis instead of the more finely calibrated five year age groups. Gender specific average length of stay by broader age bands were not available for several years in the early 1980s and thus data for both genders combined have had to be used assuming identical patterns of length of stay for both males and females in those years. Length of stay data were not published prior to 1960, and are estimated in the following analysis by extrapolating back from the patterns of later years.

Four data points are selected: 1950-52, 1965-67, 1980-82 and 1995-97, which give three equal 15 year intervals. The results of hospitalisation expectancy calculations at birth and at age 65 years are presented in Figure 9.10. The results for the earlier years are less reliable for reasons outlined above and should therefore be regarded as indicative only.

Figure 9.10 Trends in hospitalisation expectancy at birth and at age 65 years, total population, by gender, 1950-52 to 1995-97



Sources: Calculated by the author based on public hospital discharge data and data on average length of stay published by the Ministry of Health, and complete life tables constructed by Statistics New Zealand

Figure 9.10 is dominated by the steep downward slope between 1980-82 and 1995-97, which gives the impression of an overall decreasing trend of hospitalisation expectancy. Trends between 1950-52 and 1965-67 are less reliable because of the need for indirect estimations of the average length of stay for 1950-52.

Between 1965-67 and 1980-82, hospitalisation expectancy seemed to have stabilised. Increases in the number of discharges were offset by reductions in the average length of stay at all age bands, especially at ages less than five and ages 45 years and over. The increase in male hospitalisation expectancy at age 65 years is likely to be an over-estimation of the average length of stay. At older adult ages males tend to have shorter hospital stays than females, and thus the combined average length of stay data for both genders, which had to be used around 1981, would almost certainly over-estimate values for males. The opposite effect would then explain for the reductions in female hospitalisation expectancy over the 15 year period.

Despite doubts about the 1980-82 estimations, reductions in hospitalisation expectancy between 1980-82 and 1995-97 are indisputable. These sharp declines were entirely driven by reductions in the average length of stay, cutting duration by half or more at all age bands. The most dramatic reductions in the average length of stay occurred at ages

65 years and over, which also had the largest increases in the hospitalisation rates. To a considerable extent the systematic declines in hospital stay reflect the effect of government's push away from hospital based health service delivery.

The recent downward trends of hospitalisation expectancy was in direct contrast to the steady increases in the hospital discharge rates as is shown in Figure 9.7. This contrasting pattern suggests that the increased incidence of hospital contacts was more than off-set by much shorter durations for such contacts. After combining both the incidence and the duration dimensions and adjusting for the level of mortality, it is shown that the resulting level of hospital utilisation had reduced significantly over the last 15 year period. The time expected to be spent in contact with hospital services has thus been increasingly compressed into a smaller range.

What makes the declines in hospitalisation expectancy of even greater significance is that these declines occurred in an environment of declining mortality at the older ages. It gives the strongest empirical support yet of compression of the most severe forms of morbidity, using data on hospitalisation as the proxy. It can thus be reasonably concluded that improved survivorship at older ages in the last 15 years were not realised at the expense of prolonged hospital stay, and thus a decreased quality of life for the sick elderly[7].

9.3 Discussion

The analysis of morbidity patterns and trends faces a general lack of adequate data sets. The multi-dimensionality of morbidity demands analyses which are data hungry exercises, yet cross-sectional data are seldom sufficient to cover the key dimensions of interest, and are even more problematic for analysing changes over time. Analyses are often restricted to exploratory and experimental studies, exactly the approach followed in this chapter.

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[7] This conclusion is consistent with that arrived at by Henderson et al (1990) from British data. These authors analysed, using mean and medium hospital days and percentages, hospital use in the last year of life among those who died at ages 65 years and over.

An experimental analytical technique of health expectancy, combining the level of mortality encapsulated in the life table and level of ill-health, is the anchor for this chapter. It has been used to present two groups of population health status indices; namely, the more conventional disability-based health expectancy and an innovative technique, hospitalisation expectancy.

The analysis in the chapter has established from the study of disability-based health expectancies that the more severe forms of disability, when measured at a population level, tend to be restricted to a small portion of life. This seems to support arguments in favour of morbidity compression. The results from the cross-sectional and time series analysis of hospitalisation expectancy generally validates this inference, pointing to a compression of morbidity of the most severe form accompanying a trend of improving life expectancy.

An issue of more immediate policy relevance is the diverging patterns and trends of mortality and hospitalisation. Not only has the level of hospital utilisation has moved in the opposite direction to a declining mortality trend, but the pattern of cause of hospitalisation has also diverged from that for mortality. Causes of death are becoming increasingly concentrated around cancers and cardiovascular diseases. In 1995-97, these two major causes account for roughly 70 percent of all deaths, even after controlling for population age structure, and close to 80 percent at ages 65 years and over. Yet these two causes are responsible for less than a quarter of all hospital discharges in 1995-97 after controlling for the age structure, and for 40 percent or less at ages 65 years and over where the risk of hospitalisation increases exponentially with age. Other non-chronic or less lethal causes are the leading causes of hospitalisation. These include accidents, infectious and respiratory diseases, diseases of the digestive system, and, for females, diseases of the genitourinary system.

In short, frail individuals in modern day society may suffer from a range of non-lethal, chronic degenerative diseases, but are less likely to die from these diseases. From a policy perspective, health interventions aiming at reducing deaths may be increasingly less effective in reducing levels of ill-health requiring hospitalisation. The diverging

patterns of causes of mortality and hospitalisation thus seem to provide two different, and potentially conflicting, policy foci. This point will be returned to in Chapter Fifteen.

The present chapter has, however, presented data which give some degree of empirical support to the compression theory, at least in terms of morbidity - mortality interactions for a population at the later stages of an epidemiologic transition. For non-Maori at least for the survival component, one can take this investigation back in time into the last part of the nineteenth century to review these issues in more detail.

The next few chapters will, then, look at trends in synthetic and cohort life tables to analyse changes in survivorship and investigate whether or not compression has occurred over the long-term for mortality. The results will be used as a baseline for the computation of projections in Chapter Fourteen.

CHAPTER TEN
NON-MAORI COHORT MORTALITY TRENDS

One of the key issues identified from the review of non-Maori historical mortality trends in Chapter Eight was the impact of cohort exposure to risks of death early in life on their mortality experiences later in life. Major changes in mortality trends do not happen at one point in time as might be suggested by synthetic life tables. Instead, major mortality changes, as observed in period measures, are the manifestation of the collective exposure to risk of mortality accumulated by different cohorts over different time periods. These changes are therefore likely to be long term processes, and often have their origins far earlier in a cohort's mortality history. This chapter is devoted to the analysis of non-Maori mortality trends by birth cohorts to uncover these antecedents, and to understand their implications for future mortality developments.

For the purpose of this analysis, non-Maori birth cohorts are grouped into three major categories corresponding to thirty-year periods of birth.

1. Those who were born between 1871 and 1901: most of these people have completed their life course, so that cohort life tables covering all ages can be estimated. Cohorts in this group are referred to hereafter as the "complete" birth cohorts.

2. Those who were born between 1901 and 1931: by 1996 these people were at older ages between 65 and 95. Cohorts in this group are referred to hereafter as the "advanced" birth cohorts.

3. Those who were born between 1931 and 1961: half of these cohorts are the post-war baby boomers who are at middle ages in 1996. But the oldest born prior to the war are moving towards older age. Cohorts in this group are referred to hereafter as the "younger generation" birth cohorts.

The groupings are based on life-cycle stages as at 1996. It should not be interpreted as implying cohorts in the same group all share the same mortality exposure. Indeed, the underlying argument of this thesis is to the contrary.

Essentially the same information analysed in Chapter Eight is re-organised to present a birth cohort perspective. Abridged cohort life tables by five year birth interval are constructed based on abridged synthetic life tables analysed in Chapter Eight. Series of abridged cohort life tables for cohorts born between 1871 and 1961 are included in Appendix Seven. Methods for constructing cohort life tables can be found in Appendix Six. For reasons of simplicity, the results of cohort analyses are presented in this chapter by ten year birth intervals. Passing reference is also made to any major changes within the ten year interval. The complete birth cohorts are analysed in greater details, and discussion on the advanced and the younger generation of cohorts in the two later periods is necessarily rather brief.

10.1 Mortality Trends of the Complete Birth Cohorts, Born 1871-1901

10.1.1 Changes in Levels of Mortality

Non-Maori cohorts born between 1871 and 1901 benefited progressively from significant mortality reductions at younger ages. Cohort life expectancies at selected ages are summarised in Table 10.1.

Table 10.1 Life expectancy at selected ages for non-Maori complete birth cohorts, by gender, 1871-1901

	Males			Females		
	1871-1881	1881-1891	1891-1901	1871-1881	1881-1891	1891-1901
At birth	55.5	58.2	60.2	58.9	62.5	65.4
At age 1	61.3	63.6	64.9	63.9	67.1	69.6
At age 15	52.2	53.0	53.8	54.5	56.4	58.4
At age 45	27.6	27.7	28.0	30.1	31.2	32.5
At age 65	12.9	12.9	12.9	14.8	15.7	16.6
At age 85	4.4	4.4	4.8	4.9	5.2	5.9

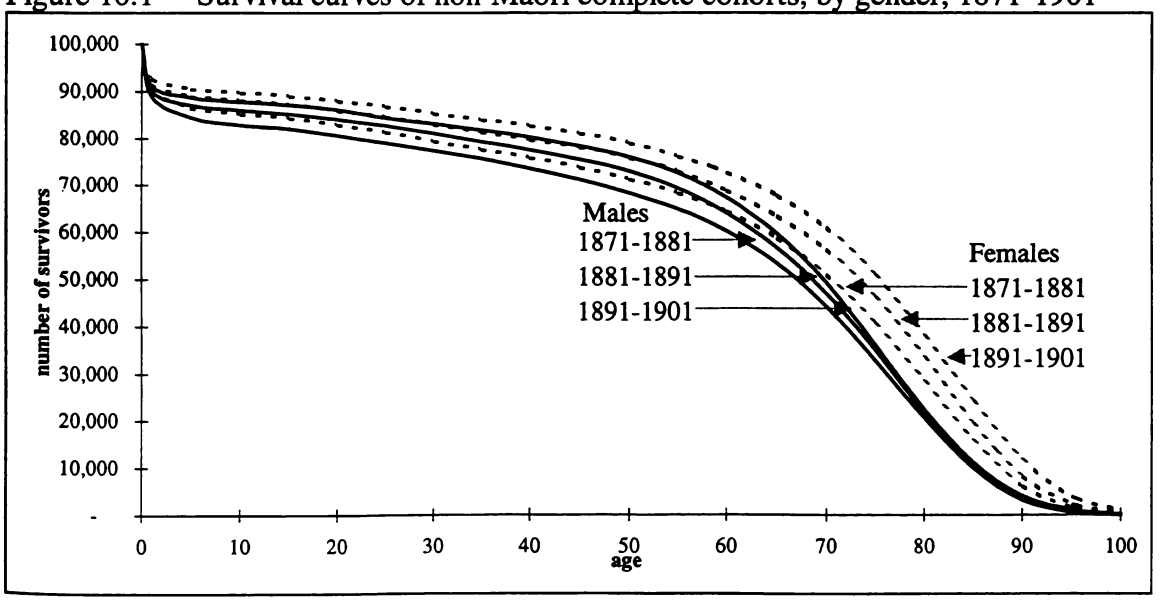
Source: Cohort life tables constructed by the author, based on published and unpublished raw data by Statistics New Zealand

As expected, cohort life expectancy at birth for non-Maori complete cohorts were consistently higher than those from synthetic life tables for the calendar years when these cohorts were born (see Appendix One for life expectancies at selected ages from synthetic life table). These differences reflect the mortality transition in the last century as

reviewed in Chapter Eight. For males the differences between cohort and synthetic life expectancy at birth were generally at around three years, but for females, the difference increased steadily to five and half years for the 1891-1901 complete cohort. As a result, the gender differential in cohort life expectancy at birth for complete cohort born in 1891-1901 was over five years, double the gender differential as measured by the corresponding synthetic life tables.

Male complete cohorts made stronger gains in survivorship at younger ages than their female counterparts. This gains for males, however, seemed to have come at a cost later in their lives. Male cohort life expectancies at older adult ages were levelled between successive cohorts. A deterioration in male cohort survivorship at ages 60 years and over occurred during *the period of conflicting forces of mortality change* and continued on to *the period of stagnation and deteriorations in life expectancy trends* (see Chapter Eight). Males born between 1881 and 1891 were particularly hard hit by these adverse changes. In comparison, non-Maori female complete cohort mortality patterns did not exhibit these characteristics and consequently increases in their life expectancy at birth were greater than their male counterparts. Cohort survival curves are provided in Figure 10.1 to help to further elaborate these points.

Figure 10.1 Survival curves of non-Maori complete cohorts, by gender, 1871-1901



Sources: Cohort life tables constructed by the author, based on published raw data and complete life tables constructed by Statistics New Zealand

All cohort survival curves in Figure 10.1 demonstrated the classical horizontal "s" shape as plotted in Figure 10.3. The high infant mortality rates produced the initial sharp fall. Accelerated declines in survival curves at older ages were much less steeper than those shown in the contemporary synthetic life tables.

Persistent inter-cohort reductions in infant and childhood mortality helped to separate the successive cohort survival curves from the very young ages. The space thus created was further enhanced in the curves for females to mid to late adult ages, and persisted to the most advanced ages. This resulted in a parallel shift of female cohort survival curves, each maintaining an almost equal distance from the previous curve. In contrast, the space between male complete cohort survival curves at younger ages quickly diminished. The three male curves converged at late adulthood at a progressively earlier age.

The following analysis looks at the mortality pattens of each of the three ten-year complete cohorts. Male and female cohorts are discussed separately.

Non-Maori Male Complete Cohorts

Non-Maori males born between 1871 and 1881 benefited considerably from mortality reductions before age 60 years. Over 60 percent of the original cohort survived to age 60 years. Mortality reductions were more significant among the younger members of the cohort born in the late 1870s, though the increases in survivorship of these younger members were interrupted at ages 35 to 40 years by the epidemics at the end of First World War.

At age 60 years, however, the first of two waves of increases in inter-cohort survivorship had hit this cohort. The first wave started in 1936 and lasted to 1951. Mortality increases at ages 60 to 70 years between 1931 and 1941 constituted a fundamental part of the force which collided with the continuing mortality declines at younger ages during *the period of conflicting forces of mortality change*. The probability of this cohort surviving through the 70 to 75 years age group was lower than the cohort born twenty years earlier.

The second wave of inter-cohort mortality increases occurred between 1956 to 1976 at ages 85 years and over, during the period of widespread deterioration in non-Maori male mortality. Compared to the first wave hitting this cohort, increases in mortality rates at more advanced ages were less noticeable. The younger members of the cohort who were particularly advantaged by mortality declines during infancy and young adulthood appeared to be more adversely affected by the second wave of inter-cohort mortality increases.

The 1881-1891 male complete cohort, like their immediate predecessors, benefited handsomely from the persistent declines in mortality rates at younger ages, interrupted only by the epidemics at the end of First World War. However, the two waves of inter-cohort mortality increases mentioned above hit this cohort particularly hard. The impacts of this lasted to their most advanced ages.

Signs of inter-cohort mortality increases for the 1881-1891 male complete cohort first emerged at an earlier age of 50 years, towards the end of *the period of conflicting forces of mortality change*. The general recovery of survivorship at mid to late adult in the next period between 1941 and 1956 did not seem to have benefited this cohort. The trend of inter-cohort mortality increases continued and peaked at ages 75 to 85 years, at the height of widespread non-Maori male mortality deteriorations at around 1961 to 1971. Not until the late 1970s significant mortality reductions at older ages started to reverse the trend of worsening survivorship at ages 90 years and over, and hence produced zero net movement in life expectancy at age 65 years from the previous cohort.

Compared to the two earlier cohorts, the effect of inter-cohort mortality increases on non-Maori males born between 1891 and 1901 was restricted to a smaller age range of 55 to 80 years. The trend of inter-cohort survivorship improvements at younger ages decelerated. The impacts of epidemic mortality (and possibly the aftermath of the First World War) at the end of the Great War were more marked in this cohort at ages 20 to 25 years than their predecessors.

This complete cohort was not affected at all by the first wave of inter-cohort mortality increases which had exerted adverse impacts on the two previous cohorts. Instead, some

significant improvements in the cohort survivorship were made at the late reproductive ages of 30 to 50 years. These improvements were among the more dominant features in this cohort's mortality pattern.

The previous trend of inter-cohort mortality increases at ages 55 to 60 years was reversed in this cohort, although the level of mortality at these ages were still higher than that of the 1871-1881 cohort. Starting from age 60 years when the cohort entered *the period of stagnation and deterioration in life expectancy trends*, marked deteriorations in survivorship re-emerged. The deteriorations again peaked at the height of widespread non-Maori male mortality deteriorations at ages 70 to 75 years. The probability of surviving these ages for the 1891-1901 complete cohort was lower even than that of cohorts born half a century earlier.

After age 80 years significant increases in cohort survivorship had taken place, during *the period of significant life expectancy increases*. For example, the probability of surviving between ages 80 and 100 years for this cohort nearly doubled that for the two earlier cohorts. However, with less than a quarter of the original cohort surviving to age 80 years and the relatively high level of mortality operating at these advanced ages, the overall impact of these survivorship improvements on life expectancy was only minor.

Non-Maori Female Complete Cohorts

Mortality analysis for non-Maori female complete cohorts is more straightforward compared to that for non-Maori males since non-Maori females had fewer twist and turns in their cohort mortality trends. A trend of across-the-broad declines in females cohort age specific mortality rates was most distinguishable. The two major periods of mortality increases affecting non-Maori males were less visible in female complete cohorts, and the effects were restricted to slowing down the pace of inter-cohort mortality declines in age specific mortality rates rather than to reverse the trend.

Females born between 1871 and 1881 attained a better survivorship than had previous cohorts at every age group. Improvements in survivorship were generally significant at

most ages, except that a few age groups had shown a slowing down in the trend of inter-cohort age specific improvements. The first slow down in inter-cohort mortality declines occurred at ages 15 to 20 years when the older members of the 1871-1881 cohort experienced a slightly worse mortality rate between 1891 and 1896[1]. A more marked slow down was observed at ages 40 to 50 years between 1911 and 1926. This was mainly attributable to the widespread mortality increases at adulthood just at the end and after the First World War, which marked the end of a major phase of maximum short-run increases in life expectancy.

The younger members of female 1871-1881 complete cohort suffered minor mortality increases at ages 60 to 65 years during *the period of conflicting forces of mortality change* between 1936 and 1941. From ages 65 years, however, inter-cohort survivorship improvements accelerated up to the very old ages.

The trend of inter-cohort reductions in age specific mortality rates was more visible and across all age groups for the 1881-1891 female complete cohort. Increases in survivorship were most significant at ages one to five years between 1886 and 1896. The only slow down in the inter-cohort trend of mortality reductions was found at ages 55 to 60 years when mortality increased among the older members between 1936 and 1941, which constituted a major force of mortality changes during that period. Like their immediate predecessor, inter-cohort survivorship improvements accelerated from age 60 years onwards.

Non-Maori females born between 1891 and 1901 continued the trend of inter-cohort mortality reductions at every age. A few age groups demonstrated a slow down in this trend. The first occurred at ages 20 to 25 years between 1916 and 1921. The effects of epidemic mortality during this time, although minor compared to those on males, were compounded by the widespread stagnation in the trend of female mortality reductions at the time. Like their predecessors, the trend of inter-cohort mortality reductions was affected in the earlier part of *the period of conflicting forces of mortality change* at an younger age of 40 to 45 years. Similar effect on the younger members of this complete

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[1] A pandemic of influenza on a relatively small scale was recorded in 1894, which would affect survivorship in 1896.

cohort again occurred at ages 65 to 70 years during *the period of stagnation and deterioration in life expectancy increases*. Compared to the previous cohorts, the acceleration in inter-cohort survivorship improvements started at a later age of 70 years but at a faster pace.

In sum, male complete cohorts demonstrated strong cohort mortality effects. A distinct pattern emerged in males cohort in which the substantial inter-cohort survivorship improvements at younger ages were compensated by mortality increases later in life. In contrast, female complete cohort mortality trends seemed to be more dominated by period effects, namely, the widespread stagnation in the trend of mortality reductions at around 1921 and 1941.

10.1.2 Shifts in the Momentum of Mortality Reductions

An analysis of the contribution to increases in complete cohort life expectancy at birth by life-cycle stages was carried out, similar to those for the synthetic life tables in Chapter Eight. Increases in life expectancy at birth between successive cohorts and the proportion of these increases attributable to mortality reductions at five main life-cycle stages are estimated in Table 10.2. Methods of estimation are provided in Appendix Two.

Table 10.2 Increases in cohort life expectancy at birth and the percentage of increases attributable to mortality reductions at different ages, by gender, for cohort born between 1871 and 1901

	Males		Females	
Age group	1871-81 to 1881-91	1881-91 to 1891-01	1871-81 to 1881-91	1881-91 to 1891-01
e_0 increases	2.7 years	2.0 years	3.6 years	2.9 years
< 1 (p_0)	26%	39%	16%	25%
1-14 ($_{14}p_1$)	51%	27%	39%	18%
15-44 ($_{30}p_{15}$)	22%	25%	21%	22%
45-64 ($_{20}p_{45}$)	3%	11%	8%	15%
65-84 ($_{20}p_{65}$)	-1%	-2%	13%	16%

Sources: Computed by the author from published raw data and completed life tables constructed by Statistics New Zealand

A shift to older ages in the momentum of increases of life expectancy at birth, underlying the shift in the cohort age at death, was more visible for females. Significant reductions in infant mortality successfully captured increasingly larger proportion of life expectancy increases. Its net effect was to eliminate deaths at the very early ages and therefore helped to propel the age at death distribution further up the age scale.

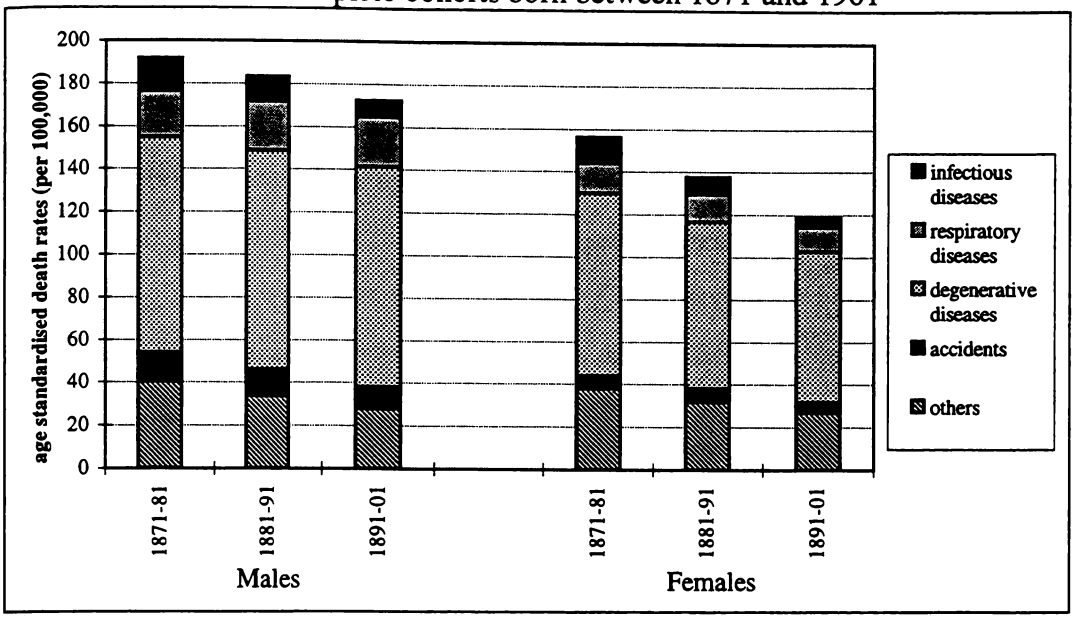
Mortality increases at older ages among males complete cohorts gave rise to little net movement in survivorship at these ages. Female complete cohorts, on the other hand, produced systematic shifts in age at death as suggested by the parallel shift of survival curves in Figure 10.2, and shifts in the momentum of mortality reductions as shown in Table 10.2. These issues are investigated further in the Chapter Twelve when looking at possible mortality compression by birth cohorts.

10.1.3 Changes in Patterns of Cause of Death

Degenerative diseases (cancers and cardiovascular diseases) were the most dominant causes of death. Figure 10.2 shows the breakdown of the age standardised death rates by major causes of death. The available data show that the risk of cancer deaths for males increased at all adult ages and was principally responsible to the stagnation in the inter-cohort trend of life expectancies at adult ages. At age 80 years and over, substantial reductions in the cardiovascular disease death rates had more than offset the increases in the cancer death rates, and produced net declines in mortality rates at these ages. The male age standardised degenerative disease death rates stayed at just above 100 per 100,000, increased marginally between the successive complete cohorts.

The female degenerative disease death rates reduced systematically from 86 per 100,000 for the 1871-1881 cohort to 71 per 100,000 for the 1891-1901 cohort. Between ages 40 and 55 years, the risk of degenerative diseases deaths increased marginally, though degenerative diseases were less lethal at these ages. At older ages, however, significant reductions in the degenerative disease death rates were recorded at all age groups. Mostly of these reductions were driven by declines in the cardiovascular disease death rates.

Figure 10.2 Age standardised death rate* by major causes of death, by gender, non-Maori complete cohorts born between 1871 and 1901



* age standardised to the combined non-Maori male and female populations in 1996

Note: "others" include other chronic diseases (ICD chapters 3-6, 9, 10, 12 and 13), maternal and infant deaths (ICD chapters 11, 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Computed by the author based on death rates for major cause groups compiled by I. Pool, and from cause of death data published by Ministry of Health and population data published by Statistics New Zealand

With the exception of respiratory diseases, general declines in the age standardised death rates occurred for all other major causes. Most of these declines were attributable to their receding impacts at younger ages, and therefore trends in the cohort age standardised rates mirrored those in synthetic life tables for the earlier periods, especially for infectious diseases.

The age standardised respiratory disease death rates changed little between the three complete cohorts. Female rates reduced marginally to 11 per 100,000 and male rates increased marginally to 23 per 100,000 in the 1891-1901 cohort. Reductions in the respiratory disease death rates at infancy and early childhood were cancelled out by increased pneumonia and chronic bronchitis deaths at ages 65 years and over starting from the late 1950s. For both genders of all three complete cohorts, the 1881-1991 cohort was the most affected by the increases in the respiratory disease death rates at late adult ages.

For each complete cohort the percentage breakdown of the age standardised death rates by major causes of death are shown in Table 10.3. Signs of heavy concentration of cohort causes of death in degenerative diseases was evident, similar to those observed in synthetic analysis in Chapter Eight. Deaths due to degenerative diseases accounted for just under 60 percent of the age standardised death rate. Though this figure was roughly 10 percentage points less than that seen in the most recent synthetic measures, it increased quickly with successive cohorts. The increased percentage share of the respiratory disease death rate reflected its stationarity and contrasted with general declines in all other major causes.

Table 10.3 Percentage distribution of the age standardised death rate by major causes of death, by gender, non-Maori complete cohorts born between 1871 and 1901

	Males			Females		
Causes of death	1871-81	1881-91	1891-01	1871-81	1881-91	1891-01
Infectious diseases	8%	6%	4%	8%	6%	4%
Respiratory diseases	11%	13%	14%	9%	10%	10%
Degenerative diseases	52%	56%	60%	55%	57%	60%
Accidents	7%	7%	6%	4%	4%	4%
Others	21%	18%	16%	24%	23%	23%
Total	100%	100%	100%	100%	100%	100%

Note: "others" include other chronic diseases (ICD chapters 3-6, 9, 10, 12 and 13), maternal and infant deaths (ICD chapters 11, 14 and 15) and ill-defined causes (ICD chapter 16)

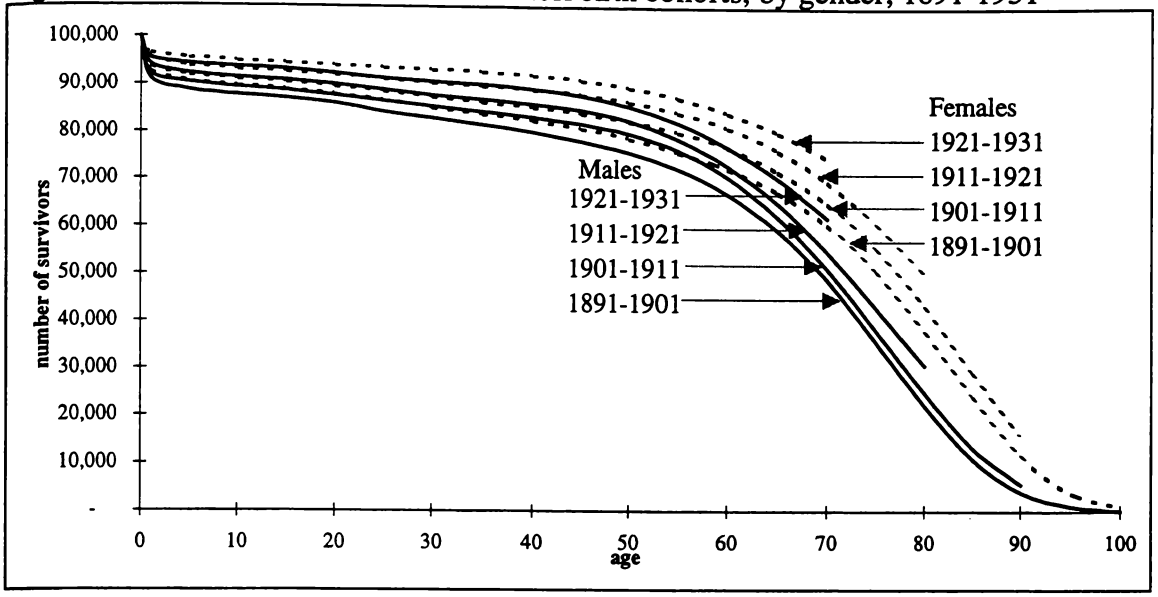
Sources: Computed by the author based on death rates for major cause groups compiled by I. Pool, and from cause of death data published by Ministry of Health and population data published by Statistics New Zealand

10.2 Mortality Trends of the Advanced Birth Cohorts, Born 1901-1931

Survival curves of non-Maori advanced cohorts born between 1901 and 1931 are presented in Figure 10.3. Survival curves of the 1891-1901 complete cohort are also included in Figure 10.3 as a reference point for comparisons.

After persistent mortality declines at younger ages, the initial drop in the advanced cohort survival curves at infancy diminished, and the curves between ages one and 50 years appeared increasingly flatter with successive cohorts. In comparison, mortality developments became more significant at older ages.

Figure 10.3 Survival curves of non-Maori birth cohorts, by gender, 1891-1931



Sources: Cohort life tables constructed by the author, based on published raw data and complete life tables constructed by Statistics New Zealand

A convergence of the survival curves at older ages previously observed for male complete cohorts did not reappear. Instead, a tendency of divergence at older ages for the male advanced cohort survival curves can be seen from Figure 10.3. While the survival curve of the 1901-1911 advanced cohort moved in parallel with its immediate predecessor, the two successive advanced cohorts had accumulated increasingly larger gains in their survival curves. Life table first quartile age at death was delayed systematically from 56.3 years for the 1901-1911 cohort to 58.4 years for the 1911-1921 cohort, and to 61.7 years for the 1921-1931 cohort[2].

The general lack of movement in male mortality at younger ages between 1931 and 1941 slowed the trend of inter-cohort mortality declines. This particularly affected the 1911-1921 cohort at ages 15 to 30 years.

The effects of deterioration in adult male mortality rates during *the period of stagnation and deterioration in life expectancy trends* were evident in the mortality patterns of the advanced cohorts. Age specific mortality rates either levelled off or increased, at a progressively younger age: at ages 55 to 70 years for the 1901-1911 cohort; ages 45 to 60 years for the 1911-1921 cohort; and ages 35 to 50 years for the 1921-1931 cohort.

2 Life expectancy cannot be calculated directly for these cohorts.

The older the age group, the worst the effects of mortality deteriorations. For example, the probability of surviving from ages 60 to 65 years for the 1901-1911 cohort was lower than that of the 1871-1881 cohort, or lower even to those born another twenty years before that.

Starting from the early 1970s the advanced cohorts benefited from general survivorship improvements at adult ages. Increases in survivorship were visible at an progressively earlier age of 70, 60 and 55 years respectively, and at an accelerating pace.

Female advanced cohorts continued to make significant gains in survivorship at most ages. The first quartile age at death increased at a faster pace than their male counterparts, at 62.1 years, 66.1 years and 74.9 years for the three successive cohorts. Female advance cohort survival curves maintained their momentum and continued to shift in parallel towards upper ages, capitalising on the gains built at younger ages.

Compared to male advanced cohorts, the impacts of general stagnation and reversals in the trends of mortality reductions in the 1960s were much less significant for female advanced cohorts. The 1901-1911 female advance cohort did not appear to be adversely affected by this cross-sectional phenomenon, with only the 60 to 65 years age specific mortality rate showing a moderate slow down in the inter-cohort trend of age specific mortality reductions. The 1911-1921 cohort mortality rates at ages 50 to 55 years levelled off with the previous 1901-1911 cohort at the same ages. The 1921-1931 cohort appeared to be more affected than the other two, with marginal increases in mortality rates at ages 40 to 50 years. Starting from the early 1970s, accelerated survivorship improvements among female advanced cohorts generally started at a later age of 65 to 70 years and accelerated at a slower pace compared to that for males.

Age specific death rates for most major causes of death reduced between successive advanced cohorts of both genders. The most impressive was the continuing declines in the cardiovascular disease death rates at late adult ages. Age specific respiratory disease death rates at late adult ages also declined, reversing from the early trend. The only major exception to this general declines were the continuing small incremental changes in the cancer death rates between ages 40 and 75 years for both genders. At older ages,

however, where data are available, it seems that age specific cancer death rates had stabilised and started to trend downwards.

10.3 Mortality Trends of the Younger Generation Birth Cohorts, Born 1931-1961

Mortality rates of non-Maori cohorts born between 1931 and 1961 have been generally lower at early ages than those of earlier cohorts. The principal feature in the mortality patterns thus far for the younger generation of cohorts are the fluctuations in mortality rates at young reproductive ages. The range of ages affected by these mortality fluctuations has spread with successive cohorts.

The 1931-1941 cohort was the least affected by these mortality fluctuations. Male cohort mortality rates at ages 30 to 35 years stayed almost the same as those of its immediate predecessor, while the trend of inter-cohort mortality reductions continued at all other ages. Similar patterns were found for the 1931-1941 female cohort, with marginal age specific mortality reductions at ages 30 to 40 years.

Male cohort born between 1941-1951 again showed almost zero net inter-cohort change in mortality rates at ages 20 to 30 years. Their female counterparts also showed minimal inter-cohort movement in the mortality rates at ages 15 to 35 years. Female mortality rates at ages 20 to 25 years increased slightly from the previous cohort of 1931-1941 at the same age

The ages at which mortality rates fluctuated spread wider in the 1951-1961 cohort to five to 35 years for both males and females. Substantial increases in male mortality rates occurred at ages 15 to 30 years, and to a lesser extent at ages 30 to 35 years. More moderate increases in female mortality rates were found at ages 15 to 30 years.

The net effect of mortality fluctuations at the early reproductive ages among the three successive younger generation of cohorts was for a convergence of cohort survival curves at younger ages. Survival curves for these cohorts are shown in Figures 10.4a and

10.4b in the next section and comparisons with those for earlier cohorts. In general, mortality fluctuations affected a wider age range for females, but were more adverse for males in terms of magnitude of change. Interestingly, poorer mortality experiences at the early reproductive ages have not seemed to have affected the trend of inter-cohort mortality reductions after age 35 years. For example, the male 1951-1961 cohort who had experienced substantial mortality increases at younger ages also attained large mortality reductions at ages 35 years and over

Age specific death rates reduced for all causes and at most ages, where data are available. The cancer death rates reversed from previous trends and started to decline at mid adult ages. Declines in the respiratory disease death rates at mid adult ages were also noticeable. But, the accident death rates at the early reproductive ages seemed to be on an upward trend, particularly affecting the 1951-1961 cohort. These causes were therefore responsible for mortality fluctuations at early reproductive ages, the most prominent feature of the younger generation of cohort mortality patterns to date.

10.4 Discussion: Implications for Future Cohort Mortality Developments

A finding of significance from cohort mortality analysis is an extension of cohort survival curves. Survival curves for the nine successive cohorts are produced in Figures 10.4a and 10.4b, and are also compared with the survival curve from the 1996 synthetic life table.

These figures show that the successive non-Maori cohorts have made considerable gains in survivorship. Most of these gains were made at younger ages. In sequence, cohort survival curves have progressively closed in on the 1996 synthetic curve. As room for further improvements in survivorship at younger ages diminishes, as is reflected by the narrowing space between the curves at these ages, developments at older ages become critical.

Figure 10.4a Non-Maori male survival curves, 1891-1961 birth cohorts and 1996 synthetic life table

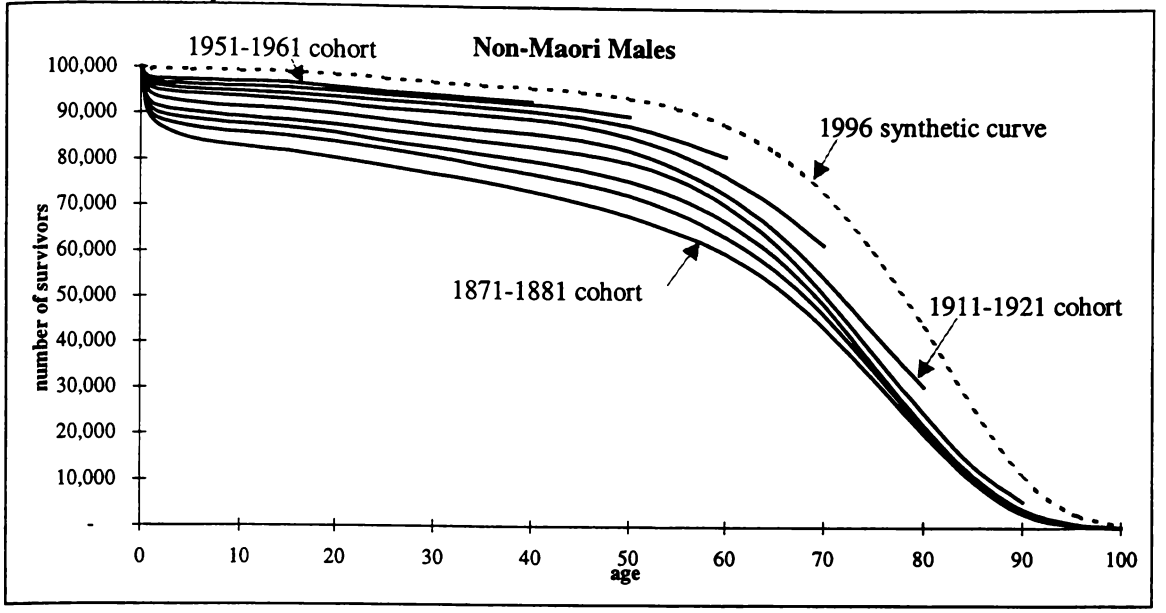
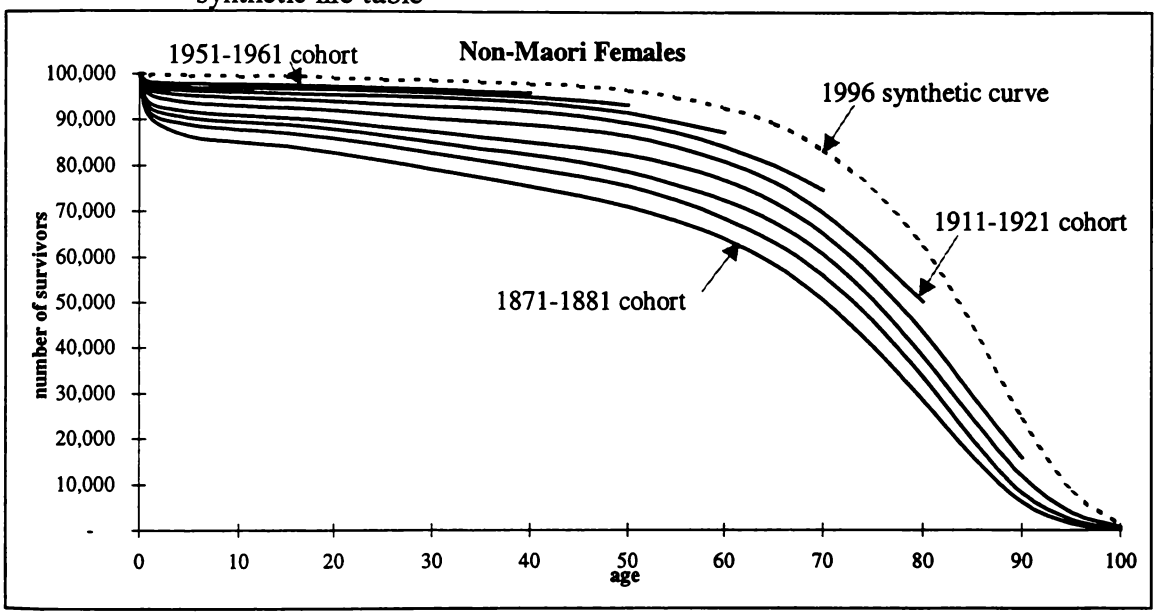


Figure 10.4b Non-Maori female survival curves, 1891-1961 birth cohorts and 1996 synthetic life table



Sources: Cohort life tables constructed by the author, based on published raw data and complete life tables constructed by Statistics New Zealand

The distinct pathways of each cohort at older ages is highly significant. At first there were a converging trend at older ages, but more recently male cohort survival curves have begun to shift in a similar fashion to that displayed by their female counterparts. The parallel shifts of cohort survival curves, showing no sign of approaching a boundary, is contradictory to that implied by the hypothesis of a compression of mortality. Instead of meeting a right-hand boundary which would progressively reduce the space of rightward

extension and steepen the slope of decline at older ages, cohort survival curves have maintained both the space and slope dimensions of a rightward extension.

A direct implication of the parallel shifts in cohort survival curves is that period trends of mortality compression in both the relative and absolute senses observed from synthetic life tables are not a true reflection of the underlying trends of cohort mortality change. This seems particularly the case for non-Maori males whose survival curves have only begun to shift at older ages parallel to that for their immediate predecessors. The early signs are that cohort survival curve extension will continue, and have every chance of crossing the 1996 synthetic curve. Accordingly, the significant increases in old age survivorship occurred during *the period of significant life expectancy increases* between 1971 and 1996 are likely to continue, at least for the short to medium terms, should current cohort mortality trends persist.

The complete male cohorts born in the period 1871 to 1901 displayed a consistent pattern of substantial gains in survivorship early in life but systematic mortality increases at late adulthood. A similar level of consistency were found in female cohort mortality patterns. Compared to their male counterparts, female complete cohorts made less significant gains in survivorship at younger ages but minimal negative effects in mortality at older ages. These findings support the hypothesis that accumulated cohort exposure to the risk of mortality contributes to cohort mortality experience later in life. An inference which can be drawn from this is that differentials in cohort patterns of accumulated exposure to the risk of mortality will help to produce variance in cohort mortality pathways at older ages.

The advanced cohorts born in the period 1901 to 1931 are currently at ages 65 to 95 years, at ages at which the large majority of life table decrements occur. These cohorts are therefore in a position to influence overall mortality trends when measured cross-sectionally. So far both genders have been favoured by persistent mortality declines at older ages. However, a pattern of trade-off between mortality reductions at younger ages and at older ages is again emerging. Unlike the complete cohorts whose significant mortality reductions at younger ages were at the expense of mortality fluctuations and even deterioration later in life, the advanced cohorts displayed the opposite pattern. A

general lack of mortality movement or some minor fluctuations early in life favoured some significant decreases in mortality at older ages.

Gender comparisons of trends in cohort survivorship among the advanced cohorts offer a further valuable insight into the effects of accumulated cohort risk exposure. Compared to females, male cohorts experienced fewer gains in survivorship at younger ages and more extensive mortality deterioration at adult ages. At the more advanced ages, however, gains in male survivorship were generally greater than for females. The pattern of trade-off between mortality reductions at younger ages and at older ages seem to suggest that the recent trend of mortality declines at older ages among the advanced cohort is likely to continue.

The younger generation of cohorts have made progressively fewer gains from mortality reductions at earlier ages. In fact, the minor fluctuations at the early reproductive ages may well be a critical factor in determining their mortality trends later in life. Following the trade-off scenario, mortality declines at the middle to late adult ages among the younger generation of cohorts may accelerate even further than the earlier cohorts.

Cohort mortality data analysed in this chapter do not indicate a compression, if anything they point to extension. But their pathways have not been systematic and monotonic. Instead, various cohorts, mainly males, have been exposed both to improvements and deterioration in survival, in very different ways. The discussions on cohort mortality paths help to set a baseline for cohort-based approach to mortality projections in Chapter Fourteen. Before this, however, there is a need to test in a more refined way for the occurrence or non-occurrence of mortality compression. This question will be explored in detail in the next two chapters (Eleven and Twelve).

PART FIVE:

EXPLANATORY ANALYSIS OF NON-MAORI MORTALITY AND MORBIDITY: IMPLICATIONS FOR FUTURE HEALTH OF ELDERLY NEW ZEALANDERS

CHAPTER ELEVEN
MEASURING MORTALITY COMPRESSION IN NON-MAORI SYNTHETIC LIFE TABLES

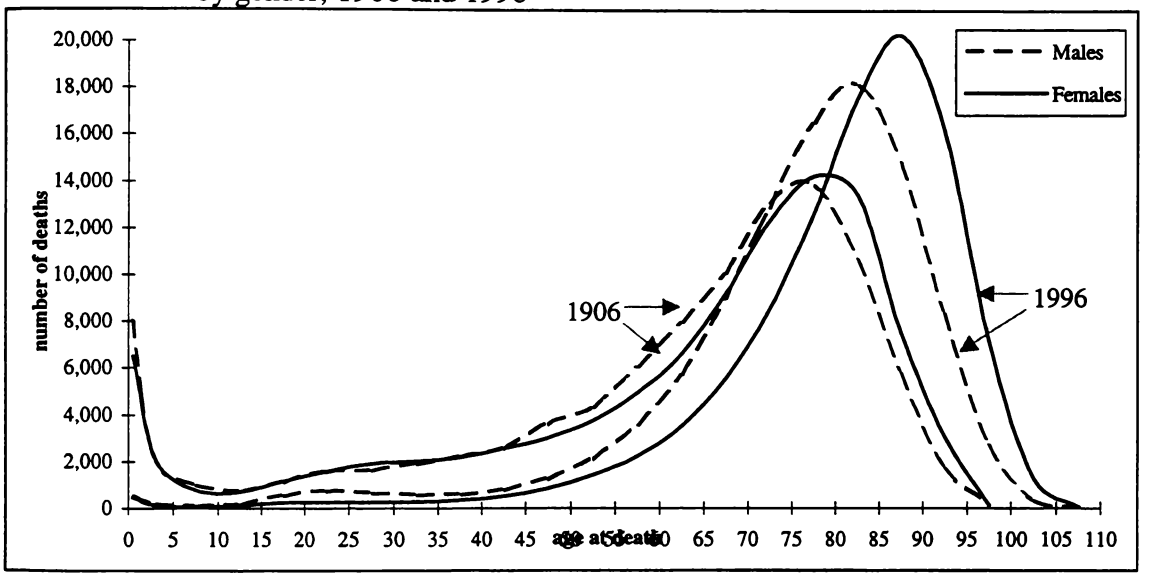
An historical shift towards older ages in the momentum of mortality reductions was identified from the analysis of the century long non-Maori mortality transition in Chapter Eight as a major emerging substantive issue in the contemporary study of population health. At the turn of the twentieth century mortality reductions at ages less than 45 years had accounted for 80 percent of the increases in life expectancy at birth. By the end of the twentieth century, the corresponding figure had dropped to less than 25 percent. After substantial improvements in survivorship at infancy, childhood and the reproductive period, mortality rates at these ages have now reduced to close to a theoretical minimum. Consequently, mortality rates at these younger ages are becoming less dominant in determining the future movements of life expectancy. At the other end of the age spectrum, greater numbers of people are surviving to, and hence more deaths are occurring at, older ages. Further boosted by significant improvements in survivorship in the most recent period, mortality at older ages is starting to exert a dominating influence over the overall mortality pattern.

This chapter is devoted to this issue of the shifts in the momentum of mortality risk towards advanced ages, using the compression of mortality hypothesis further developed in Chapter Six as the framework. A summary of the issue is first given in this chapter, covering an historical overview of the changing situation and a brief reiteration of the concept of the compression of mortality. Further refinements to the conceptual and methodological aspects of the concept are proposed, including the development of operational measures of mortality compression. The analytical strategies here developed are then applied to the non-Maori population to test for mortality compression over the most recent *period of significant life expectancy increases* at older ages between 1971 and 1996.

11.1 An Overview

This shift in the underlying momentum of future life expectancy changes was a direct consequence of a shift in the force of mortality, which can be approximated by the age at death distribution from the life table. The changes in the age at death distribution between 1906 and 1996 are illustrated in Figure 11.1. The two years were chosen to represent the beginning and the end of a century long mortality change. Life tables prior to 1906 were restricted to an open-ended age group of age 80 years and over because mortality data were unavailable for finer age group breakdowns, and this thus precludes their use for detailed mortality analyses at more advanced ages.

Figure 11.1 Age at death distribution ($_nd_x$) from the life tables, non-Maori population, by gender, 1906 and 1996



Sources: Computed by the author from published raw data and life tables constructed by Statistics New Zealand.

Two peaks occurred in the 1906 age at death distribution, first at infancy and early childhood, and then at around ages 55 to 85 years. In contrast, by 1996 the peak at infancy had receded to very low levels, but the peak at older ages had been greatly reinforced. For females, the peak in 1996 was delayed to even older ages and appeared sharper. The space between the 1906 and 1996 curves, representing improved survivorship at younger ages and a heavier concentration of death at older ages, is clearly noticeable at every age. Since the total space under each curve is fixed to represent the hypothetical 100,000 newborns of the conventional life table, the contrasting patterns

between the 1906 and 1996 curves confirms the hypothesis of a sequential re-distribution of age at death.

Figures 11.2a and 11.2b depict non-Maori male and female trends in quartiles and percentiles of age at death distribution between 1881 and 1996. These figures suggest that the shift in the age at death distribution had been a long term systematic process.

Figure 11.2a Selected quartiles and percentiles of the age at death distribution ($_nd_x$) from the life table, non-Maori male population, 1881-1996

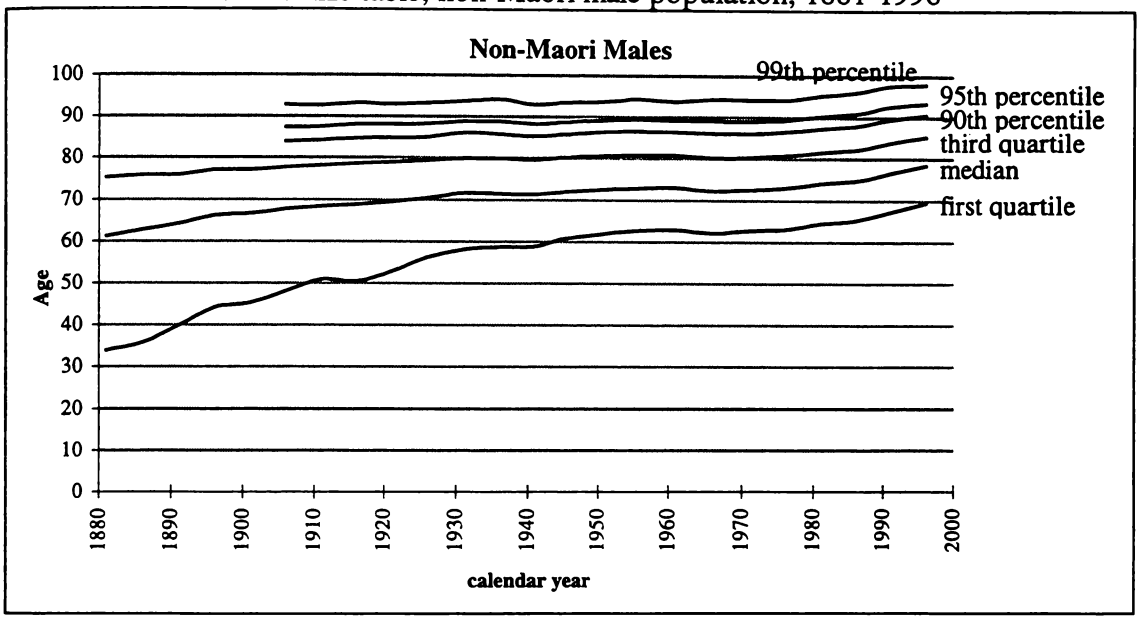
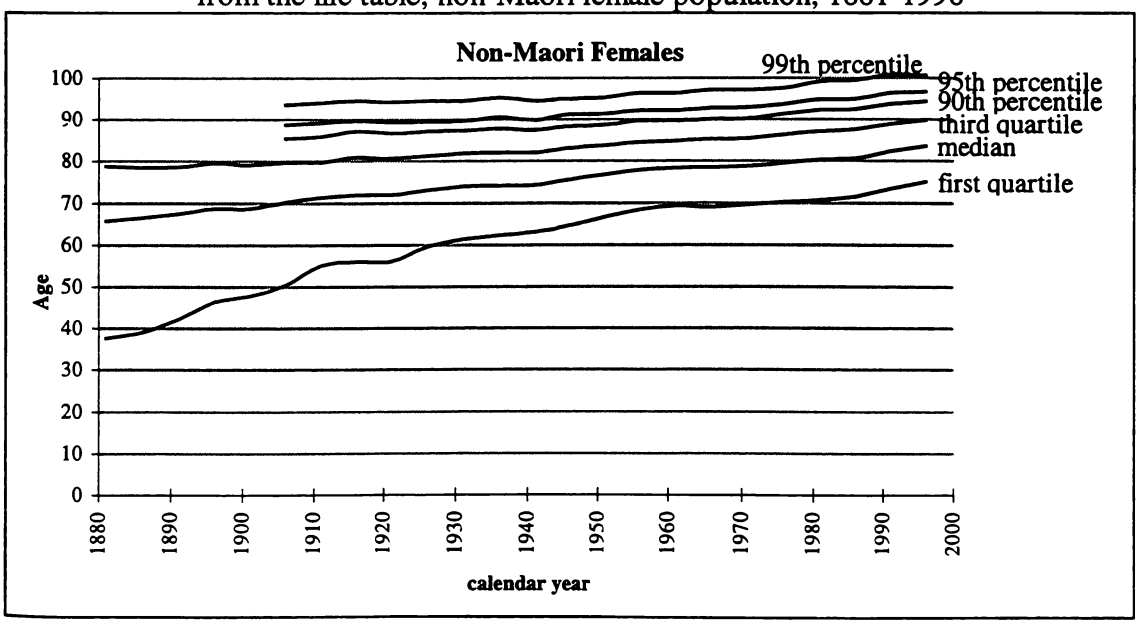


Figure 11.2b Selected quartiles and percentiles of the age at death distribution ($_nd_x$) from the life table, non-Maori female population, 1881-1996



Sources: Computed by the author from published raw data and life tables constructed by Statistics New Zealand.

The first quartile age at death increased rapidly, from 34.0 years for males and 37.7 years for females in 1881 to double that, 69.4 years and 75.1 years, in 1996. As a result, the remaining three-quarters of deaths were increasingly "compressed" into a narrower age range. For other higher quartiles and percentiles, there were regular linear increases up to the late 1970s. Since then significant reductions in mortality rates at older ages produced a parallel and exponential upturn in all curves, a most distinguishable departure from the previous linear trends.

The greatest convergence of the quartile and percentile curves occurred during the period encompassing the phases of maximum short-run increases in life expectancy at birth prior to 1930. Over the next 30 years, the first quartile age at death increased at a slightly faster rate than the others. Since 1960, however, near parallel movements have occurred in all quartile and percentile curves for both males and females.

A more important feature from Figures 11.2a and 11.2b is that the third quartile and all higher percentiles curves have maintained equal distances from each other from the early twentieth century when data on ages 80 years and over became available. The patterns of almost parallel increases is of great interest, especially in light of the significant mortality reductions at older ages in more recent years. It appears that, instead of converging into a very narrow age range, the age range for three-quarters of deaths was maintained and had shifted in tandem with the increase in the first quartile age at death.

The exact nature of this sort of spread of age at death has some serious implications for future mortality developments. Will the future age at death distribution maintain this characteristic spread, or will it converge and thus signal the limit to any further mortality declines? The rest of this chapter is devoted to analysing synthetic life tables in order to attempt to understand this mortality trend and its future implications.

11.2 The Compression of Mortality - A Conceptual Framework

Chapter Four provided an overview of contemporary theories and international research on the recent mortality trends at older ages of populations mainly in developed countries.

A further brief review of this issue is provided below to fit that earlier overview to the findings so far in this thesis, so as to help to refine the analytical approach to be followed in this and the next chapters (Twelve).

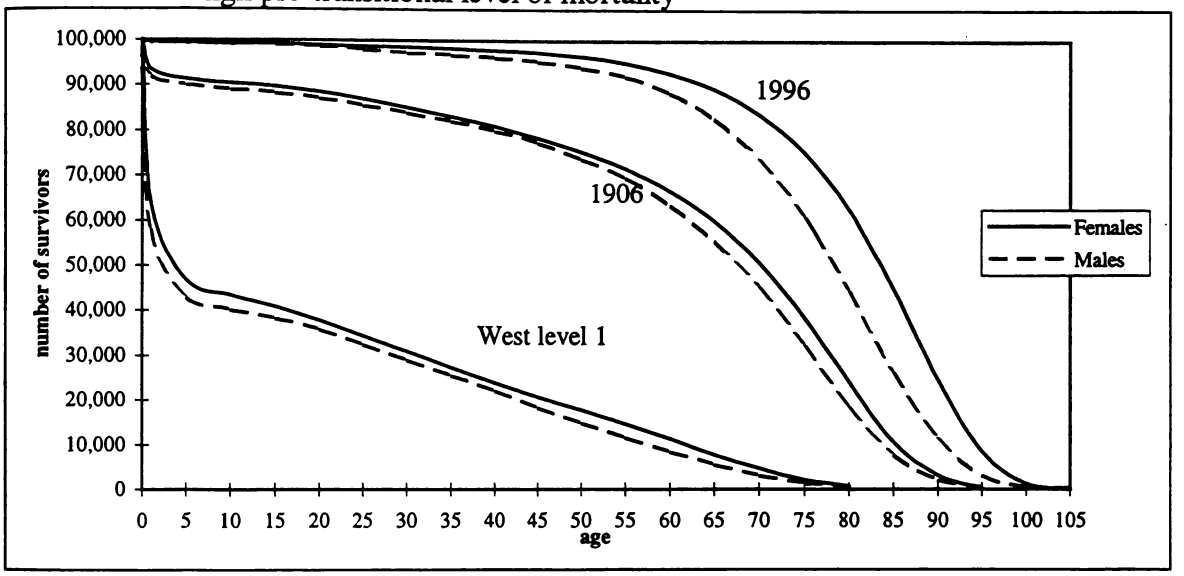
In an article published in 1980, Fries argued that the human life span is subject to a fixed biological limit which is in the proximity of 115 year, and that the average length of life, measured by the life expectancy at birth, is unlikely to exceed 85 years. This limit to life expectancy is now rapidly approaching in most developed countries. The combination of a fixed boundary and accumulating momentum of movements toward this boundary will, Fries argued, naturally lead to a congestion of the age at death distribution at around the mark of 85 years of age. The age range in which the great majority of deaths occurred will become increasingly narrower. This idea of a heavy clustering and compression of age of death into an increasingly narrower age range is referred to as the "compression of mortality".

The final outcome of a compressed mortality pattern is a highly compressed age at death distribution (see Figure 4.1) with a very well-defined tall peak and a narrow base. As a corollary, the peaks in Figure 11.1 and the percentile curves in Figures 11.2a and 11.2b would, if Fries is correct, converge and stay within a narrow range once the supposed limit to life expectancy is reached.

A statistical manifestation of the concept of compression of mortality in a life table context is the notion of "rectangularisation of the survival curve". This notion is based on the idea that as mortality declines persist and deaths continue to concentrate at older ages, the survival curve will assume a horizontal line depicting low mortality at younger ages, and this will be followed by a near vertical drop at the upper end at the age of "natural deaths" due to senescent frailty (Fries, 1980). Theoretically, the horizontal tendency at younger ages and the vertical tendency at the very old ages will move further and further from the axes to combine to produce a right-hand "rectangular" effect in the survival curve (see Figure 4.2). The survival curves in 1906 and 1996 are produced in Figure 11.3 to help to illustrate this concept. Those for 1996 are closer to rectangular than the curves observed earlier in the twentieth century.

Also included in Figure 11.3 are the survival curves from model life tables West Level one, corresponding to high pre-transitional level of mortality. These latter group of survival curves are included to illustrate the reverse to the effect of left-hand rectangularisation, where the survival curves are almost parallel and close to the two axes. This shows the progress has been made thus far towards the proposed right-hand rectangularisation. The model life table West Level one has been fitted to Maori age-data by Pool (1991) as a means for indirectly estimating Maori mortality in earlier times.

Figure 11.3. Survival curves of the non-Maori population, by gender, 1906 and 1996; and survival curves by gender from model life table West Level one with high pre-transitional level of mortality



Sources: 1906 - computed by the author from published raw data by Statistics New Zealand.
1996 - extracted from life tables constructed by Statistics New Zealand.
Model life table West Level one - extracted from Coale-Demeny, *Regional Model Life Tables and Stable Populations* (Princeton, 1966), reproduced in United Nations (1967).

Under the pre-transitional mortality regime in West Level one tables, life expectancy at birth was estimated at 18 years for males and 20 years for females. Extremely high levels of infant and early childhood mortality dominate the overall mortality pattern. Nearly 40 percent of the newborns were expected to die in their first year of life, and less than 50 percent survived to five year of age. By age 50 years, less than 20 percent of the original cohort was still alive. Despite the accelerating risk of death at older ages, the survival curves tapered off instead of decreasing exponentially. This was because the small numbers of survivors reaching these older ages allowed little scope for the effect of

exponentially increasing mortality risk to be evident at a population level. The considerable falls to half of the survival curves at very young ages created a concave survival curve. The space above the curve, corresponding to the lost in life years, resembles a rectangle, but left-hand oriented.

The non-Maori survival curves in 1906 exhibit a classical horizontal "s" shape, typical of mortality patterns of population in mid-transition. The impact of infant mortality was significant, as is reflected in the initial sharp fall in the survival curves before stabilisation at childhood. A slowly accelerating decline in survival curves starts from age 15 years, suggesting that mortality was a substantial risk at every age. The impact of mortality intensified with age. The levelling off of the survival curve from age 85 years onward is explained by the significantly reduced number of survivors at these advanced ages, leaving considerably less scope for the effect of accelerating increases in mortality rates to be evident at those ages at a population level.

Consistent declines in infant mortality had practically wiped out the initial sharp fall in the survival curves by 1996. Similarly, mortality declines at childhood and reproductive ages pushed up the 1996 survival curves to widen their space from the corresponding 1906 curves. With 95 percent of the hypothetical newborn cohort expecting to survive to age 50 years because of the very low levels of mortality at childhood and young adulthood, the 1996 survival curves stayed nearly flat between birth and age 50 years. Mortality rates then started to increase exponentially with age, and thus increasingly larger numbers of the original cohort fail to survive to the next higher age group, producing an exponential drop in the survival curves. The levelling off at very low levels in the 1996 curves was delayed to age 90 years or even later compared with 80-85 years for non-Maori in 1906, and perhaps as low as 70-75 years for Maori in the 1890s, when they had a regime resembles West Level one (Pool, 1991).

Compared to the survival curves in 1906, the 1996 curves demonstrated a considerable upward and rightward expansion, filling up much of the space in the upper right-hand corner of Figure 11.3. In doing so, the 1996 survival curves began to resemble rectangle in their appearance.

11.3 Measuring Compression of Mortality of Non-Maori

Though results of studies on mortality compression are starting to accumulate in the literature (see for example Coale, 1996; Go et al, 1995; Nusselder and Mackenbach, 1996; 1997; Paccaud et al, 1998; Rothenberg, 1991), disagreements remain over specification of the concept and its more refined operational measurement. The objective of this section is to deal with these outstanding issues before preceding with the empirical analysis on non-Maori reported in the next section. In this section, the concept of mortality compression is refined with proposed specification to a number of its parameters. Methods for measuring mortality compression are then developed.

11.3.1 Refining Operational Specifications of Compression of Mortality

In presenting his hypotheses, Fries (1980; 1983; 1988; 1989) gave no precise definition nor any specifications as how to measure the compression of mortality or the rectangularisation of the survival curve. Instead, he offered a series of predictions deduced from some fragmented evidence. As a result, the concept has been subjected to some very loose interpretations in the literature (see Anson, 1992).

A number of parameters of this concept need to be more rigorously defined. First, the compression of mortality can take two forms:

1. The empirical form - determined from the empirical population based on population and mortality data.
2. The standardised form - determined from the life table population, a refined measure which is free of age compositional effects.

Linking the two forms of compression is the age structure of the study population. Given that the compression effect is essentially a phenomenon hidden in the mortality trend over time, longitudinal comparability is therefore important in detecting this trend. Measuring compression in the standardised form is more meaningful in a longitudinal context than its empirical counterparts. This is because standardised measures will not be influenced by the age distribution of the observed population, making comparisons

between populations and over time possible (Nusselder and Mackenbach, 1996). In fact, the concept of the rectangularisation of the survival curve is strictly applicable only to the life table survival curve. Testing rectangularisation using age at death data from the empirical population as was done by Go et. al. (1995) invalidates their own assessment process. For these reasons, determining the compression of mortality in its standardised form, that is, from a series of non-Maori life tables, is adhered to for this and other chapters.

Second, much of the controversy has arisen because of disagreements over the relevant age range to be measured. The study of the upper tail of the survival curve can be problematic since data are sparse and not necessarily reliable. Results are often highly sensitive to the age cut-off points at both ends, as demonstrated by Fries himself (1984). It becomes clear that conventional techniques employing standard deviations are inadequate to account for the spread.

This is because observation suggests that only a demographically insignificant number of people in any cohort will survive to very advanced ages. The demographic study of mortality concerns the population, not a small group of hardy survivors. For the purpose of filtering out the outliers which may give rise to some erratic behaviour of the tail of the survival curve, an upper age cut-off point should be introduced in addition to the lower age cut-off point often already in use.

Third, in measuring the rectangularisation of the survival curve the criteria of a narrowing age range of the bulk of age at death distribution and an increasing life expectancy were typically applied. The first criterion is the principal pre-requisite for rectangularisation. An increasing life expectancy, though desirable, is not a pre-requisite of rectangularisation. Rectangularisation, or mortality compression, is the heavy clustering of death around an age interval, and this can happen in both high and low mortality regimes. The level of mortality, as reflected in life expectancy, is not be a pre-condition, but a parameter for identifying the type of compression as discussed in the next point. Given the current low level of mortality, any improvements in life expectancy will further extend the survival curve and will tend to produce the counter effect of widening the base of the age at death distribution. The criterion of an increasing life

expectancy in measuring mortality compression is therefore relaxed in the subsequent analysis to focus better on the core issue.

Fourth, a critical question is really to view mortality compression not only in absolute terms, but also in a relative sense so as to account for the level of mortality. Absolute compression is determined by the absolute magnitude of the distribution to be measured (e.g. age range in number of years), while relative compression is determined by taking the absolute magnitude as a function (e.g. ratio) of an appropriate indicator of the level of mortality. Intuitively, using Figures 11.2a and 11.2b of quartile and percentile trends of age-at-death distribution as an example, absolute compression can be understood as the convergence of the curves over time and can be measured by the distance between these curves themselves. The convergence, the equivalent of an absolute mortality compression, can happen anywhere along the age spectrum, including the lower age range. Relative compression can then be measured as a ratio of the absolute distance between the curves to their collective distance from the origin, age 0. This collective distance from the origin is an indicator of the level of mortality.

One possible scenario depicted in Figures 11.2a and 11.2b is that the curves maintain their absolute distance and move further away from the origin. In this scenario absolute compression has not occurred because the distances between the curves stay the same, but relative compression is achieved since the denominator of the ratio calculation has increased and consequently the ratio has reduced. This simple example helps to highlight the point that measuring mortality compression in both senses is necessary, and results of each measure should not be interpreted in isolation, but should be used to complement each other.

11.3.2 Methods for Measuring Mortality Compression in Life Tables

Movements towards mortality compression as proposed by Fries start to be dominated by the mortality experience at older ages once mortality at younger ages is reduced to sufficiently low level as shown in Figure 11.3. To recall from Chapter Eight, it was shown that major changes in non-Maori mortality rates at older ages occurred only

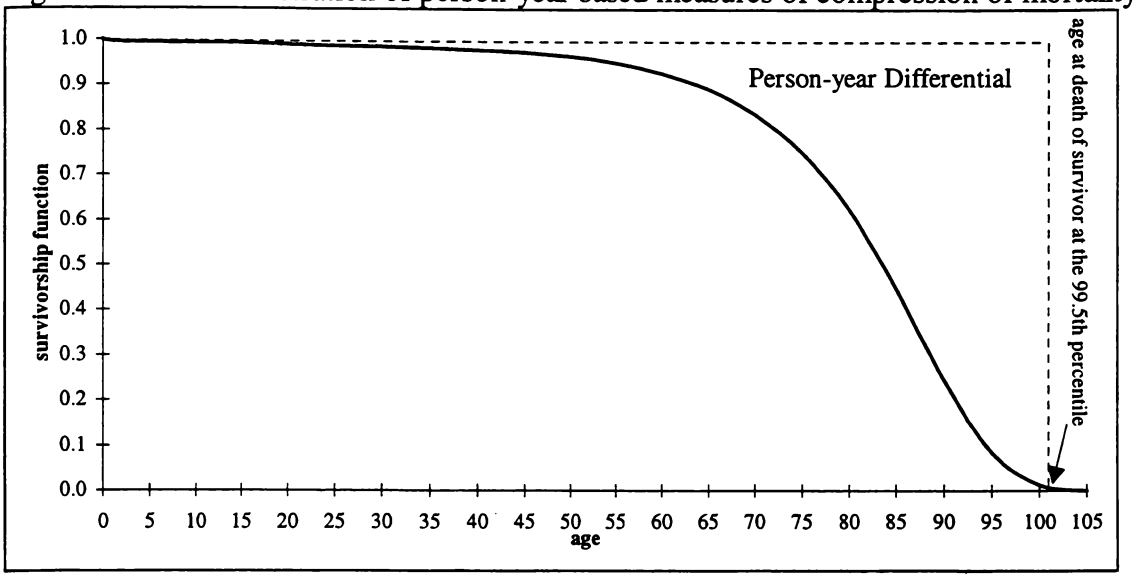
during *the period of significant life expectancy increases* between 1971 and 1996 when the level of mortality at younger ages was already low. This period is therefore chosen as the most appropriate time interval for the following analysis to determine whether mortality compression is occurring in the non-Maori population.

Measures of mortality compression used in the literature range from visual inspection of the survival curve to more sophisticated statistical indices. Each has its own merits, but none enjoys popular acceptance which would support its wider application. The primary objective of testing the rectangularisation of the survival curve is to determine how rectangular is the survival curve; or, how different is the survival curve from a perfect rectangle as defined by parameters inherent in the survival curve itself. Viewed from this first principle, the most direct and intuitive approach would be to measure the area under the survival curve, the total person-years as this is conventionally known, and its difference from a theoretically defined rectangle.

The difficulties as with most other approaches remains: how to determine the upper age boundary so that a rectangle can be defined. Determining this upper age cut-off point, or the right boundary of the rectangle, should be guided by two criteria. First, the proportion of people included in the study group after both the lower and the upper age cut-off points are imposed should be significantly large to substantiate extrapolations of the findings to the entire population. Second, results of analysis after adopting this upper age cut-off point should not be unduly influenced by statistical variations typical at the upper ages. For the purpose of the present analysis, the upper age cut-off point is set at the age at death of the 99,500th survivor of the any original 100,000 persons in the life table cohort at birth. These specifications are illustrated in Figure 11.4. To standardise the measurements shown in the illustration and those used in the analysis, the l_x function is converted with l_0 equals to one. The upper age cut-off point is the age at which the life table l_x function drops below 0.005[1].

[1] A range of upper age cut-off points, the 95th, 99th, 99.5th and 99.9th percentiles, were tested for their sensitivity and suitability. The results support the selection of the 99.5th percentile as the upper age cut-off point.

Figure 11.4. An illustration of person-year based measures of compression of mortality



Sources: Developed by the author

The vertical dotted line denotes the right boundary drawn at age at death of survivor at the 99.5th percentile. A perfect rectangle defined by the parameters inherent to the survival curve itself is therefore formed, with the two axes and the two dotted lines as its boundaries.

Two empirical measures of mortality compression are developed by the present author based on the area within this rectangle are thus developed. The first, person-years differential (*PD*), is the space inside the rectangle between the survival curve and the upper and right boundaries of the rectangle, as shown in Figure 11.4. This index is a measure of the average person-years required to produce a perfectly rectangularised survival curve. It can also be interpreted as an estimate of the average person-years gained if the entire original cohort were to survive to the upper boundary (as defined in this case by the age at death of survivor at the 99.5th percentile).

A second measure is the person-years ratio (*PR*), calculated as the ratio of *PD* to the entire rectangle. This index can be understood as a reciprocal measure of the percentage of "rectangularity" of the survival curve. A value of zero indicates perfect rectangularity, and a value of 0.5 would be the equivalent of a triangular survival curve at an inverse 45 degrees to the age axis.

PD is a measure of mortality compression in its absolute sense. The present author would further define that an absolute compression is said to occur when the area *PD* decreases over time. Moreover, *PR* is a ratio and suited for measuring mortality compression in its relative sense. A relative compression is said to occur when *PR* decreases over time.

The two person-year based empirical measures are calculated for non-Maori survival curves between 1971 and 1996. Some other commonly used measures in international literature are also presented for comparisons. These other measures are the standard deviation of the age at death distribution *SD* (Manton et al, 1984), the related coefficient of variation *CV* (Nusselder and Mackenbach, 1996) defined as the ratio of the standard deviation to the mean, Keyfitz's *H* measuring the convexity of the survival curve known as entropy[2], and its numerator *NH* proposed by (Nusselder and Mackenbach, 1996). Entropy *H* is calculated by its discrete approximation with an initial cohort of one at the starting age.

The indices used in the analysis of non-Maori mortality compression are summarised in Table 11.1. Like *PD*, *SD* and *NH* are absolute measures and are suited to measure compression in its absolute sense. Similarly, *PR*, *CV* and *H* are ratios and are suited to measure compression in its relative sense.

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2 The entropy, *H*, was first constructed by Keyfitz (1977) to measure the elasticity of life expectancy to mortality reductions. It can be used for a variety of demographic studies, including measuring the convexity of the survivorship function. Its mathematical presentation:

$$H = \frac{-\int_0^{\omega} (\ln l_x)\, l_x \, dx}{\int_0^{\omega} l_x \, dx}$$

where: l_x is the survivorship function at age x with $l_0 = 1$
ω is the oldest age in the life table.

A discrete approximation of *H* is proposed by Nusselder and Mackenbach (1996):

$$H_x = \frac{\sum_n d_x * e_{x+0.5n}}{e_x}$$

where: ${}_nd_x$ is the proportion of deaths between age x and age x+n
e_x is life expectancy at age x

Table 11.1 Indices of compression of mortality used in the analysis

Statistical property	Absolute measure	Ratio
Empirical area under the survival curve	**PD** (person-years differential)	**PR** (person-years ratio)
Spread of the age at death distribution	**SD** (standard deviation)	**CV** (coefficient of variation)
Convexity of the survival curve	**NH** (numerator of the entropy)	**H** (entropy)

Sources: Compiled by the author

Compared with the more commonly used measures (ie. *SD*, *CV*, *NH* and *H*), those here developed (*PD* and *PR*) are superior because of their greater sensitivity to mortality movements at older ages. Instead of spreading attention to all parts of the survival curve, these indices are essentially oriented from the area showing the greatest distance from the rectangle, that is, older ages. Measures such as entropy (*H*) focus on the convexity of the overall curve but, as demonstrated in the following analysis, they may not be able to detect the sometimes erratic movements of the tail. Minor improvements in mortality would push further the age at death of survivor at the 99.5th percentile and hence the right boundary of the rectangle, and the entire column area above this minor extension of the tail would add significantly to *PD* and *PR*, yet *H* would not be sensitive to this. Only by measuring from this perspective can one give the full account of the effects of mortality movements at the very old ages. Another advantage offered by empirical measures like *PD* and *PR*, the average number of person-years required to produce a perfectly rectangularised survival curve and the percentage from perfect rectangularity, is that they have intuitively straightforward meanings.

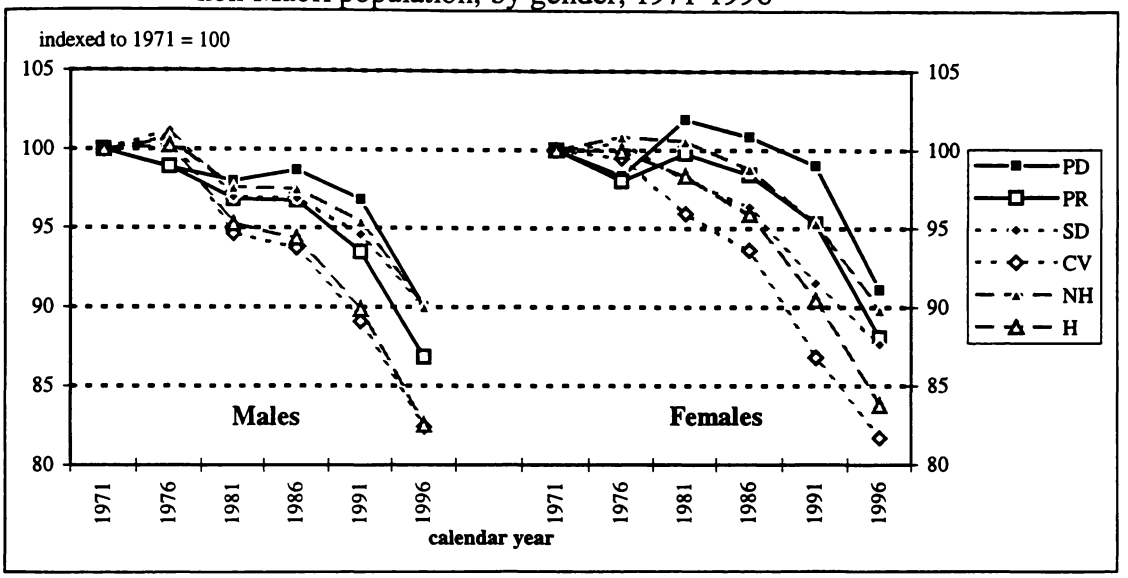
11.4 Results of Compression of Mortality Analysis

All six indices are calculated for non-Maori survival curves from complete life tables for the six census years 1971, 1976, 1981, 1986, 1991 and 1996. The calculations are done over two age bands: i) for all ages in Figure 11.5 and ii) for ages 65 years and over later in Figure 11.6. The aim of using these two age band is to separate the effects on the survival curve at older ages due to survivorship improvements at childhood and young adult ages. The results of the six indices calculated for both genders and by two age bands are included in Appendix Eight.

11.4.1 Measuring Mortality Compression, All Ages Included

To assist with the analysis of time series data, the results are indexed to the corresponding 1971 values. The indexed time series data for all ages are plotted in Figure 11.5. A systematic downward trend would suggest the increasing occurrence of mortality compression, and vice versa.

Figure 11.5 Indexed time series data of compression of mortality indices for all ages, non-Maori population, by gender, 1971-1996



Sources: Calculated by the author based on complete life tables constructed by Statistics New Zealand

Non-Maori Male Trends

For males, the two person-years based indices have shown a trend towards compression in both the absolute and the relative senses in the period under investigation. This observation is generally supported by the other four measures also included in the analysis. Moreover, With the exceptions of some minor perturbations between 1981 and 1991, the trend was reasonably smooth.

The forces of mortality compression between 1971 and 1981 have come from two different sources. To recall from Chapter Eight, between 1971 and 1976 some minor increases in mortality rates at adolescent and young adult ages occurred, as a continuation from the previous period. Mortality rates changed a little at the adult ages before 65 years, and declined moderately at ages 65 to 85 years, recuperating from the

last period. Mortality rates at ages 85 years and over had, however, continued their increase from the last period. This lack of movement at younger ages and possible deteriorations at more advanced ages produced a slight drop in *PD* and *PR*. The worsening of survivorship at ages 15 to 30 years which favoured an expansion effect had unduly influenced the other four indices, and was principally responsible for the slight increases in their time series trends.

Between 1976 and 1981, considerable reductions in infant and childhood mortality took place. The previous trend of mortality increases at adolescent and young adult ages had stabilised and reversed. At the adult ages, the pace of mortality declines started to pick up at middle ages and spread across to more advanced ages. The changes at older adult ages would produce an expansion tendency, but were overwhelmed by mortality reductions at younger ages, especially below the age of 25 years, leading to a net compression effect. Changes in mortality at younger ages have again exerted greater influence on *SD*, *CV*, *H* and *NH* as evident in their much sharper fall compared to *PD* and *PR*.

A deceleration and a slight reversal in the compression trend was observed between 1981 and 1986. This change was mainly attributable to consistent mortality declines at almost all adult ages including the most advanced ages. The resurgence of minor mortality increases at teenage and young adult years also contributed to the flat net movement.

Mortality declines accelerated between 1986 and 1996. Declines were most impressive over the first quinquennium and started from a progressively younger age at around 60 years. Declines in infant mortality in the ten years were also substantial. Survivorship improvements at younger and middle to late adults ages pushed further up the survival curve to compensate for the rightward extension at advanced ages. This was reflected in a slightly sharper reduction in the *PR* trend compared to the *PD*. The accelerated compression trend between 1991 and 1996 was explained by mortality reductions up until the age of 85 years, followed by some minor increases at older ages after persistent declines over the previous fifteen years.

Non-Maori Female Trends

For females, a smooth trend toward compression emerged between 1981 and 1996. Prior to this time, however, some fluctuations in *PD* and *PR* trends occurred. These earlier fluctuating trends were in contrast to those shown by the other four measures. The reasons for these differences are given below.

The compression trend between 1971 and 1976 was largely explained by mortality declines in the adult years up until the 90s, but followed by stationary and minor increases at the most advanced ages. Mortality declines were most marked at ages 75 to 90 years, and were the largest of all inter-censal declines throughout the entire period under investigation (since 1971). These changes effectively pushed up the survival curve towards the top of the rectangle while leaving the right-hand tail mostly unchanged, thereby producing a net fall in *PD* and *PR*. As also experienced by young non-Maori males, mortality rates at teenage and young adult years increased. Although the increases in mortality rates at these ages were only small, their effects were significant enough to produce a net rise in the values of the other four indices.

From 1976 on mortality reductions began to appear at advanced ages. Between 1976 and 1981 some significant reductions were recorded at all ages at 85 years onwards. The immediate effect of this was to stretch the base of the rectangle, leading to a sharp reversal in the *PD* and *PR* trends. Other indices failed to reflect these dramatic changes at these very old ages mainly because only around 30 percent of the birth cohort were expected to survive to age 85 years, and less than five percent to age 95 years, and therefore mortality developments at these advanced ages were insignificant. As will be shown in the analysis that follows, by excluding the effects of mortality before the age of 65 years, changes at age 85 years and over become more evident in these other indices as well as in *PD* and *PR*.

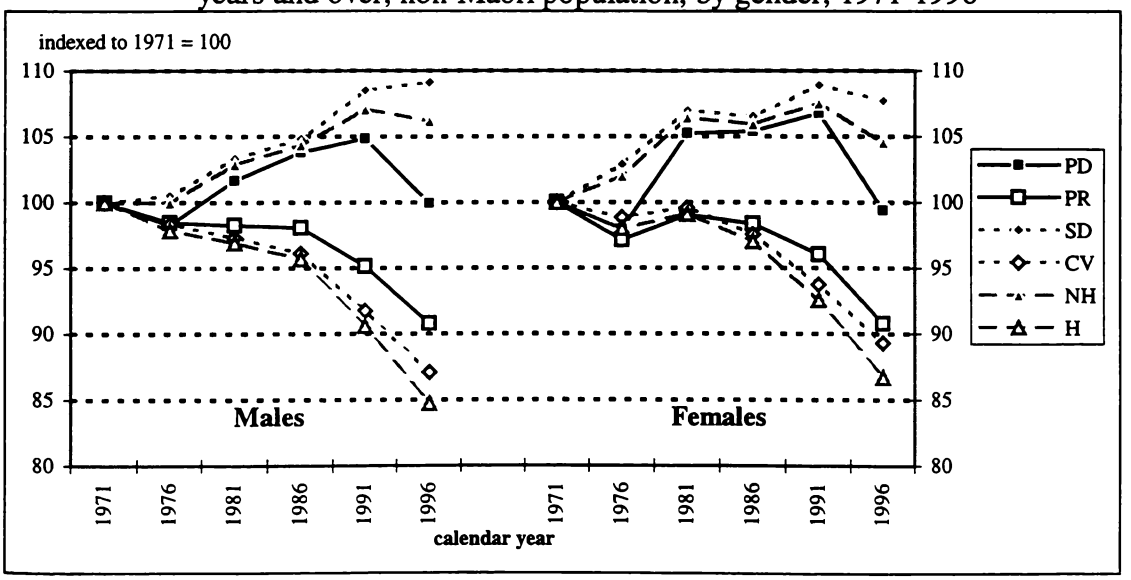
Mortality reductions continued between 1981 and 1986, but slowed considerably at older adult ages compared to the previous ten years. The survival curve resumed its trend towards compression. This trend accelerated between 1986 and 1996 despite some resurgence in mortality reductions at the very old ages. The main cause of this

acceleration was that mortality reductions became more visible at even younger ages of 50 years or younger, and had the effect of delaying to even older ages the exponential fall in the survival curve. The upward push of the survival curve thus counteracted the less significant rightward expansion. Substantial declines in the infant mortality rates especially those occurring between 1986 and 1991, and minor reversals between 1991 and 1996 in mortality declines at the most advanced ages also contributed positively to the compression trend.

11.4.2 Measuring Mortality Compression, Ages 65 Years and Over

Analyses presented thus far have not attempted to separate the effects due to mortality at infancy, childhood and young adult ages from those at older adult ages. As shown in the previous discussion, deaths at younger ages tend to exert unduly greater impact on most conventional life table derived mortality indices. To exclude this impact non-Maori survival curves are re-constructed with a starting age of 65 years. The time series of the six indices thus calculated are tabulated in Appendix Eight. These data are indexed to their respective 1971 values and presented in Figure 11.6.

Figure 11.6 Indexed time series data of compression of mortality indices for ages 65 years and over, non-Maori population, by gender, 1971-1996

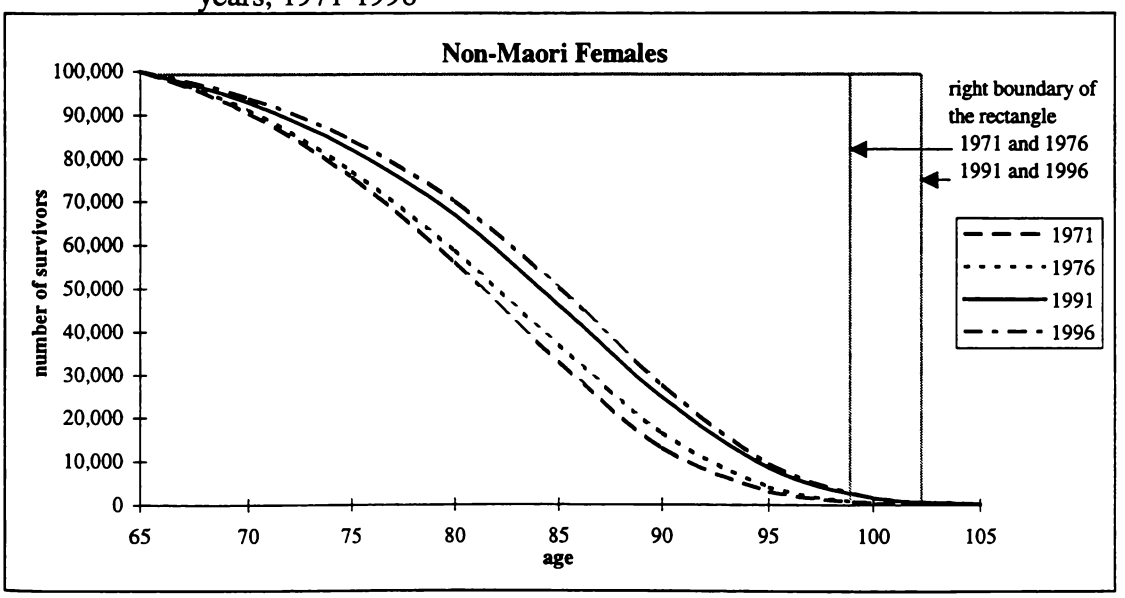


Sources: Calculated by the author based on complete life tables constructed by Statistics New Zealand

The most prominent feature to emerge from Figure 11.6 is the separate pathways of *PD* and *PR*; that is, trends in the absolute measure are contradictory to those suggested by the relative measure. This observation is also supported by other absolute and relative indices. While *PR* indicates a steady trend towards mortality compression at ages 65 years and over in its relative sense, *PD* fluctuates mainly in the expansion territory. Absolute compression occurred between 1971 and 1976 and between 1991 and 1996. In the fifteen years between 1976 and 1991, however, a distinct trend of absolute mortality expansion had been observed.

To explain the fluctuating trends of *PD* it is necessary to take a closer look at the survival curves during this period. The reconstructed survival curves starting from age 65 years for both genders exhibit the same characteristics, except that the female curves were further extended up the age scale reflecting the overall lower level of mortality of females. For reasons of simplicity, female survival curves for selected years, marking the turning points in the *PD* trends, are presented in Figure 11.7 and are used as the reference points in the following analysis. While the emphasis is on females, the general explanations about the changes in female survival curves are also applicable to the male survival curves.

Figure 11.7 Survival curves of non-Maori females at ages 65 years and over, selected years, 1971-1996



Source: Calculated by the author based on complete life tables constructed by Statistics New Zealand

In the two intervals where absolute mortality compression was detected (ie. between 1971 and 1976, and between 1991 and 1996), the survival curves expanded upward to create additional space in the middle of the late adult age range. Between 1971 and 1976, the largest gains in survivorship, as exemplified by the space between the survival curves, were at ages 85 to 90 years where mortality reduction was the greatest. Between 1991 and 1996, the largest gains in survivorship occurred at the relatively younger ages of 80 to 85 years. In both instances, the additional space gained did not persist to the tail end of the curves.

In contrast, survivorship improvements between 1976 and 1991 were significant across most ages. The momentum of survivorship improvements seemed to have lasted to the most advanced ages. This resulted in an almost parallel shift in the survival curves, and thus gave rise to absolute mortality expansion.

The persistent declines in mortality rates at older ages had stretched the base of the survival curve more to the right, and in doing so increased the space between the survival curve and the rectangle as illustrated in Figure 11.7. However, when viewed from the perspective of the entire rectangle, the space was becoming proportionately smaller as reflected in the downward trends of *PR*. Relative mortality compression was thus seen to have occurred.

To recapitulate the results of the analysis so far, between 1976 and 1991, the survival curves at age 65 years and over had gone through a simultaneous absolute expansion and relative compression. Mortality compression in both the absolute and the relative forms was found only in the most recent five year inter-censal interval.

These findings are somewhat expected. Early in this chapter, the analysis of trends in the quartile and percentile of age at death distribution has unveiled a pattern of stable spaces between the first quartile and the 99^{th} percentile throughout recent periods. The distance between the curves might have fluctuated little over the years, corresponding to periods of absolute compression or expansion; but their collective distance from age 0 had progressively increased, leading to relative compression. The implications of this key finding will be discussed in Section 11.5.

11.4.3 Comparing Results for the Two Age Bands

In the rest of this section, a brief comparison is made of the trends shown by the two age bands. For both genders, the PR trends at ages 65 years and over generally followed those for all ages in Figure 11.5. The trends in *PD*, however, had shown some deviations from that for all ages. For males, one major deviation was that between 1976 and 1981: the curve for all ages followed a trend of compression, and that for ages 65 years and over followed a trend of absolute expansion. It will be recalled from earlier analysis that the net compression effect was produced by mortality reductions at younger ages, which counterbalanced considerable mortality reductions at older ages. Once the former effect is screened out, the latter development of absolute mortality expansion thus become clearer.

Other minor deviations occurred between 1986 and 1991 for both males and females, whereby a compression trend was recorded for all ages but an absolute expansion was detected only at ages 65 years and over. To a lesser extent, a similar contrast was observed for females in the previous quinquennium between 1981 and 1986. The substantial decline in the infant mortality rates between 1986 and 1991 partly explained this deviation. The main reason is that, as mortality reductions started to spread progressively to mid adult ages of 50 years or younger, the compression trend observed earlier were being driven by mortality reductions at these ages before 65 years. After reconstructing the survival curve from age 65 years onwards, developments at the more advanced ages in the form of a rightward extension are given a wider scope in the analysis and has played a more important role in driving the measures.

The comparisons of trends for the two age bands have highlighted the importance of mortality changes at older ages. These comparisons also help to demonstrate the versatility of the two person-years based measures over the other indices. For example, in Figure 11.5, the expansion trends of *PD* and *PR* for females between 1976 and 1981 contradict sharply with trends in the other four indices. This is because the compression effects from substantial mortality reductions at younger ages had dominated in these other indices. Compared with Figure 11.6, when measured from age 65 years onwards, all other four indices, however, have reversed their previously downward trends to

follow that shown by *PD* and *PR*. For males similar comparisons can also be made for *SD* and *NH* in the same five year interval.

11.5 Discussion

The objective of the above analyses may appear to be of a purely technical nature: to determine whether or not non-Maori death patterns are becoming more compressed. However, it is not only of academic interest to understand the different competing estimates of human longevity, for the issue of mortality compression has far-reaching policy implications. Its outcome determines the size and the mix of the future older population, and, through interactions with the even less well known subject of morbidity, they together define the health status and health needs of future populations.

During the period 1971 to 1996 non-Maori survival curves were increasingly rectangularised relative to the already low mortality rates. When all ages are included mortality compression was found to have occurred in the last two decades in both its absolute and relative senses. Mortality was increasingly compressed into a narrower age range and into a smaller proportion of the life span.

The issue becomes less clear cut, however, when focusing on what is mainly chronic disease mortality at late adult ages of 65 years and over. Significant mortality reductions at these older ages during this period invariably challenge the notion that life expectancy is approaching its biological limit. The idea of the compression of mortality has been supported here only in its relative sense, that is, in relation to slowly improving life expectancy. A process of absolute mortality expansion at ages 65 years and over was operating for much of the period. This latter finding indicates that the life expectancy is not yet approaching its biological limit, and some further increases can be realistically expected.

The contrasting patterns of the absolute and relative measures is of interest. Developments at the tail of the survival curve are interesting in their own right, for they may have slowed the course of relative compression but not significantly enough to

change the course of longevity. However, while mortality reductions at older adult ages up to age 90 years are still occurring, mortality reductions at the more advanced ages have already started to show signs of slowing down. It can be shown mathematically that as long as the pace of relative compression exceeds the speed of expansion of the survival curve, as reflected by changes in life expectancy, the stage is set for the imminent absolute compression. In a low mortality environment like New Zealand, slowly increasing life expectancy and the persistent trend of relative compression lay the foundations for eventual absolute compression. Should the current trend persist, the question of an absolute compression becomes a case of "when" rather than "if", and to what extent.

To put this issue back into its broader historical context of the shift in age at death, the trends presented here suggest that the shift will continue at its current pace. Given that the majority of life table deaths are currently in the age range of 65 to 95 years for non-Maori males and 70 to 100 years for non-Maori females, there is still significant scope for further shifts. The pace of the shift is not expected to slow down until after the stage of absolute mortality compression is reached and has been under operation for some time. The sharp turn in the absolute trends between 1991 and 1996 is difficult to gauge. One cannot be sure whether it is the commencement of a new trend of absolute compression or a simple period effect.

To understand this issue it is necessary to turn to cohort analysis. A review of trends of non-Maori birth cohorts was carried out in Chapter Ten. An analysis of the possible forces of cohort mortality compression will be the focus of the next chapter (Twelve).

CHAPTER TWELVE
MEASURING MORTALITY COMPRESSION IN NON-MAORI COHORT LIFE TABLES

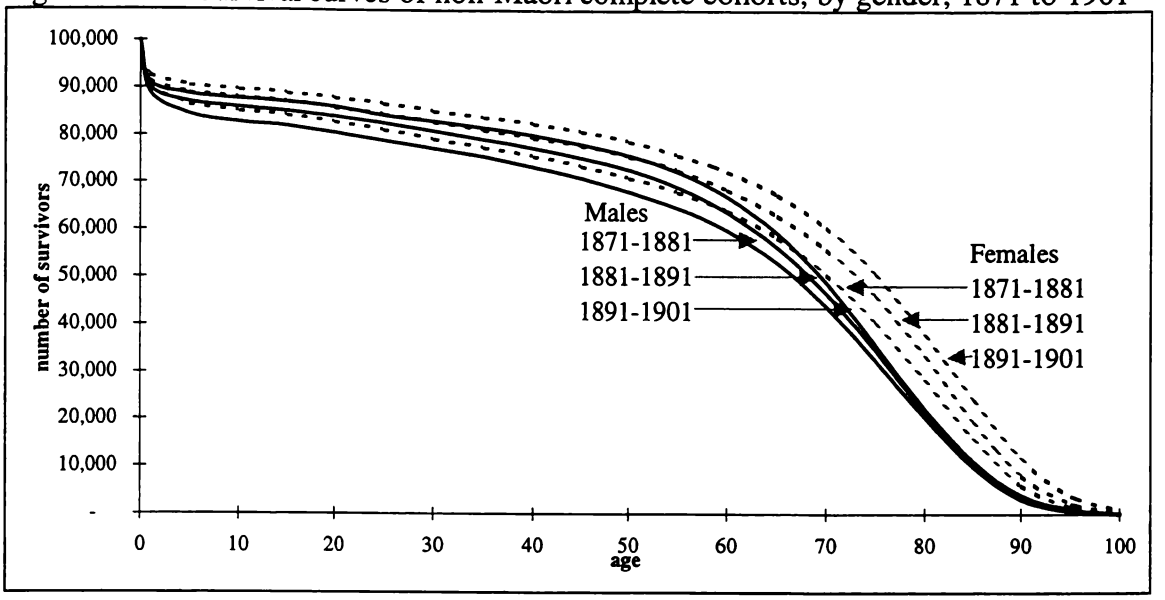
The long term non-Maori mortality changes as measured by synthetic life tables, manifested in the shift in age of death distribution, was studied in the previous chapter (Eleven) using as a framework the notion of the compression of mortality. Mortality compression measured by synthetic life tables reflects the collective mortality experience of a number of distinct birth cohorts and tends to imply a shortened time frame in which this phenomenon might have occurred. Since synthetic measures of mortality combine the experience of different cohorts, they are unlikely to give an accurate picture of any particular cohort. Moreover, synthetic measures fail to capture the way in which past experience, as long ago as the late nineteenth century, affect recent pattern of health as measured by mortality trends. A better approach, therefore, is to study each cohort separately and to make inter-cohort comparisons for any given life-cycle stages. This is the objective of the present chapter. The results of the analysis would help to grasp what can be expected in future mortality trends, from both cohort and, sometimes more conveniently, synthetic (cross-sectional) perspectives.

The possibility of cohort mortality compression is investigated in this chapter following the same methodological framework as that developed in Chapter Eleven to investigate mortality compression in recent synthetic life tables. Only non-Maori complete cohorts born between 1871 and 1901 are included in this analysis, whereas in Chapter Ten younger cohorts were also studied. Evidently, to analyse compression the research must be restricted to cohorts which are already at older ages.

12.1 Preliminary Analysis of Cohort Mortality Patterns and Trends

Non-Maori complete cohort survival curves first presented in Chapter Ten (Figure 10.1) are reproduced in Figure 12.1 for reference. The key features of these curves as described in the Chapter Ten were the classical horizontal "s" shape dominated by the initial sharp fall at infancy and early childhood, and convergence of male curves and parallel shifts in female curves at late adult ages.

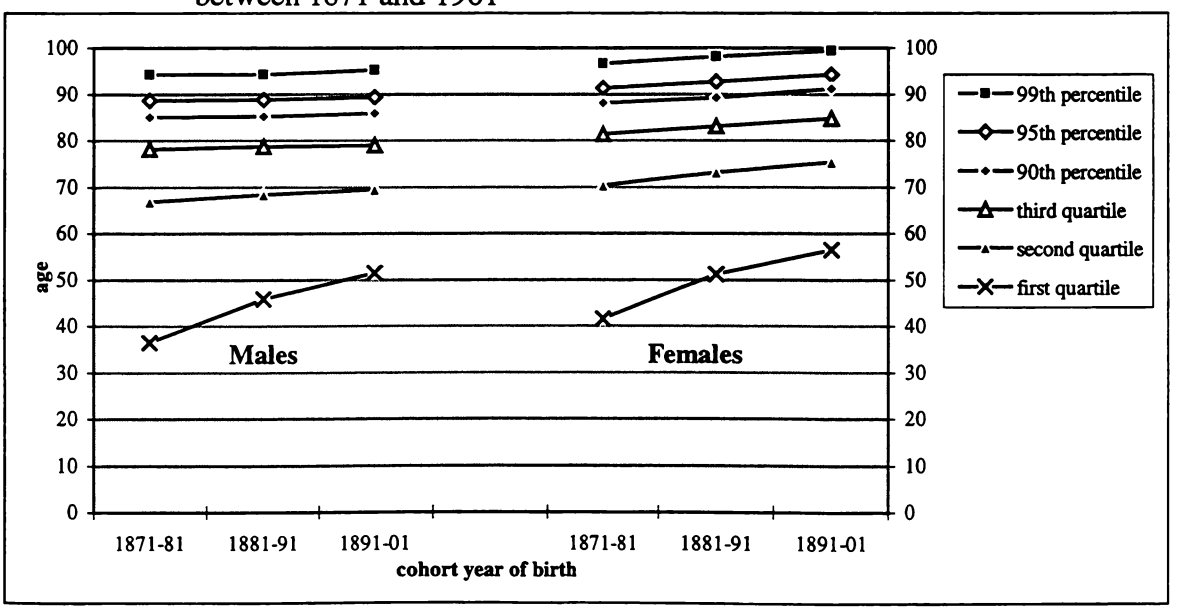
Figure 12.1 Survival curves of non-Maori complete cohorts, by gender, 1871 to 1901



Sources: Cohort life tables constructed by the author, based on published raw data and complete life tables constructed by Statistics New Zealand

Selected quartiles and percentiles of age at death distributions of the three complete cohorts are calculated and plotted in Figure 12.2. The brief trends shown there mirror those of the early synthetic life tables in Figures 11.2a and 11.2b when mortality reductions at younger ages tended to dominate overall mortality patterns.

Figure 12.2 Selected quartiles and percentiles of the age at death distribution ($_nd_x$) from cohort life tables, by gender, non-Maori complete cohorts born between 1871 and 1901



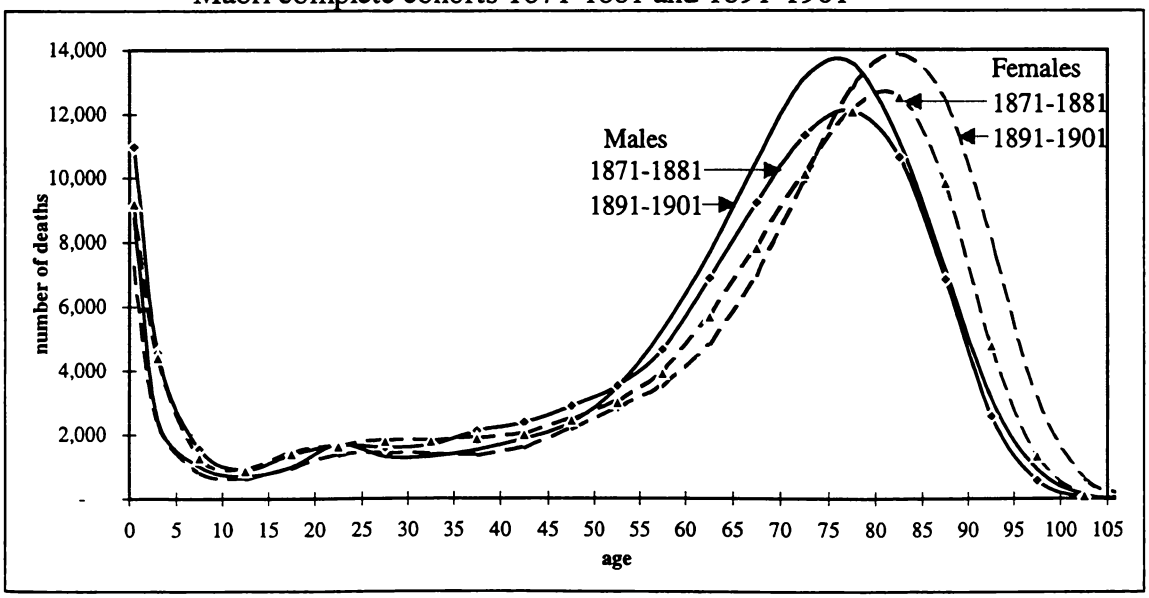
Sources: Cohort life tables constructed by the author, based on published raw data and complete life tables constructed by Statistics New Zealand

The trends in the first quartile of age at death displayed in Figure 12.2 are characteristic of rapid increases in survival at younger ages. The net effect is to compress the other three quartiles of death into an increasingly narrower age range. A narrowing age range between the second and the third quartiles of death is also visible.

The upper quartiles and percentiles appear to increase in tandem. Increases for females are much sharper than those for males, reflecting greater improvements in female survivorship. A more precise description of changes in age at death at the upper quartiles is, however, difficult to draw from this simple quartile and percentile analysis.

The age at death distributions for the 1871-1881 and the 1891-1901 cohorts, representing the two extremes of changes in age at death, are provided in Figure 12.3. Complete cohort patterns of age at death differ from those observed from synthetic life tables because of the more polarised distributions of cohorts. The two peaks in the cohort distributions capture both the high infant and early childhood mortality rates during the early to middle stages of the epidemiologic transition, as against the high concentration of death at late adult ages typical of a low mortality regime. In Figure 12.3, the first peak is as equally prominent as the second. Inter-cohort mortality reductions at the early years of life are significant for both genders.

Figure 12.3 Age at death distribution ($_nd_x$) from cohort life tables, by gender, non-Maori complete cohorts 1871-1881 and 1891-1901



Sources: Cohort life tables constructed by the author, based on published raw data and complete life tables constructed by Statistics New Zealand

At the more relevant adult age range, some interesting developments occurred. Not only does the peak of age at death distribution at the adult ages expand and thus produce counter effects to mortality compression, but expansions have gone in opposite directions for the two genders.

At ages 55 to 75 years the male 1891-1901 cohort suffered a greater number of deaths than their 1871-1881 counterparts. It appears that at older ages the 1891-1901 cohort had surrendered many of the mortality advantages that they accumulated at younger ages. Compared to the curve of the 1871-1881 cohort, the peak at adult ages of the 1891-1901 cohort has expanded leftwards, that is, a mortality expansion towards the lower age range. The ages at which this expansion occurred corresponded to the widespread male mortality deteriorations which took place during *the period of stagnation and deterioration in life expectancy trends.*

Female cohorts demonstrated a shift in the age at death distribution to the right due to the greater number of deaths occurring at ages 80 years and over. Survivorship advantages of the 1891-1901 female cohort over their 1871-1881 counterparts attained at younger ages had lasted to the end of life. The 1891-1901 curve is visibly lower than the 1871-1881 curve at all ages before 75 years. The parallel shift on the right hand side of the peak at ages 80 years and over correspond to the significant mortality reductions at older ages in the most recent *period of significant life expectancy increases.* Developments on the right hand side of the peak clearly dominate that on the left, and thus give rise to an expansion effect.

For both genders, though the peak of age at death distributions at adult ages has grown taller, the base of the peak has also grown wider, and is in different directions for the two genders. The taller peak and wider base combine to produce a net counter effect to mortality compression. In summary, mortality reductions at infancy tended to dominate overall changes when the level of mortality was high. Thus measures of mortality compression when all ages are included would tend to support its existence in the simplest form. At the more relevant adult age range, however, expansion effects were detected. The next section seeks to measure objectively the possibility of cohort mortality compression.

12.2 Measuring Cohort Mortality Compression

In this section, cohort mortality compression is measured for two age bands: (i) for all ages and (ii) for ages 65 years and over, to separate the effects on the survival curve from survivorship improvements at childhood and young adult ages. The six indices used for measuring synthetic mortality compression in Chapter Eleven are calculated for complete cohorts. These indices are person-years differentials (*PD*) and person-years ratio (*PR*) developed by the present author (see Section 11.2.2); standard deviation (*SD*) and the coefficient of variation (*CV*) of the age at death distribution; Keyfitz's entropy (*H*) and its numerator (*NH*). For *PD* and *PR* calculations, age at death of survivor at the 99.5th percentile from the cohort life table is set as the right hand boundary of the rectangle (see Figure 11.4)[1]. Statistical derivations of the six indices can be found in Chapter Eleven (see also Table 11.1). The numerical values of these indices are included in Appendix Nine.

12.2.1 Measuring Cohort Mortality Compression, All Ages Included

The six indices calculated for each cohort across it entire life-cycle are indexed to the respective 1871-1881 cohort values. The indexed cohort data for all ages are plotted in Figure 12.4. A systematic downward trend would suggest the occurrence of mortality compression, and vice versa.

Strong compression effect across the entire life-cycle emerge from *PD* and *PR* trends, and are supported by all other indices. As expected, these compression trends are predominantly driven by mortality declines at infancy and childhood. In the case of males, the compression effect is further reinforced by mortality deteriorations at mid to late adult ages which helps to accelerate the vertical drop of the survival curve in Figure 12.1.

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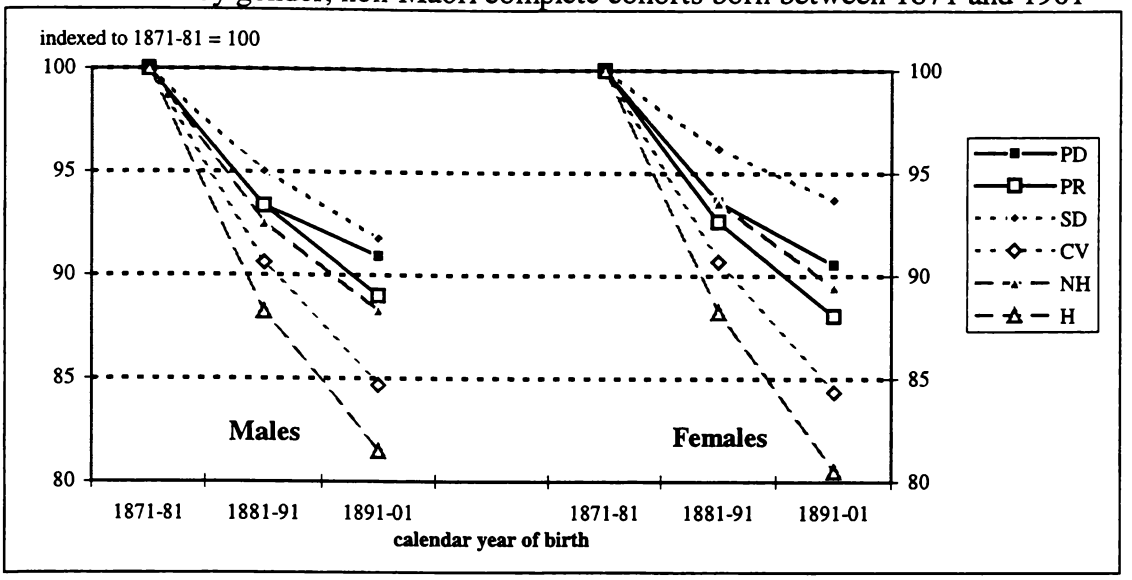
[1] As is done in the synthetic analysis, the chosen value is tested for validity and sensitivity with other upper age cut-off points and results are found satisfactory.

Figure 12.4 Indexed time series data of compression of mortality indices for all ages, by gender, non-Maori complete cohorts born between 1871 and 1901



Sources: Calculated by the author based on published raw data and complete life tables constructed by Statistics New Zealand

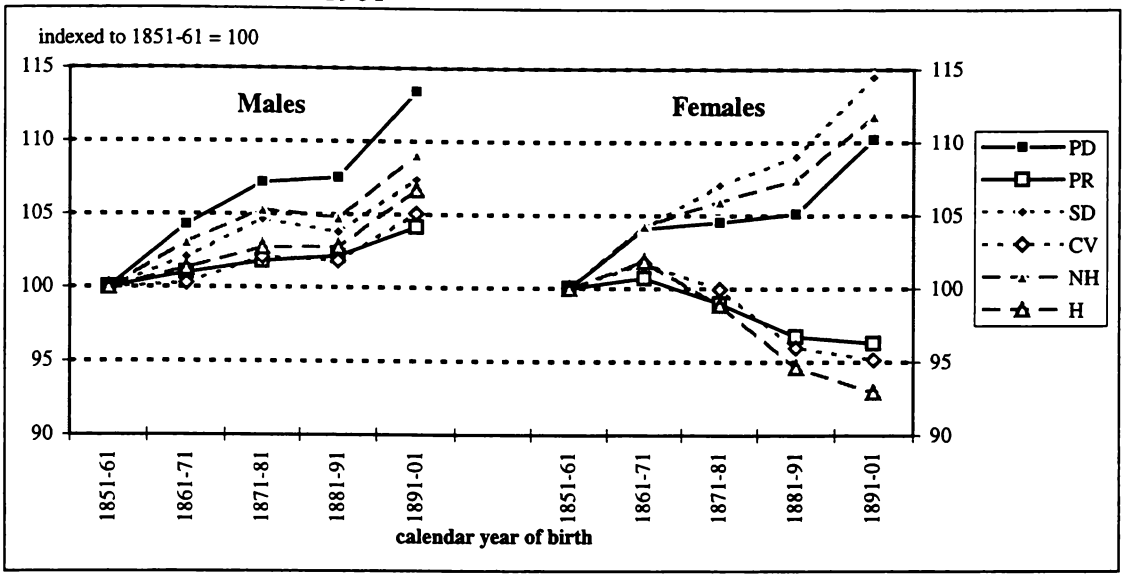
The 1891-1901 cohort had benefited from mortality reductions at ages 80 years and over in the early part of *the period of significant life expectancy increases*. These changes have brought about a rightward extension of the tail of the survival curve and act to decelerate the compression trend for both genders.

The *PD* and *PR* values calculated for the 1891-1901 cohort roughly correspond to values calculated for the 1906 synthetic life tables. This helps to highlight the impacts of mortality reductions at younger ages which feature prominently in both of these particular cohort and synthetic mortality patterns.

12.2.2 Measuring Cohort Mortality Compression, Ages 65 Years and Over

Turning attention to late adult age range, the six indices are calculated for cohort survival curves reconstructed at ages 65 years and over. To build up a longer time series data, two earlier cohorts born between 1851-1861 and 1861-1871 are also included. The time series data indexed to the 1851-1861 cohort values are presented in Figure 12.5. The numerical values of these indices can be found in Appendix Nine.

Figure 12.5 Indexed time series data of compression of mortality indices for ages 65 years and over, by gender, non-Maori complete cohorts born between 1871 and 1901



Sources: Calculated by the author based on published raw data and complete life tables constructed by Statistics New Zealand

After excluding mortality changes before the age of 65 years, the strong compression effects observed previously across all ages disappears. Male time series data indicate strong expansion trends, in both the absolute and the relative senses. For females, absolute expansion accompanying relative compression is detected, similar to the synthetic trends of the most recent period. For both genders, the relative trends are considerably weaker than the absolute trends. In general, the given pattern suggests an overall expansion effect at work, particularly between the last two cohorts in Figure 12.5.

To better understand the contrasting gender cohort trends, survival curves at ages 65 years and over for the five cohorts are reconstructed in Figures 12.6a and 12.6b with an initial cohort size of 100,000 at age 65 years. The most distinguishable feature of male cohort survival curves is the heavy clustering, even up to the very old ages. Changes in male cohort survivorship before the age of 85 years are barely discernible. This is consistent with the nearly flat synthetic trends in the probability of surviving from age 65 to 85 years between the 1920s and the 1970s in Figure 8.2a. At advanced ages, some significant inter-cohort survivorship improvements occur. However, with less than 20 percent of the original cohort at age 65 years survived to 85 years, the net effect of mortality reductions at these advanced ages is merely to extend the tail but not to shift

the survival curve as is the case for female cohorts. The combination of a lack of movement in the survival curves before age 85 years and an extension of the tail gives rise to a net expansion effect.

Figure 12.6a Survival curves of non-Maori male complete cohorts at ages 65 years and over, 1851 to 1901

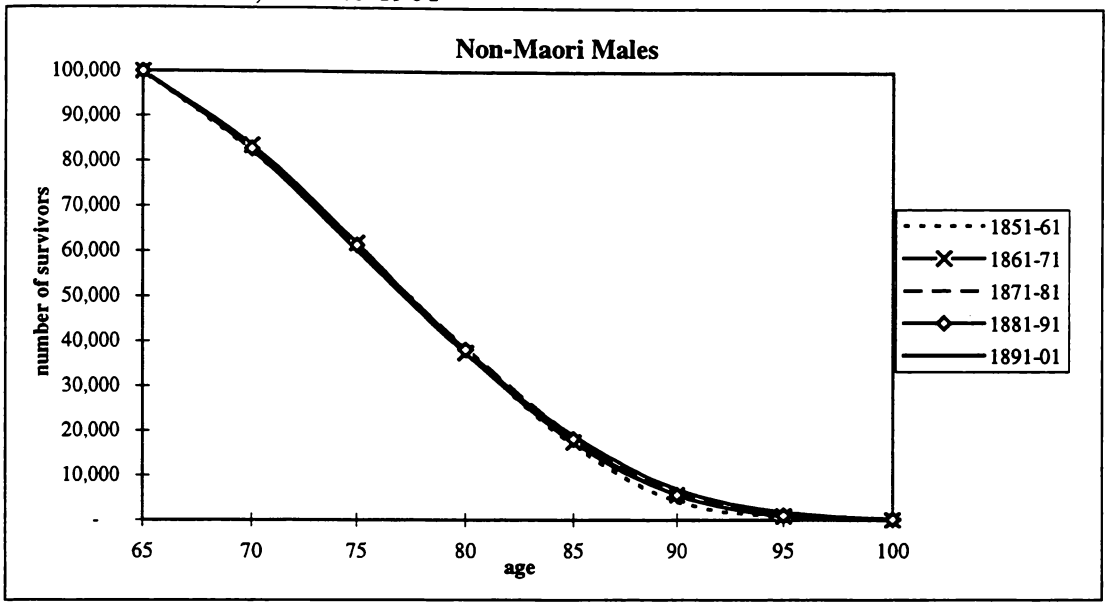
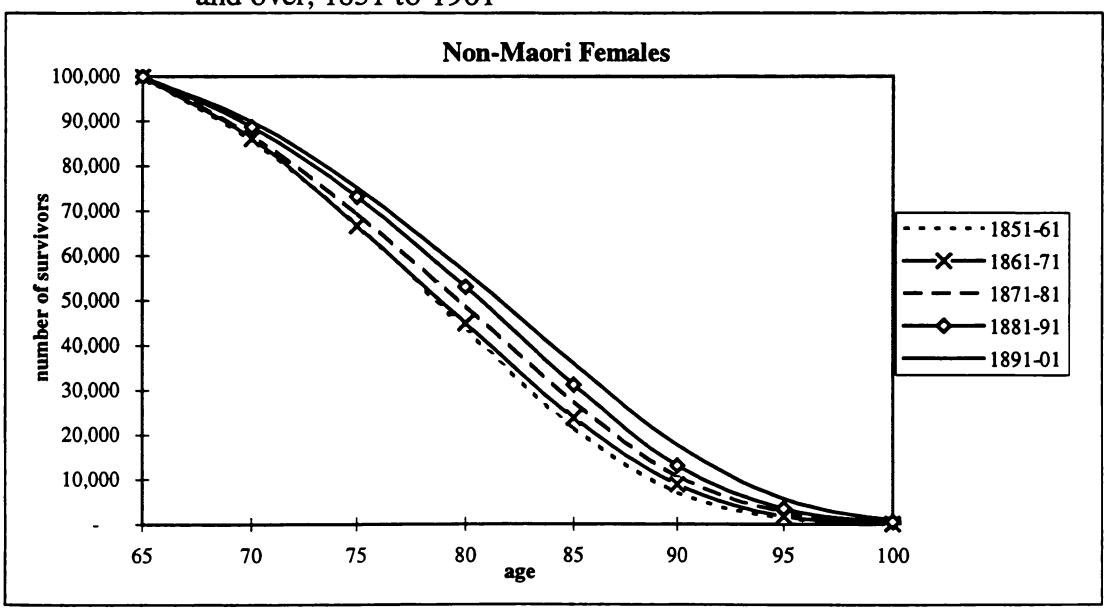


Figure 12.6b Survival curves of non-Maori female complete cohorts at ages 65 years and over, 1851 to 1901



Sources: Cohort life tables constructed by the author, based on published raw data and complete life tables constructed by Statistics New Zealand

Female cohorts have made steady improvements in survivorship, transforming their survival curves from concave to convex in shape. Inter-cohort survivorship improvements spread from the advanced to the less advanced ages. For example, the majority of inter-cohort changes between the 1851-1861 and the 1861-1871 cohorts, as measured by the vertical distance between the two curves, are at ages 85 years and over. This produce expansion effects in both the absolute and the relative senses. The age at which largest inter-cohort changes occur has shifted down the age spectrum for the two successive cohorts to produce relative compression.

For both genders, the 1891-1901 cohort exhibits the largest inter-cohort change in Figure 12.5. This is because mortality reductions at late adult ages occuring during *the period of significant life expectancy increases* has exerted considerable influences on the inter-cohort trends. Compared to the two earlier cohorts, the male 1891-1901 cohort exhibits a more decisive trend towards expansion in Figure 12.5, as a result of substantial mortality reductions at ages 75 years and over. Similarly, their female counterparts had benefited considerably from rapidly accelerating mortality reductions at ages 70 years and over (as also evident from Figure 12.6b), and hence trending convincingly towards mortality expansion.

The same analysis is extended by the present author using age 50 years instead of 65 years as the lower age cut-off point. This is done to include the bulk of the peak of age at death distribution which accounts for roughly three-quarters of all life table deaths. The results of the this further analysis, not reported here, reveal a pattern essentially a mirror image of that in Figure 11.5 and thus lends support for the validity to the above findings.

In summary, after removing the impact of mortality at younger ages and focusing on adult mortality, male cohort mortality patterns at adult ages reveal a trend towards expansion in both the absolute and the relative senses. Female cohort mortality patterns at the adult ages show absolute expansion but relative compression, in accordance with the synthetic patterns for the most recent years. The ramifications of these cohort trends at adult ages are discussed in the next section.

12.3 Discussion: Future Cohort Mortality Trends

Mortality patterns of non-Maori complete cohorts are dominated by the relatively high levels of infant and childhood mortality. Mortality rates at these early years of life were decreasing rapidly and had disproportionately affected overall mortality trends. Not surprisingly, when all ages are included, strong trends of mortality compression emerge in both the absolute and the relative senses. Mortality compression driven by inter-cohort mortality reductions at younger ages is expected to continue among the advanced cohorts and the younger generation of cohorts, although the scope for mortality reductions at younger ages has diminished progressively as seen in Figures 10.4a and 10.4b.

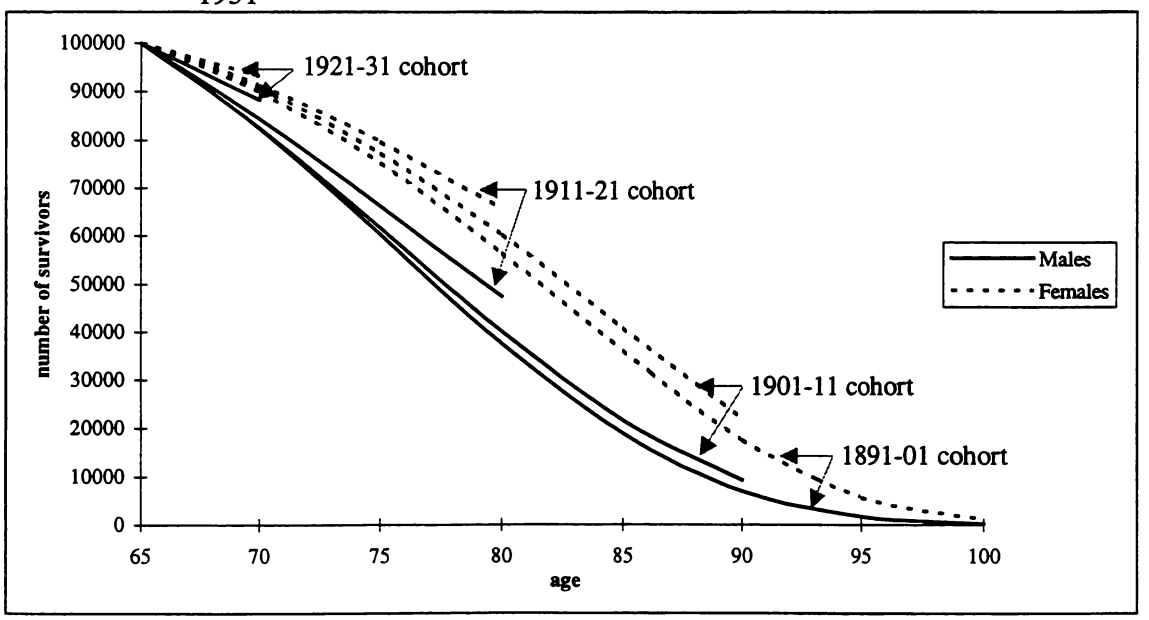
At the late adult age range, however, the hypothesis of a cohort mortality compression is not supported by data on the complete cohorts. Instead, convincing trends towards expansion for males, and females in an absolute sense, can be observed. For both genders, the exponentially increasing mortality rates operating at late adult ages produce steady falls in cohort survival curves. Curves in Figures 12.6a and 12.6b are triangular in shape before reaching ages 90 years and over. As a result of the triangular survival curves, non-Maori complete cohort mortality patterns at ages 65 years and over are dominated by movements of the tail. A minor extension of the tail can produce significant expansion effects. Only when the survival curves become more convex in shape, as start happening in the younger female cohorts of 1881-1891 and 1891-1901, the assessment of the effects of these tail movements can be kept in a more balanced perspective.

Female cohort mortality trends show some compression effects in a relative sense, albeit that this trend is somewhat weak. In contrast, male cohort mortality continue to expand relatively. Given the lower level of mortality of females compared to that of males, this seems to support the argument first put forward in Chapter Eleven that relative compression lays the foundations for absolute compression. Provided that the momentum of inter-cohort mortality reductions were to spread to ages 65 years and younger, as is already happening among the advanced cohorts, relative mortality compression at late adult ages might soon be achieved by male cohorts, and their female

counterparts could expect to consolidate their current relative trend. The question of whether or not absolute cohort mortality compression will occur in the immediate future, however, is more difficult to assess.

It will be recalled from Figures 10.4a and 10.4b that early signs of parallel shifts in the survival curves to the right for both genders were observed in the advanced cohorts and the younger generation of cohorts. These younger cohorts had benefited from significant mortality reductions at adult ages which took place during *the period of significant life expectancy increases*. Ages where the largest inter-cohort mortality declines occurred had progressively spread with the successive cohorts from more advanced ages to less advanced ages, from at ages 85 years and over for the 1901-1911 cohort to as early as age 65 years for the 1921-1931 cohort. This point is illustrated by the survival curves of the advanced cohorts in Figure 12.7, using the 1891-1901 cohort survival curve as a reference for comparisons.

Figure 12.7 Survival curves of non-Maori cohorts at ages 65 years and over, 1891 to 1931



Sources: Cohort life tables constructed by the author, based on published raw data and complete life tables constructed by Statistics New Zealand

12.3.1 Likely Future Developments

The gains from inter-cohort survivorship improvements for male 1901-1911 advanced cohort are concentrated at the very old ages of 85 years and over, as is reflected by the widening distance between the 1891-1901 and 1901-1911 cohort survival curves at these ages (see Figure 12.7). The likely net effect is to extend significantly the tail of the 1901-1911 curve and hence to produce mortality expansion in the absolute sense. Therefore, a short-lived acceleration in absolute expansion of male mortality, as already observed between the complete cohorts of 1881-1891 and 1891-1901, is expected to continue with the advanced cohort of 1901-1911. What is not shown in Figure 12.7, is that the survival curve of the older members of the male 1911-1921 cohort also exhibit characteristics of the accumulated survivorship gains at very old ages described above, and can expect to carry the momentum of absolute expansion a little further.

In contrast, among females of the 1901-1911 cohort and the two younger advance cohorts (1911-1921 and 1921-1931) for both genders, inter-cohort mortality declines had spread to younger ages of 65 years or under. These cohorts had made substantial gains in survivorship starting from middle adult ages, particularly the two younger male cohorts (see Figure 12.7). In the absence of an exceptional circumstance of dramatic inter-cohort mortality deterioration at older ages, a rightward shift of cohort survival curves will continue, possibly widening the distances from their immediate predecessors as age increases. Under this scenario, cohort survival curves will transform from a concave shape to a convex shape. This will produce a deceleration in absolute mortality expansion, or even an absolute compression in the case of female cohorts. This is because the upward push of the convex curve, magnified by the greater proportion of survivors at these early ages, would counteract the movements at the tail of the curve.

A stronger trend of relative compression is expected to accompany the above described changes when they occur. This is because the size of the inherent rectangle is becoming increasingly larger, with the area above the survival curve at the initial ages being quickly filled up by the convex curve. The increase in the size of the rectangle and the area under the curve will more than compensate for the extension of the tail.

In short, for the advanced cohorts, the trend of absolute mortality expansion at late adult ages is expected to decelerate, and reverse among female cohorts. Accompanying this will be a more defined trend for cohorts towards relative mortality compression.

Considerable variations in cohort mortality patterns analysed in this and earlier chapters underline marked inter-cohort differentials in terms of accumulated exposure to risk, and in terms of carrying risk at earlier ages forward to later ages. As each cohort has been subject to a different mix of decrement and survival at each life-cycle stage, one could infer that there are varying levels of inter-cohort exposure to risk, and that cohort heterogeneity varies between cohorts. This last-mentioned concept underpins the rationale for the analyses in the next two chapters (Thirteen and Fourteen).

The remaining chapters synthesise the empirical and explanatory analyses of Parts Four and Five. They then employ this synthesis as a baseline for projection, the policy implications of which are reviewed in the final chapter.

PART SIX:

NON-MAORI HEALTH IN RETROSPECT AND PROSPECT

CHAPTER THIRTEEN
HISTORICAL TRENDS: TOWARDS A SYNTHESIS

In Parts Four and Five, both empirical and explanatory analyses have been presented. This chapter is designed to synthesise these analyses by two means. The first part of the present chapter provides an overall summary of the key findings in the detailed analyses so far of this thesis. In the second part of the chapter, a more quantitatively-based synthesis of non-Maori cohort mortality and morbidity brings together the key findings and explores some of their ramifications. Implications for policy are discussed in Chapter Fifteen.

13.1 Summary of Key Empirical Findings

This section summarises the key findings from Parts Four and Five of this thesis. It should be noted that these key findings are closely interrelated, although they have been grouped into five sub-sections for ease of presentation. The grouping and the order of the key findings do not necessarily adhere to the sequence of chapters in Parts Four and Five.

13.1.1 Shifts in the Force of Mortality

An analysis of the century long non-Maori mortality transition studied in Chapter Eight revealed some remarkable shifts in the force of mortality, from the young to the old. Changes in non-Maori mortality were viewed across by five successive periods, prior to 1931, 1931-1941, 1941-1956, 1956-1971, 1971-1996, each characterised by the dominant forces of mortality change operating at that time. In general, up until the middle of the twentieth century non-Maori mortality patterns were dominated by mortality reductions at younger ages. This concentration was due to the relatively high levels of mortality at younger ages and thus large scope for survivorship improvements. By the mid-1950s, non-Maori survivorship before age 45 years was already very high. Hence, despite persistent mortality declines at younger ages in the subsequent years, their overall impacts on mortality patterns were limited. By contrast, changes in mortality at

middle to late adult ages started to emerge as the major force and dominated overall non-Maori mortality patterns in the second half of the twentieth century. Stagnation and even deterioration in trends for mortality rates at older ages between 1956 and 1971, and significant reductions in the old age mortality rates between 1971 and 1996 characterised the last two major periods of non-Maori mortality changes studied in Chapter Eight.

The mechanism for this shift in the force of mortality was the changing disease profile, a shift from communicable diseases to chronic degenerative diseases, reflecting changes in the prevailing health conditions. The changing patterns of cause of mortality are re-visited later in the following sub-sections. Along with the shifts in the force of mortality to older ages, the scope for and the momentum of further mortality reductions became limited. Currently, increases in life expectancy are becoming increasingly dependent on mortality movements at older ages where the majority of death already occur. As a direct result, the focus for population health both in analytical and policy terms has also shifted to the upper age spectrum.

13.1.2 Cohort Effects in Mortality

An important issue emerging from the analysis in Chapter Eight and later explored in Chapters Ten and Twelve are the effects of cohort differentials on mortality change. It was argued throughout Parts Two and Three of this thesis that cohort mortality experiences later in life are determined in part by the form of mortality and morbidity exposure that cohort have experienced earlier in their lives. Section 13.2 of this chapter is devoted to a synthesis of cohort mortality and morbidity experience.

Marked cohort effects were detected for non-Maori complete (ie. older than 100 years of age) cohorts born in the 30 years before and around the turn of the twentieth century. Mortality patterns for these cohorts were characterised by trade-offs between enhanced early survivorship and longevity later in their lives, reminiscent of what is postulated in key dimensions of the evolutionary theory of senescence reviewed in Chapter Three. Male complete cohorts displayed a consistent pattern of significant gains in survivorship at younger ages but were penalised by systematic increases in mortality at middle to late

adult ages. Female complete cohorts exhibited a similar though less strong cohort effect, in which their survivorship improvements at younger ages were less marked but compensated by more moderate inter-cohort mortality fluctuations at older ages. Stagnation in female inter-cohort mortality trends at older ages and actual deterioration for males dominated the period between 1956 and 1971, termed in the text *the period of stagnation and deterioration in life expectancy trends.*

A reverse pattern of trade-offs was detected for non-Maori advanced cohorts born in the 30 years period between 1901 and 1931. For the male advanced cohorts, the general lack of mortality movements and some minor fluctuations at younger ages favoured some significant mortality reductions at late adult ages. Again, female advanced cohorts exhibited similar but more moderate inter-cohort mortality movements than had their male counterparts. Inter-cohort mortality reductions at late adult ages among the advance cohorts demarcated the period between 1971 and 1996, termed here *the period of significant life expectancy increases at older ages.*

The younger generation of cohorts born between 1931 and 1961 has made progressively fewer gain from mortality reductions at younger ages. However, inferring from the pattern of trade-offs demonstrated by their predecessors, mortality fluctuations at the early reproductive ages experienced by these youngest cohorts may help to propel an acceleration in mortality declines at the middle to late adult ages.

13.1.3 Contrasting Cohort and Synthetic Trends of Mortality Compression

The two methodological approaches taken here, synthetic and cohort, have yielded two sets of somewhat contrasting empirical results in terms of the hypothesis of mortality compression. Since cohort experiences of risk exposure are unique to any particular cohort, and synthetic measures collapse together the mortality experiences of different cohorts, and make assumptions about population heterogeneity, it is not surprising that cohort and synthetic analyses produce somewhat different results.

Results for the synthetic analysis over the most recent period between 1971 and 1996 in Chapter Eleven support the hypothesis of mortality compression in a relative sense, that is, in relation to an increasing life expectancy. Until 1991 survivorship curves at ages 65 years and over were expanding in absolute magnitude, though the most recent five year inter-censal interval suggests the occurrence of absolute compression at older adult ages.

Results of mortality analysis for complete cohorts born between 1871 and 1901 in Chapter Twelve lend limited support to the inferences made in Chapter Eleven from synthetic data. Female cohort mortality trends have shown some weak compression effects in a relative sense, while male cohort survivorship has continued to expand. However, looking further ahead, the trends of the advanced cohorts, born between 1901 and 1931, show that inter-cohort mortality declines had gradually spread from the very old to middle adult ages, and that the cohort survival curve was starting to transform from a triangular to a rectangular shape. A new trend of relative mortality compression can thus be expected for the advanced cohorts. This cohort inference will guide non-Maori mortality projections over the next 30 years in Chapter Fourteen.

13.1.4 Compression Effects of Disability and Hospital Utilisation

The next issue raised in this thesis was the impacts on survival of shifts in morbidity. Analyses of disability and hospital utilisation data suggest accelerated risks of morbidity by age from 60 years onwards. The age specific prevalence rate for morbidity at older ages, measured by either disability or by patterns of hospitalisation, are high. When viewed in relation to the level of mortality, however, some consistent patterns of morbidity compression effects have been revealed. Results for disability-based health expectancy indicate that the major part of life expectancy, around three-quarters, can be expected to be free of disability. The occurrence of disability is being compressed into a narrower time span and is becoming concentrated at older ages, especially for the more severe forms of disability. This latter finding is also confirmed by an experimental index custom designed for this thesis and termed hospitalisation expectancy, which measures for any given age the expected number of future hospital days per survivor. The future number of days when people could be expected to be in contact with public hospitals

accounted for a tiny fraction of the life-cycle. Moreover, even this duration declines exponentially with age after reaching the end of the reproductive years.

After being adjusted for the risk of dying, the probability of a survivor developing conditions requiring continuous assistance and/or hospitalisation is small at most ages. When measured at a population level, the time expected to be spent in states of poor health is confined to a fraction of future life expectancy. Since data on disability requiring continuous daily assistance (level three disability) and hospitalisation reflect the more severe forms of ill-health, it is not surprising that the two resultant measures, expectation of life with dependency for continuous daily assistance (the reciprocal of Active Life Expectancy) and hospitalisation expectancy, show strong correlations and both fit closely with the survival curve of the life tables. Thus the compression of more severe form of morbidity can be inferred from these cross-sectional measures.

Furthermore, there is strong evidence to support what at first seems paradoxical: a declining trend for hospitalisation expectancy over the period 1981 to 1996, in the face of an increasing trend of the hospital discharge rates. Hospitalisation expectancy at all ages reduced by half over that 15 year period due to sharp reductions in the average duration of hospital stays. This was particularly the case at ages 65 years and over, at which age range the greatest increases in the hospital discharge rates occurred.

The significance of this recent decline in hospitalisation expectancies is that it not only coincided with steady increases in the hospital discharge rates, but also coincided with significant reductions in levels of mortality at older ages. The supposed escalation in hospital utilisation during the period, as reflected by increases in the hospital discharge rates, discloses only one side of the story. More frequent hospital contacts were counterbalanced by much shorter spells for hospital stays. After adjusting for the declining levels of mortality, the effects of an increased incidence for hospital contacts were more than off-set by the considerably shorter duration of hospital stay. The net result is a compression of morbidity of the most severe form accompanying a trend of improving life expectancy.

This is not to argue that levels of mild to moderate disability were being compressed — regretfully our data permit us neither to confirm nor to reject this hypothesis. But these results do suggest that new methods of treatment may permit earlier discharge from hospital, and thus from the most extreme forms of disability, a shift from continuous to intermittent care, and perhaps from a situation of very limited quality of life (ie. bedridden in a hospital and dependent on others) to one with a rather better quality of life and some limited degree of independence. Clearly this finding is of major significance for health policy, and will be revisited later in this thesis.

Also unfortunately, the available data do not support an analysis of cohort trends in hospitalisation expectancy. Nevertheless a limited exploration of aspects of cohort hospital utilisation is attempted in Section 13.2 later in this chapter.

13.1.5 Contrasting Mortality and Morbidity Profiles

From the available historical data on public hospital discharges, sharp rises in the hospital discharge rates over the last half century were in direct contrast to declining levels of mortality. An increasing rate of hospital discharge in relation to chronic morbidity was found in association with increases in life expectancy. Mortality and morbidity have thus seemed to have moved in opposite directions, when morbidity is viewed from the dimensions of either the incidence of hospital discharges or the prevalence of chronic morbidity. However, as mentioned above, by aggregating the duration of hospitalisation and combining it with the levels of mortality, the resultant measure of hospitalisation expectancy indicates a significantly different picture. As a result, hospitalisation expectancy has shown a most definite decreasing trend between 1981 and 1996, in line with the decreasing levels of mortality.

The issue of whether or not levels of mortality and morbidity are converging or diverging remains unclear. It is indisputable that there are diverging trends in the patterns of causes of mortality and in the causes of morbidity. While causes of mortality are heavily concentrated around two principal categories: cancers and cardiovascular diseases, the

causes of morbidity as measured by public hospital discharge data reveal a more even spread across the major cause groups.

In 1996, cancer and cardiovascular deaths account for around 70 percent of all deaths after controlling for population age structure, and close to 80 percent at age 65 years and over. The impact of communicable diseases on mortality have receded significantly from a quarter of all deaths a century ago to just over 10 percent in 1996. In contrast, the two leading causes of death, cancers and cardiovascular diseases, account for less than a quarter of all hospital discharges in 1996, even after controlling for the age structure, and for 40 percent or less at ages 65 years and over. Accidents are the leading cause of hospitalisation. Moreover, the discharge rates for communicable diseases rival those for cardiovascular diseases. Other chronic or non-fatal causes such as diseases of the digestive system, and, for females, diseases of the genitourinary system are more prevalent than cancers.

The contrasting patterns of cause of mortality and cause of morbidity highlight the difficulties in making simultaneous approaches to the two seemingly inter-related phenomena. A simple interpretation may be that non-fatal chronic degenerative conditions are less likely to kill, and therefore sustain a high level of prevalence in the population. The question remains, however, whether or not mortality reductions benefit frail individuals who suffer from a range of non-fatal chronic conditions, but who are not likely to die from these conditions.

13.2 A Synthesis of Cohort Effects of Mortality and Morbidity

A considerable proportion of this study has been devoted to a cohort analysis, setting this thesis apart from most conventional cross-sectionally based studies. As noted earlier in Chapter Three, in the absence of panel-type longitudinal micro-level data on determinants of mortality and morbidity which would otherwise allow empirical modelling of individual heterogeneity and thus of frailty, macro-level cohort mortality and morbidity patterns and trends can be understood as population level ramifications of individual heterogeneity. Cohort level modelling therefore translates the individual

probabilistic process of frailty into the more readily measured factor of population heterogeneity.

This section attempts a macro-level synthesis of cohort determinants of mortality, and the relationship of this with prior patterns of exposure to morbidity. A range of macro-level analytical processes are developed for this purpose, ranging from simple correlation analysis of cohort mortality at different life-cycle stages to an extended life table modelling of parameters in cohort heterogeneity.

13.2.1 Quantifying the Cohort Effects of Mortality

Trade-offs between improved survivorship at younger ages and increased mortality at middle to late adult ages is the central theme which emerged from the mortality analysis of non-Maori complete cohorts. Cohort effects in mortality not only feature in gender specific mortality patterns and trends, but also exhibit in gender differentials as summarised in Section 13.1.2 above. In an exploratory attempt to quantify the cohort effects in mortality described above, in Figure 13.1 for both genders and for all three complete cohorts by 10 years intervals, mortality rates in the first 15 years of life (the x-coordinate) are correlated with mortality rates at ages 55 and 80 years (the y-coordinate). Note that neither axis in Figure 13.1 has a zero base.

Male cohort mortality rates exhibit strongly negative correlations (with linear correlation coefficients of r = -0.998, extremely close to the theoretical maximum of minus one[1]). Male inter-cohort survivorship improvements in the first 15 years of life, represented by the x-axis and measured by the horizontal distance between data points, were highly significant and were marginally greater than that for female cohorts. Male cohort mortality rate at ages 55 to 80 years, represented by the y-axis, had worsened systematically, and thus gave rise to a negative linear correlation with mortality rate at ages from birth to 15 years. For male cohorts, the larger the gains in the probability of surviving the first 15 years of life, the greater the mortality increases at ages 55 to 80 years. For female complete cohorts, moderate mortality reductions at ages 55 to 80 years

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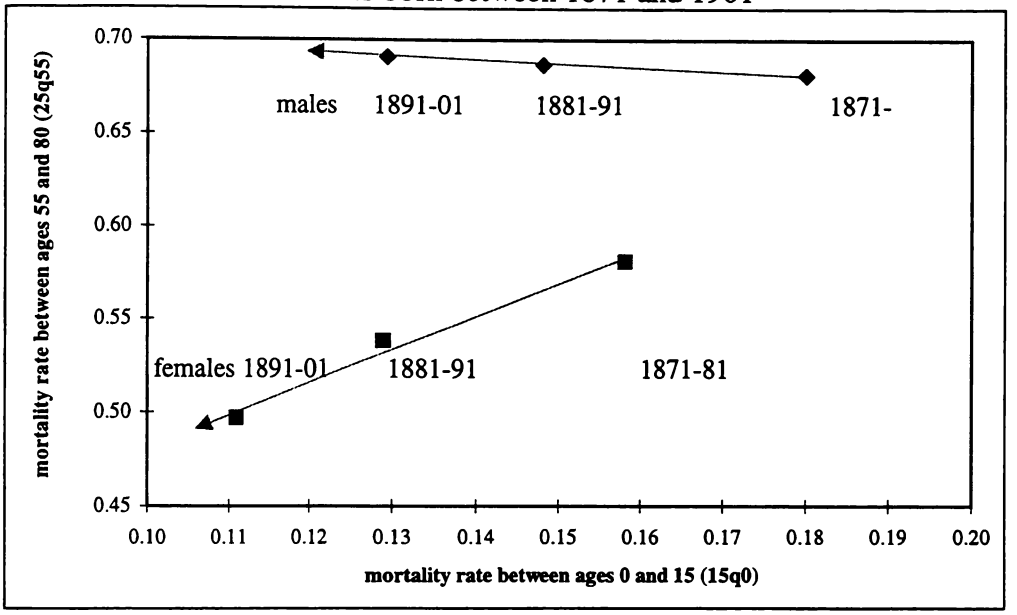
1 The high r-value is partly a result of the very small sample size of three.

compensated for their lesser survivorship improvements at younger ages than their male counterparts. This resulted in a more reduced but strongly positive correlation (with r = 0.992).

Figure 13.1 Correlating mortality rates between birth and age 15 years ($_{15}q_0$) and mortality rates between ages 55 and 80 years ($_{25}q_{55}$), by gender, non-Maori cohorts born between 1871 and 1901



Sources: Extracted from cohort life tables constructed by the author

The linear line fitted for male cohorts in Figure 13.1 has a slope of -0.19 and a y-intercept of 0.72, giving a linear equation: $y_m = -0.19x_m + 0.72$. The corresponding linear regression formula for female cohorts is: $y_f = 1.75x_f + 0.31$. The negative slope for male cohorts is statistically significant at a 0.05 significance level. This negative correlation confirms the operation of an inverse selectivity. If we assume that all cohorts had experienced the same exposure to risk at adulthood, then the survival of the less robust individuals was likely to be favoured by reduced mortality risks in their early years of life. Upon surviving to mid adult ages when the force of mortality accelerated, these individuals then began to suffer adverse mortality experiences in the form of inter-cohort mortality increases.

Male cohort effects in mortality noted above intensify when the focus of adult mortality is narrowed to ages 70 to 80 years. The strength of the negative correlation between mortality rate at ages from birth to 15 years (the x-coordinate) and mortality rate at ages 70 to 80 years (the y-coordinate) increases as reflected by its steeper slope ($y_m = -0.24x_m$

+ 0.57), indicating a heightened intensity of reverse selection effects at ages 70 to 80 years. In contrast, the strength of the correlation for female cohorts of the corresponding age ranges decreases ($y_f = 1.38x_f + 0.22$), which would suggest that the effects from mortality reductions early in life were less concentrated in this older 10 year age range.

This brief analysis above provides the context for further investigation in the following sub-sections. Results suggest that, for the majority of males, the negative correlation of mortality patterns shown in Figure 13.1 were mainly driven by inter-cohort mortality increases at ages 70 to 80 years. As will be shown in the next sub-section when looking at cohort mortality and morbidity by cause, the age range of 70 to 80 years is at the heart of a significant new stage of changes in cohort cause of death patterns.

13.2.2 An Extended Life Table Analysis by Causes of Mortality

In this sub-section, the synthesis of non-Maori cohort mortality determinants is extended to look at cohort mortality exposure by cause of death. Following the lead from the previous sub-section, the focus here is on cohort mortality patterns in the first 15 years of life and at middle to late adult ages. Cohort life tables constructed for the purpose of cohort mortality analysis in Chapter Ten are integrated with cause of death data to provide estimates of cause specific mortality rates and numbers of death by cause groups. The same groupings of cause of death as those used in previous chapters are employed here. These are: i) infectious diseases; ii) respiratory diseases; iii) degenerative diseases (sub-divided by cancers and cardiovascular diseases where data permit); iv) accidents; and v) others.

Mortality rates and the numbers of life table deaths by cause group are used as a proxy for estimating cohort exposure to communicable diseases (infectious and respiratory diseases) and non-communicable diseases. Following a multi-decrement life tables approach, the q_x and thus the d_x functions of cohort life tables are partitioned into the six major cause groups. For reasons of simplicity, it is assumed that the risks of dying from different causes are independent of one another, so that the sum of cause-specific q_x's is equal to the life table q_x. This assumption of independence of risk tends to underestimate

the results for most cause groups because of competing risks, although as shown by Mackenbach and colleagues (Mackenbach et al, 1999) the net effect is expected in most cases to be minor. It is likely that the under-estimation is worst for the largest group of cardiovascular diseases. Nevertheless, the results presented below should be regarded as indicative only.

Cohort probabilities of death (life table q_x's) by major cause group at ages from birth to 15 years, 55 to 70 years and 70 to 80 years, assuming independence of risk, are summarised in Table 13.1. All figures are rounded to third decimal points instead of more conventional fifth decimal points in order to avoid imputing a level of spurious accuracy implied by use of multiple decimal points. Cohort cause-specific rates at the advanced ages of 85 years and over are considered to be less reliable because of the relatively small number of survivors at these ages, because of age reporting errors in both the numerator and the denominator for older people, and because of the estimation procedures used to accommodate the restrictive reporting format in the raw data in published sources for 85 years and over for most calendar years.

At infancy and childhood, reductions in the infectious disease death rates were the single most important cause of inter-cohort mortality declines, accounting for around half of the declines for all causes. Though reductions in the degenerative disease death rates for children were equally impressive in percentage terms, they only contributed to a very small fraction of the total since degenerative diseases were much less lethal at these early ages. The decline in death rate for the category "others" might be attributable to improved diagnosis and better coding of causes.

Reductions in the risk of infectious diseases were most noticeable over the first five years of life, by more than two-thirds at infancy and by more than three-quarters at ages one to five years between the 1871-1881 and the 1891-1901 cohorts. In contrast, the respiratory disease death rates stayed almost unchanged at infancy, fluctuating but moving modestly downwards at ages one to five years. At ages five to 15 years, however, inter-cohort mortality reductions in the risk of infectious diseases seemed to have stopped with the 1881-1891 cohort. For both genders, after cutting these by more than half between the 1871-1881 and the 1881-1891 cohorts, the infectious disease death

rates at ages five to 15 years made little net movement between the 1881-1891 and the 1891-1901 cohorts. Reductions in the respiratory disease death rates at the corresponding ages became more noticeable, with the possible exception of the female 1881-1891 cohort. Changes in the risk of communicable diseases dominated mortality movements at ages five to 15 years.

Table 13.1 Cohort life table probability of death by major causes (cause-specific q_x's), non-Maori cohorts born between 1871 and 1901, by gender

	Males			Females		
	1871-81	1881-91	1891-01	1871-81	1881-91	1891-01
	Ages 0 to 15					
Infectious diseases	0.034	0.019	0.011	0.040	0.022	0.012
Respiratory diseases	0.028	0.026	0.025	0.021	0.020	0.019
Degenerative diseases	0.008	0.005	0.003	0.006	0.003	0.002
Accidents	0.011	0.009	0.009	0.008	0.007	0.006
Others	0.100	0.089	0.082	0.084	0.077	0.073
Total	0.180	0.148	0.129	0.158	0.129	0.111
	Ages 55 to 70					
Infectious diseases	0.016	0.015	0.006	0.006	0.004	0.002
Respiratory diseases	0.027	0.020	0.024	0.013	0.008	0.008
Degenerative diseases	0.212	0.234	0.248	0.182	0.175	0.156
Accidents	0.015	0.013	0.013	0.006	0.005	0.006
Others	0.050	0.040	0.031	0.048	0.036	0.027
Total	0.320	0.323	0.323	0.255	0.229	0.200
	Ages 70 to 80					
Infectious diseases	0.009	0.005	0.003	0.004	0.002	0.001
Respiratory diseases	0.037	0.062	0.067	0.020	0.028	0.024
Degenerative diseases	0.406	0.403	0.418	0.350	0.318	0.296
Cancers	*0.092*	*0.093*	*0.107*	*0.070*	*0.065*	*0.065*
Cardiovascular diseases	*0.314*	*0.310*	*0.311*	*0.280*	*0.254*	*0.231*
Accidents	0.013	0.012	0.013	0.009	0.011	0.012
Others	0.066	0.055	0.042	0.054	0.043	0.038
Total	0.531	0.538	0.544	0.437	0.401	0.371

Note: "others" include other chronic diseases (ICD chapters 3-6, 9, 10, 12 and 13), maternal and infant deaths (ICD chapters 11, 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Estimated by the author based on death rates for major cause groups compiled by I. Pool and from cause of death data published by Ministry of Health, and cohort life tables constructed by the author.

As a general observation, among the three complete cohorts the 1881-1891 cohort for both males and females seems to have been particularly favoured by declines in the risk of communicable diseases, as measured by the magnitudes of the reductions in their

inter-cohort age-specific mortality rates. As noted in Chapter Eight, these changes in cause specific mortality patterns over the first 15 years of life for the complete cohorts were primarily driven by the radical declines in fertility between 1876 and 1901 (see Pool 1985; 1994).

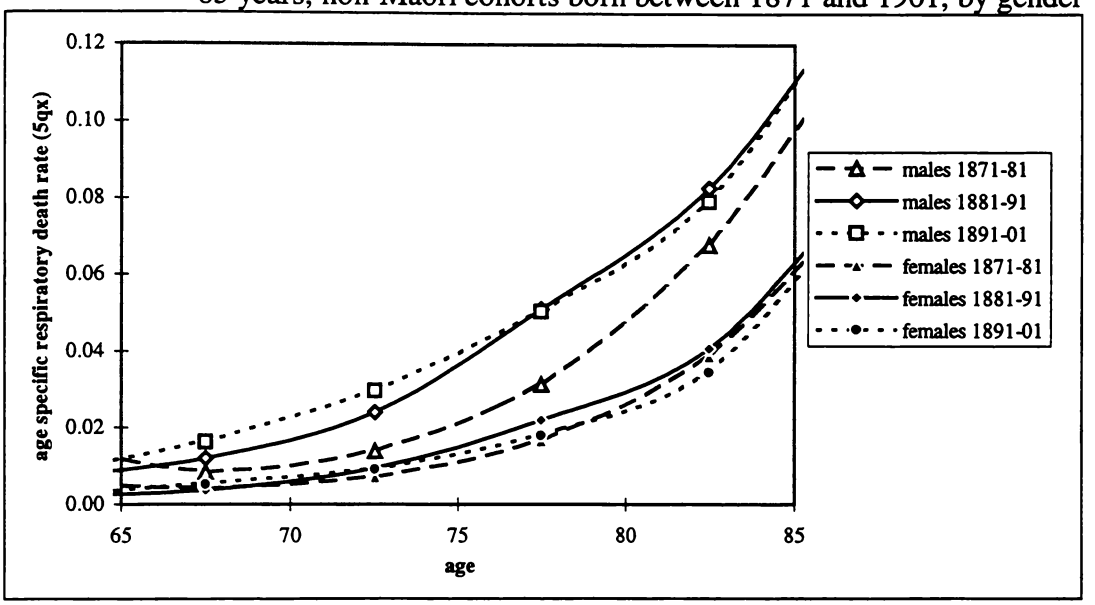
Table 13.1 highlights the point that male mortality increases at ages 55 to 80 years identified above were driven by two major causes at different ages. Increased male mortality risk between ages 55 and 70 years were mainly driven by an increased lethality of degenerative diseases. Age specific degenerative disease death rates increased systematically between the three successive cohorts while the corresponding rates for females decreased.

At ages 70 to 80 years, however, increases in the respiratory disease death rate occurred for both males and females. In fact, signs of increases in respiratory risk started to be observable as early as age 65 years. Increased pneumonia and chronic bronchitis deaths at these older ages were the primary concerns. The rise in the risk of death from respiratory cause was sharpest for the 1881-1891 cohort of both males and females, almost a reverse mirror image of inter-cohort mortality declines in the infectious diseases at childhood. Mortality risks from degenerative diseases tended to stabilise for males, while they trended downwards for females. The respective trends in degenerative diseases risk were mostly driven by trends in the cardiovascular disease death rates. The cancer death rates were generally steady for both genders, except for the 1891-1901 male cohorts which had shown a modest inter-cohort increase.

Overall, a pattern of increased risk of respiratory diseases was generally observable for both genders from age 65 years to the very old ages. The rises in risks of respiratory mortality mostly coincided with rises in cancer risks, though the former had a much steeper age trajectory. At these older ages, respiratory conditions were more likely to be a cause of death associated with the increased prevalence of chronic degenerative diseases. Unlike causes such as cancer, increases in the number of deaths due to respiratory diseases was less likely to be an artifact of improved diagnostic techniques and thus better certification over the years.

Age specific respiratory disease death rates at ages 65 and 85 years for the three complete cohorts are plotted in Figure 13.2. Ages before 65 years are not included since changes there were relatively minor. Increases in the cohort respiratory disease death rates were more marked for males than females. More importantly, the 1881-1891 cohort tended to be the worst affected, especially at more advanced ages.

Figure 13.2 Cohort age specific respiratory disease death rates between ages 65 and 85 years, non-Maori cohorts born between 1871 and 1901, by gender



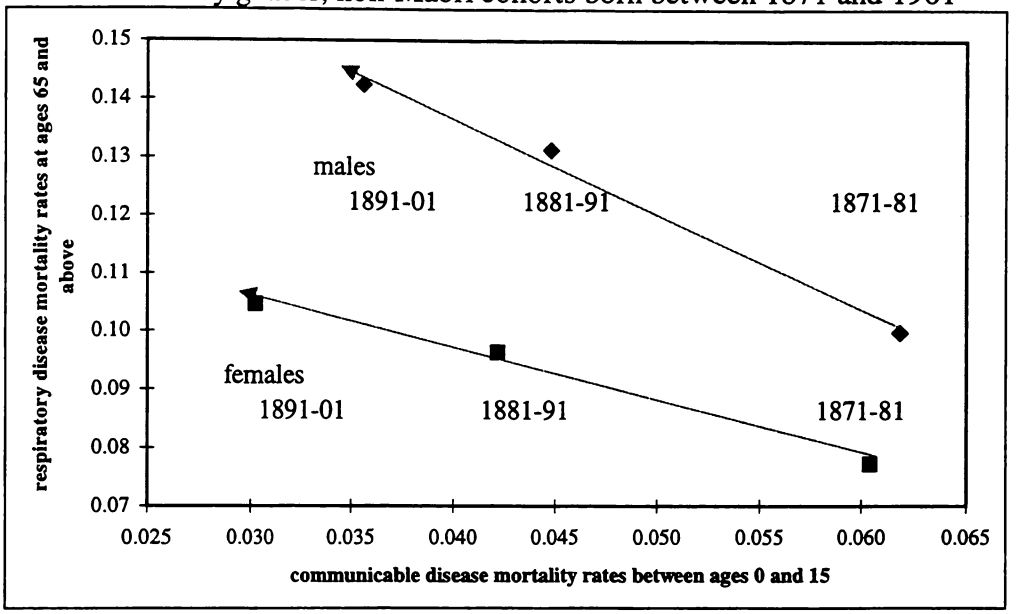
Sources: Estimated by the author based on death rates for major cause groups compiled by I. Pool and from cause of death data published by Ministry of Health.

The situation at older ages illustrated in Figure 13.2 is a complete reversal of the decreasing risk of communicable diseases at younger ages. For all three complete cohorts and for both genders, the 1881-1891 cohorts were particularly advantaged by inter-cohort mortality declines in communicable diseases at younger ages, but were more disadvantaged by increased respiratory risks at older ages. The fact that not only male, but also female cohorts exhibited the same pattern is highly significant. It reaffirms that cohort mortality effects were not an exclusively male phenomenon.

As a further confirmatory exercise, in Figure 13.3 the communicable disease death rates at ages from birth to 15 years, used here as a proxy for exposure to risk of communicable disease morbidity, are correlated with the respiratory disease death rates at ages 65 years and over. Again, neither axis in Figure 13.3 has a zero base. A remarkable but not unexpected strong negative correlation emerges for both males and females. The

respective linear equations are $y_m = -1.65x_m + 0.20$ for males and $y_f = -0.92x_f + 0.13$ for females (with correlation coefficients of -0.995 and -0.994 respectively). The negative slopes are considerably steeper than those shown in Figure 13.1. A strong inverse relationship is therefore established for both genders.

Figure 13.3 Correlating the communicable disease death rates between birth and age 15 years and the respiratory disease death rates at ages 65 years and over, by gender, non-Maori cohorts born between 1871 and 1901



Sources: Estimated by the author based on death rates for major cause groups compiled by I. Pool and from cause of death data published by Ministry of Health.

As pointed out earlier in this section, one possible explanation for the increased lethality of respiratory diseases at older ages is that respiratory diseases are not independent of non-communicable, but they are often sequelae recorded as a primary cause of death. Equally well, often cardiovascular diseases are sequelae of rheumatic fever in childhood, therefore an accompaniment of respiratory diseases. However, the nature of published data, tabulated by the underlying cause of death only but exclude an accompanying causes, precludes studies of dependence in causes of death. To investigate this point further, in the next sub-section hospital data will be drawn upon to see whether or not the rises at older ages in deaths due respiratory diseases as the underlying cause were independent, or a result, of increased prevalence of other non-communicable conditions.

13.2.3 Cohort Comparisons of Causes of Morbidity and Mortality

It would be enormously helpful to have cohort morbidity data sitting along side those on the exposure to risk of mortality profile. However, efforts to compile cohort morbidity risk profiles are serious hampered by the lack of historical data on morbidity. Public hospital discharge data started to be published in 1950 when the 1871-1881 cohort had already reached 70 to 80 years of age, which rules out the possibility of investigating cohort morbidity patterns at younger ages. The fact that the hospital discharges reporting format varied quite significantly over the years and that data by calendar years are not always available in a usable format (see Section 7.4.4) makes cohort comparisons even more difficult than the synthetic time series analysis earlier in Chapter Nine.

Notwithstanding these data problems, limited cohort specific data on hospital discharges are compiled for ages 70 to 80 years. This age range is chosen to coincide with the changes in the cohort mortality profile illustrated in Table 13.1, and can be shown by the available data. Cohort discharge rates (per 10,000) by causes at ages 70 to 80 years are presented in Table 13.2[2]. Hospital discharges at these ages accounted for almost 20 percent of discharges across all ages and over one-third at ages 65 years and over (see Figure 9.7).

An almost uniform trend of inter-cohort increases occurred across all major cause groups, with the possible exception of the category of "others". The steady increases in the hospital discharge rates at ages 70 to 80 years were generally in line with the increasing trend of hospital discharges in the second half of the twentieth century (see Chapter Nine), but not necessarily congruent with cohort mortality trends as shown in Table 13.1. For example, sharp rises in discharge rates for degenerative diseases, especially rates for cardiovascular diseases, sharply contrast with the stabilising or decreasing trends of mortality risk from degenerative diseases. This finding further supports the argument that the increased prevalence of chronic degenerative diseases was accompanied by the declining lethality of these diseases.

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[2] Hospital discharge rates in Table 13.2 are calculated as instantaneous rates of hospital discharge at any point in time within the age range, similar to the central death rates in the life table. They are not comparable with the probabilities in Table 13.1 which measure probabilities over a period of 10 or 15 years and are therefore significantly greater.

Table 13.2 Cohort public hospital discharge rates at ages 70 to 80, by major causes, non-Maori cohorts born between 1871 and 1901, by gender

	Males (per 10,000)			Females (per 10,000)		
	1871-81	1881-91	1891-01	1871-81	1881-91	1891-01
Infectious diseases	24	30	31	14	14	22
Respiratory diseases	156	197	259	77	80	92
Degenerative diseases	488	579	1036	309	393	668
Cancers	195	256	388	114	162	212
Cardiovascular diseases	294	323	648	195	231	457
Accidents	104	114	131	121	152	206
Others	756	929	834	497	682	632
Total	1529	1849	2291	1018	1321	1620

Note: "others" include other chronic diseases (ICD chapters 3-6, 9, 10, 12 and 13), maternal and infant deaths (ICD chapters 11, 14 and 15) and ill-defined causes (ICD chapter 16)

Sources: Estimated by the author from hospital discharge data published by Ministry of Health.

More interestingly, however, discharge rates for respiratory conditions do not show a dramatic rise. In particular, the respiratory discharge rates for the 1881-1891 male cohort seemed to be on a linear trajectory instead of an exponential increase as would be suggested by the corresponding sharp rise in the respiratory disease death rates. The combined patterns of mortality and hospital discharges suggest that the increased lethality of respiratory conditions were more likely to be found among members of the population with other causes co-existing, particularly with degenerative diseases as associated causes of ill-health and the ultimate death.

The hospitalisation expectancy analysis in Chapter Nine and the summary in Section 13.1.4 above showed that the hospital discharge rates cover the incidence dimension of illness but not the duration of morbidity which is vital for morbidity analysis. Data on public hospital average lengths of stay started to be published from early 1960s, but were poorly calibrated by age with an upper age group of 65 years and over. The extremely limited published data thus allow very little scope for the analysis of cohort differentials in hospital stays by finer age groups. The possibility of estimating cohort hospitalisation expectancies or similar measures is therefore discounted and thus cannot be pursued here.

13.2.4 An Empirical Extension to the Concept of Population Heterogeneity

The previous sub-sections establish empirically a cohort relationship in early exposure to the risk of communicable diseases and respiratory mortality risk at older ages. In this sub-section, an attempt will be made to integrate this important piece of empirical information with a macro-level theoretical model of population heterogeneity. The latter concept was discussed extensively in Chapters Three to Six of this thesis as a theoretical framework. That said, relatively little work has been done in the wider research arena in terms of empirical evaluations of the parameters of heterogeneity. The following exploratory work aims to give this theoretical concept an empirical face.

The simplest model of population heterogeneity discussed by Namboodiri and Suchindran (1987) is chosen for illustrative purposes. For this the actuarial function of the force of mortality at age x (μ_x), also known as the hazard function, can be written as:

$$\mu_x = Z * \mu_x' \quad \ldots\ldots\ldots (13.1)$$

where Z is a random variable with a positive value representing the amount of heterogeneity that operates in a multiplicative manner on the force of mortality, and μ_x' is the intrinsic force of mortality at age x common for all members of a population. While the observed force of mortality μ_x can be estimated from the life table, the intrinsic force of mortality μ_x' can only be theoretically defined so that the parameter Z in which we are interested cannot be evaluated empirically using equation 13.1.

Nevertheless, we can attempt an approximation. From earlier empirical evidence presented here the effects of heterogeneity later in life, represented by the parameter Z, can be thought of as inversely related to the accumulated exposure to the risk of communicable diseases at infancy and childhood. For both male and female cohorts, reduced levels of exposure at younger ages were linked to increased levels of heterogeneity at older ages, as manifested in the form of an increased risk of respiratory diseases. That is, an empirical relationship can be derived in the form of

$$Z = - \alpha / E + r \ldots\ldots\ldots (13.2)$$

where α is a multiplicative factor reflecting the strength of an inverse selection effect, E is exposure to the risk of communicable diseases at ages before 15 years, and r is the

residual effects not captured by the variable *E*. Through this empirical extension, the parameter *Z* lends itself to some empirical evaluation.

The above discussion demonstrates one of many possible way of looking at the theoretical issue of heterogeneity from an empirical perspective. More sophisticated theoretical derivations of the empirical evidence uncovered thus far would require further investigation and a much wider scope than the present study. What is clear, however, is that the levels of cohort heterogeneity have increased significantly over the three successive complete cohorts, as reflected by their declining exposure to the risk of communicable diseases at younger ages (see Table 13.1). The trends of increasing levels of cohort heterogeneity can be expected to continue among the advanced cohorts and the younger generation of cohorts, given their progressively reduced exposure to communicable diseases at younger ages. Under this scenario, variations in individual levels of frailty will be even larger among future older populations compared to those currently observed. The impacts of this increased cohort heterogeneity on the health status of the future older populations clearly have major implications for planning for future services.

13.3 Discussion: Formulating Assumptions for Projections

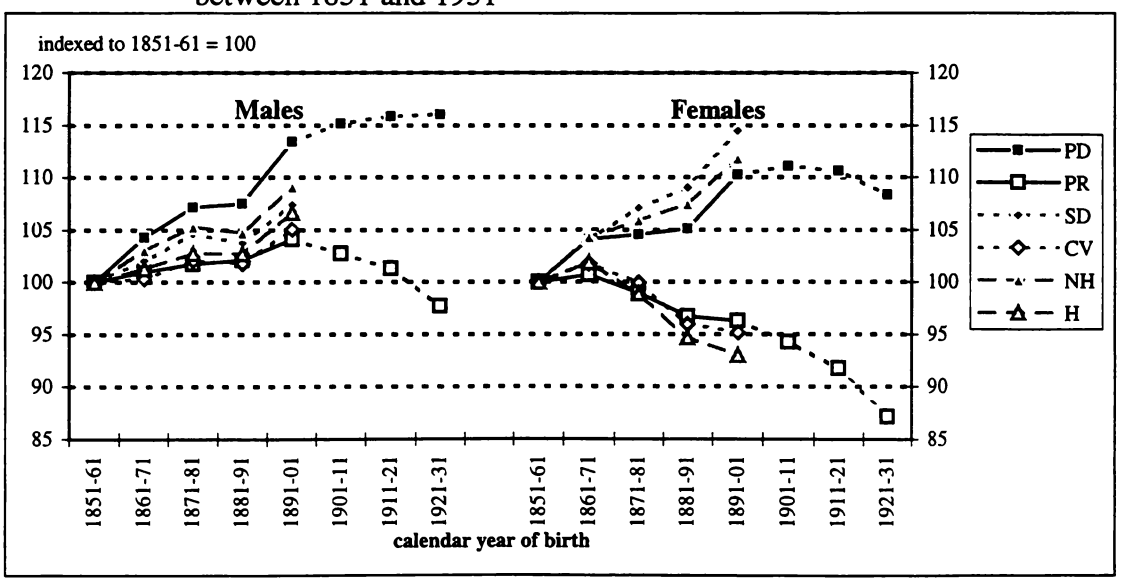
The question that now arise is how one can employ these syntheses in order to formulate assumptions for the projections that follow in the next chapter. Compared to the survival curves for the complete cohorts born between 1871 and 1901, survival curves for the advanced cohorts born between 1901 and 1931 and the younger generation of cohorts born between 1931 and 1961 are becoming decisively more rectangular due to substantial improvements which have already taken place at younger ages.

Non-Maori mortality projections in the next chapter (Fourteen) will be guided by a high level reasonably well documented inference drawn from the analyses presented in this thesis thus far, that cohort absolute mortality expansion at late adult ages will decelerate in both genders, and reverse among younger female cohorts. A necessary corollary to this projected absolute trend is a more conclusive trend of relative mortality

compression. The reasons behind this view are already discussed in Chapter Twelve. To briefly recapitulate, inter-cohort survivorship improvements among the advanced cohorts, which started at very old ages among the complete cohorts, had spread to the middle adult ages. The survivorship improvements among the advanced cohorts are expected to be fuelled in part by trade-offs from minor deteriorations in survival at young reproductive ages. The effect will be to expand the entire survival curve both upwards and to the right, thus making movements at the tail less significant. The trend of inter-cohort survivorship improvements at older ages is therefore projected to strengthen among the successive advanced cohorts before easing among the youngest generation of cohorts analysed historically here.

Projected compression trends of mortality at ages 65 years and over for the advanced cohorts, represented by the dotted *PD* and *PR* (person-years differentials and person-years ratio, area-based indices developed by the present author to measure mortality compression, see Section 11.3.2) lines, are shown in Figure 13.4, continuing from earlier trends of cohorts born between 1851 and 1901 as was shown in Figure 12.5.

Figure 13.4 Indexed time series and projected data of compression of mortality indices for ages 65 years and over, by gender, non-Maori complete cohorts born between 1851 and 1931



Sources: Calculated from cohort life tables constructed by the author

The projected trends of the advanced cohorts in Figure 13.4 closely approximate the most recent synthetic trends in Figure 11.6. In summary, an accelerating trend of relative

compression and a decelerating trend of relative expansion are expected in terms of future mortality changes of the advanced cohorts.

Limited by the available morbidity data, less sophisticated modelling of different scenarios of future morbidity developments in relation to projected mortality changes will be attempted in Chapter Fourteen. Patterns of disability observed in 1996/97 from the Disability Surveys as analysed in Chapter Nine will form the baseline. Various scenarios of future morbidity changes, capturing the essence of each of the leading theories of morbidity discussed in Chapter Five, will be simulated by changing the age specific prevalence rates of different levels of disability. The aim is for insights rather than accurate forecasting. Discussions of their policy implications in Chapter Fifteen will be structured accordingly.

CHAPTER FOURTEEN
SHIFTS IN FUTURE PATTERNS AND TRENDS IN NON-MAORI MORTALITY AND MORBIDITY

The analyses of non-Maori mortality and morbidity in the last six chapters have revealed some contrasting mortality and morbidity trends. While levels of mortality were declining, rates of hospitalisation increased steadily. Increases in the levels of hospitalisation might be explained by a range of factors not necessarily related to changes in population health status. However, an inverse association between population mortality rates and levels of ill-health has been observed historically in other developed countries dating back to the turn of the twentieth century (Alter and Riley, 1989; Riley, 1990b), and the more recent pandemic effects of morbidity have also had precedents among populations with lower levels of mortality than that for non-Maori. In short, a finding of an inverse association between non-Maori mortality and morbidity trends is not at all surprising.

Underlying the diverging mortality and morbidity trends is the contrasting pattern of causes of death and causes of ill-health as analysed in Chapters Eight and Nine. It can be concluded that frail individuals in the modern day non-Maori population may suffer from a range of chronic degenerative conditions which are serious in terms of disability but are often not life-threatening. The relationship between mortality and morbidity is therefore not a simple uni-directional one. Indeed, theories on compression of mortality do not necessarily presuppose that, at a population level, morbidity too will be compressed into the last few years of life.

The general lack of historical morbidity data in New Zealand hinders a thorough investigation into past morbidity trends and the issue of morbidity compression. International research to date generally supports a form of dynamic equilibrium in which severe morbidity rate tend to compress, and levels of mild to moderate morbidity stay mostly unchanged (Manton et al, 1997a; Robine et al, 1997; Romieu and Robine, 1997).

For non-Maori, synthetic and cohort mortality trends also show some interesting differences. At ages 65 years and over, while relative mortality compression and a more recent tendency towards absolute mortality compression were observed in the synthetic

life tables between 1971 and 1996, an expansion effect was the dominant feature in the cohort trends thus far. The reason for cohort mortality expansion is that significant mortality declines at late adult ages had started from very old ages of 90 years and over in earlier cohorts, affecting mainly the tail of the cohort survival curves. Given the wider spread of inter-cohort mortality reductions to less advanced ages, cohort trends of absolute mortality expansion are expected to decelerate in the short-term. A trend of cohort relative compression at older adult ages is also expected to follow soon among males and consolidate among females.

Non-Maori mortality and morbidity patterns and trends analysed thus far help to establish a solid platform for projecting future likely developments in these areas. In this chapter mortality trends at adult ages of the current non-Maori population up to 1996 are projected into the first quarter of the twenty-first century. Over three-quarters of all deaths in the population are now occurring at ages of 65 years and over, as a result of shifts in the age at death. Mortality rates before age 65 years have gone through dramatic declines historically and are currently at very low levels (see Figures 8.2a and 8.2b). At older ages, mortality reductions were only observed in the last few decades, but the momentum of these changes is expected to be carried through into the early twenty-first century. Given the wider scope for further mortality reductions at older ages compared to younger ages, major future mortality changes are expected to occur at geriatric ages and will dominate overall mortality trends. The focus of mortality projections carried out in this chapter is therefore set at late adult ages of 65 years and over.

The available long history of non-Maori mortality data analysed earlier in this thesis permits a cohort-based approach in projecting adult mortality trends. The main purpose of this exercise is to understand future mortality transition pathways for the older populations. The projections of mortality rates presented in this chapter differs from the practice for conventional demographic projections in a number of respects, some of which have practical advantages. By restricting to ages 65 years and over, one can avoid unnecessary assumptions about fertility. People who will be age 65 years or older in 2026 were already born in 1996, and their accumulated exposure to the risk of mortality over the 35 or more years prior to 1996 becomes a valuable input to formulating cohort-specific mortality assumptions. Age specific mortality rates are projected separately for

each five year cohort based on both mortality patterns at younger ages for that cohort and inter-cohort mortality changes. Given very little international migration at older ages, no specific allowance needs to be made for both in and out migration, implicitly assuming a closed non-Maori population. As the primary objective of this exercise is to project mortality trends, not the size of the future older population, the migration component is purposely omitted to avoid multiple sets of projection assumptions.

Later in this chapter, future likely scenarios of morbidity developments are discussed in relation to the projected mortality changes. The general lack of historical morbidity data in New Zealand hinders a thorough investigation into the past morbidity trends and inferences for the future. Future morbidity developments discussed in this chapter are necessarily tentative, based primarily on cross-sectional patterns in 1996 and recent trends in hospitalisation as analysed in Chapter Nine. The purpose is to elaborate the implications on health of the future older population under different scenarios of morbidity changes postulated by leading international research.

14.1 Non-Maori Mortality Projections, 1996-2026

In 2026, non-Maori born between 1956 and 1961, at the height of the baby-boom, will be the youngest birth cohort inside the projected age range of 65 years and over. Following the cohort-based approach, age specific survival rates by five-year age group ($_5p_x$'s) in the 30 years between 1996 and 2026 are projected separately for each five year cohort born between 1896 and 1961. The results of age specific survival rate projections incorporated into abridged cohort life tables are included in Appendix Seven. Later in this section, results for cohort-based mortality projections are re-organised to present a cross-sectional view for each census year.

14.1.1 Projected Cohort Survival Trends

Survival curves of non-Maori born between 1891 and 1961 were originally shown for 10 year intervals in Figures 10.5a and 10.5b. These curves are reproduced in Figures 14.1a

and 14.1b below and further broken down into five year birth cohorts. In addition, the projected survivorship over the next 30 years between 1996 and 2026 for each five year cohort is also included as the dotted lines. Numerical values of survivorship including projected figures can be found in Appendix Seven of abridged cohort life tables.

Figure 14.1a Observed and projected non-Maori male cohort survival curves, 1891-1961 birth cohorts by five year interval

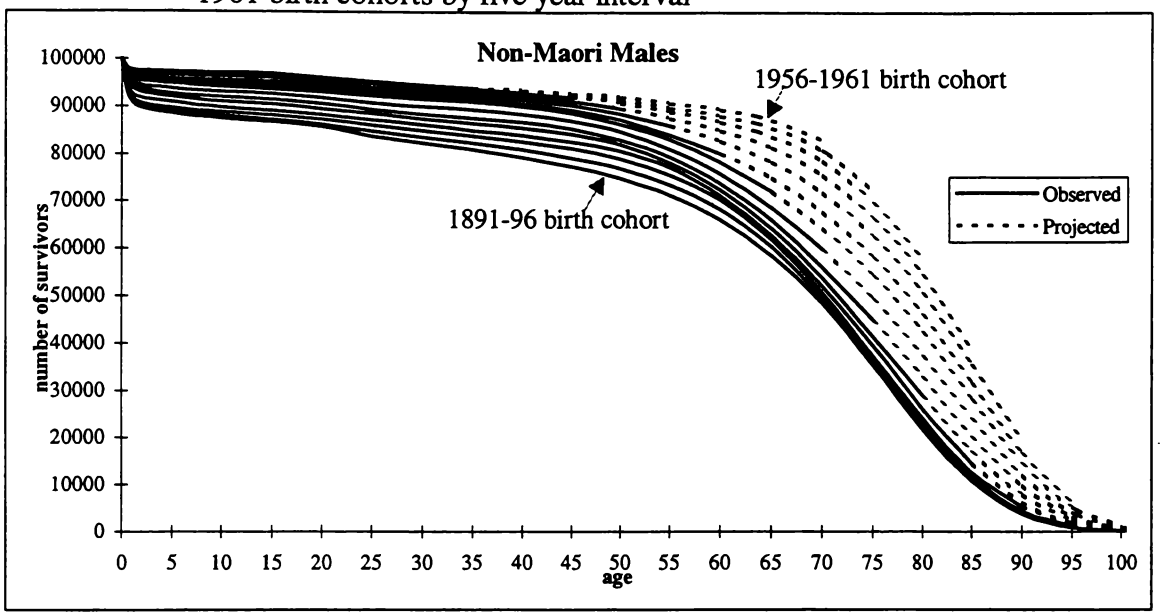
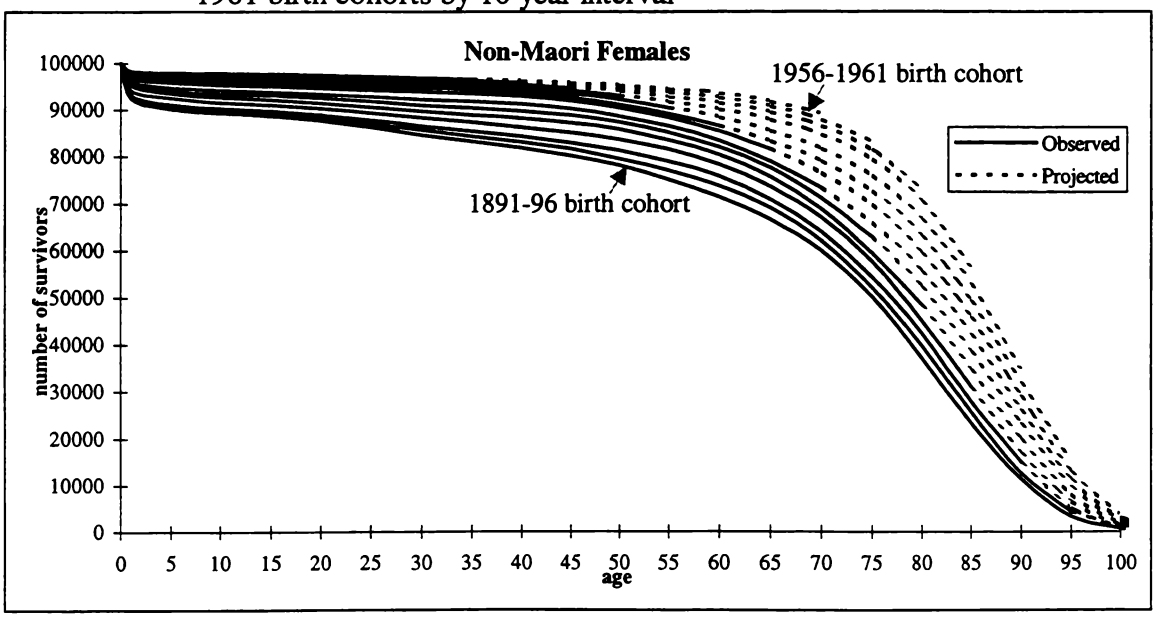


Figure 14.1b Observed and projected non-Maori female cohort survival curves, 1891-1961 birth cohorts by 10 year interval



Sources: Observed cohort survivorship - extracted from cohort life tables constructed by the author, based on published raw data and complete life tables constructed by Statistics New Zealand;
Projected cohort survivorship - from projections carried out by the author.

As a result of the persistent inter-cohort mortality reductions in the past, each cohort accumulated survivorship gains starting from increasingly younger ages. Each cohort survival curve has built up a noticeable space above that of its predecessor, and this represents the magnitude of survivorship gain. Not surprisingly, the projected survival path of most cohorts maintains the space accumulated thus far, and in the cases of younger cohorts, reinforces this with additional gains from further inter-cohort mortality reductions. In the next 30 years, the overall non-Maori cohort survival curves are expected to be more rectangular in shape as suggested by Figure 13.4. The top horizontal line will remain nearly flat well into the 60s age bracket. Under the projected mortality schema, the progressive upward push and rightward expansion of the entire curve clearly dominate changes at the upper age end. The exponential drop at older ages is expected to be delayed, and accompanied by increasingly steeper falls. These characteristics bear the hallmarks of highly rectangularised survival curves as is already starting to be observed in the latest synthetic trends.

Turning to the upper age spectrum, cohort survival curves at ages 65 years and over are reconstructed with an initial cohort of 100,000 at age 65 years. The truncated cohort survival curves, incorporating the results of projections, are presented in Figures 14.2a and 14.2b. The reconstruction of survival curves with age 65 years as the starting age and an identical initial size of 100,000, similar to those in Figures 12.6a and 12.6b, facilitates inter-cohort comparisons at older ages by assigning a mutual starting point for all cohorts. Figures 14.2a and 14.2b are not simple enlargements of selected ages of Figure 14.1a and 14.1b. The x-axis in Figures 14.2a and 14.2b, representing the age variable, is enlarged to amplify the contrasting cohort patterns. As the area in these graphs represents only about one-third of what is now a long life-cycle for most people in the population, these graphs thus exaggerate expansion effects at the expenses of compression.

For both genders a convex cohort survival curve at late adult ages is taking shape in Figures 14.2a and 14.2b. The upward expansion is particularly noticeable, ready to form the upper boundary of a rectangular curve. Significant mortality declines at older ages occurring during *the period of significant life expectancy increases* are evident in the distinctively separate individual cohort curves up to 1996, at the end of the solid lines.

Figure 14.2a Observed and projected non-Maori male cohort survival curves at ages 65 years and over, 1896-1956 birth cohorts by five year interval

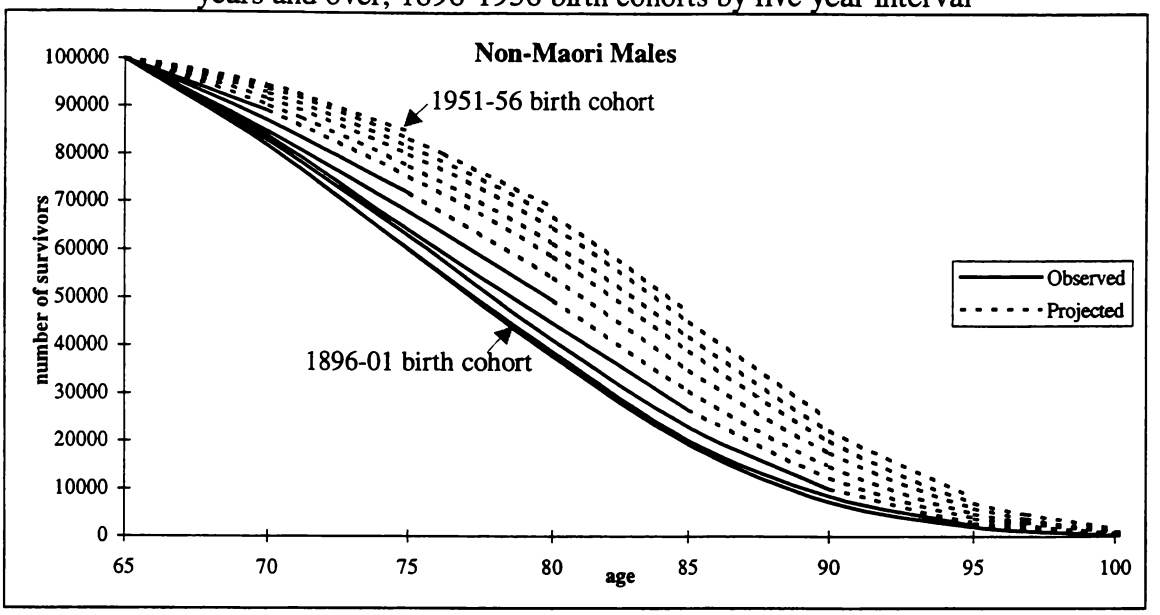
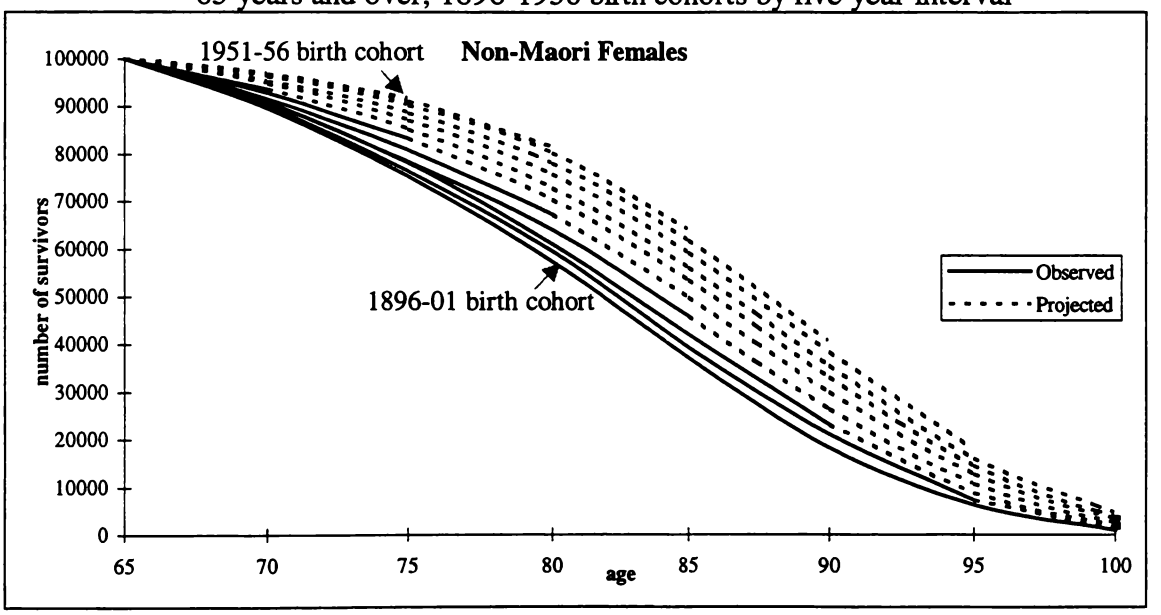


Figure 14.2b Observed and projected non-Maori female cohort survival curves at ages 65 years and over, 1896-1956 birth cohorts by five year interval



Sources: Observed cohort survivorship - extracted from cohort life tables constructed by the author, based on published raw data and complete life tables constructed by Statistics New Zealand;
Projected cohort survivorship - from projections carried out by the author.

The advanced cohorts are projected to continue a trend of inter-cohort mortality decline at late adult ages. Following the cohort pattern of trade-off between mortality reductions at younger and older ages first noted in Chapter Nine, a temporary accelerated mortality decline in the advanced cohorts is expected, which is fuelled by the general lack of

mortality movements and a minor increase earlier in life. Incorporating the projected mortality rates into survival curves for the older males born before 1911 and females born before 1906, with solid curves ending respectively at ages 90 years or above and 95 years or above, the survival advantages of these generations over their immediate predecessors are quickly diminishing due to the small number survivors remaining at the very old ages. This is despite substantial improvements in their age-specific survival rates projected at these ages. The largest projected inter-cohort survivorship improvements as can be seen in Figures 14.2a and 14.2b are from the younger advanced cohorts, including males born between 1911 and 1931 and females 1906 and 1931, with solid curves ending at ages 70 to 90 years and 70 to 95 years respectively. The momentum of inter-cohort mortality declines accumulated to date, depicted by the vertical distance between the solid curves, is projected to carry through into the twenty-first century. Further gains represented by the continuing widening of the dotted curves are expected from the accelerated inter-cohort mortality declines mentioned above.

For the younger generation of cohorts, for whom five cohorts by five year interval born between 1931 and 1956 are represented in Figures 14.2a and 14.2b, the momentum of inter-cohort mortality reductions is expected to lessen over time. This is simply because given the considerable inter-cohort mortality reductions that have already taken place, realistically, the trend cannot be expected to continue indefinitely at that pace. The situation will be very similar to the slowly decelerating decreases in the infant mortality rate in the last half century. Projected survival curves of the upcoming cohorts are therefore more densely distributed.

Consistent with the current trend, before age 90 years male cohorts are expected to make larger inter-cohort survivorship gains than females, as reflected by the more dispersed male dotted curves at those ages. The current lower level of survival rates of males will sustain their comparatively larger gains compared to that for females. At the upper age end, however, increases in female survivorship is projected to outpace those for males. At age 100 years, the tail of female survival curve is becoming noticeable thicker.

14.1.2 Projected Changes in Mortality Patterns

Cohort life expectancies at selected ages are estimated for the advanced cohorts in Table 14.1. The corresponding values for the youngest complete cohort born between 1896-1901 are also included for comparisons. Life expectancies at selected ages for complete cohorts by wider 10 year intervals were previously analysed in Chapter Ten (see Table 10.1 and Appendix Seven for the entire series of abridged cohort life tables by five year birth interval).

Table 14.1 Observed and projected life expectancy at selected ages of non-Maori cohorts born between 1896 and 1931, by gender

	1896-01	1901-06	1906-11	1911-16	1916-21	1921-26	1926-31
				Males			
At birth	60.7	61.9	63.2	64.7	66.2	68.3	70.5
At age 1	65.3	66.4	67.1	67.8	68.9	70.7	72.4
At age 15	54.3	55.0	55.6	56.2	57.1	58.5	60.0
At age 45	28.1	28.3	28.7	29.2	30.0	31.2	32.5
At age 65	13.0	13.1	13.7	14.3	15.2	16.2	17.1
At age 85	5.0	5.3	5.4	5.7	5.9	6.0	6.2
				Females			
At birth	66.1	67.5	69.6	71.4	73.3	74.9	76.9
At age 1	70.1	71.4	72.9	74.0	75.5	76.9	78.5
At age 15	58.9	60.0	61.3	62.3	63.6	64.7	65.9
At age 45	32.9	33.6	34.3	34.9	35.8	36.6	37.5
At age 65	16.8	17.3	17.8	18.4	19.3	20.1	20.7
At age 85	5.9	6.3	6.5	6.8	7.1	7.3	7.5

Sources: Estimated from cohort life tables constructed and mortality rates projected by the author

Systematic increases in life expectancy are expected for the advanced cohorts. Life expectancy at birth is projected to increase by around 10 years for cohorts born 30 years apart. It should be pointed out that the bulk of the magnitude of the expected increases have already been realised from cohort mortality experiences to date, and projections have only a minor bearing on life expectancy at birth, especially with the older cohorts. For example, assuming no changes in mortality rates at the projected ages of 70 years and over of the 1926-1931 cohorts from the 1921-1926 cohort levels, life expectancies at birth of the 1926-1931 cohort for both genders would reduce from the projected levels of 70.5 years for males and 76.9 years for females by less than half a year.

For all cohorts and for both genders, the projected life expectancy at birth is lower than the corresponding value at age one year. This is not surprising because, as noted in Chapter Eight, a higher life expectancy at birth than at age one year has only been achieved by populations with the very lowest mortality rates, and even for them typically shows up to date in synthetic rather than cohort tables. The cross-over in the synthetic trends was not achieved until 1971 for females and 1981 for males, roughly half a century later than when these cohorts had been born. However, this simple comparison helps to highlight the potential scopes for further increases in cohort life expectancy which can be expected from the youngest cohorts. To sustain this point, the projected life expectancy at birth of both genders of the 1926-1931 cohort are almost at the equivalent levels of that in the 1981 synthetic life table, and are just under five years below the levels in the 1996 synthetic life table.

At late adult ages, cohort life expectancy at age 65 years is projected to increase by four years for both genders between the youngest and the oldest advanced cohorts born 30 years apart. Similarly, cohort life expectancy at age 85 years is projected to increase between 1.2 to 1.6 years. The magnitudes of these expected increases need to be kept in perspective of the length of the time period over which these changes are expected to take place.

For male advanced cohorts, the rate of increase in life expectancy at late adult ages is expected to start slowly but pick up with the successive cohorts. The slower rate of increase between the older cohorts born before 1916 is a direct result of mortality fluctuations occurring during *the period of stagnation and deterioration in life expectancy trends*. The impacts of male adult mortality fluctuations become more evident when comparing inter-cohort life expectancy increases at different adult ages. Between the four successive cohorts of 1901-1906 to 1916-1921 inter-cohort life expectancy increases at age 65 years are at the same level as that at age 15 years, and are marginally greater than at age 45 years. This means that for these cohorts between ages 45 and 65 years male survivorship had deteriorated, off-setting survivorship gains made at other adult ages. A less marked pattern can also be found in female cohort life expectancy increases. The levels of inter-cohort life expectancy increase between the same four successive female cohorts are almost identical at ages 45 and 65 years.

Increases in female cohort life expectancy at adult ages are expected to be more evenly spread than those for males.

The substitution effects in the pattern of causes of death, between deaths due to infectious diseases and deaths due to degenerative, mainly cancers and cardiovascular, diseases, is expected to be replaced by across-the-broad reductions in all major causes of death among the advanced and the upcoming cohorts. For both genders, further significant reductions in the cardiovascular disease death rates at older ages are expected, though the pace of reduction may slow somewhat among the upcoming cohorts. Declines in the cancer death rates at older ages, which started only very recently as noted in Chapters Eight and Ten, are expected to build on the current slow pace and to accelerate a little among the upcoming cohorts when the older members reach the very old age group. The infectious disease death rates are expected to stay at the currently very low levels with diminishing scope for further significant reductions, even at the very old ages. Declines in the respiratory disease death rates started at around the same time as did decreases in the cancer death rates, but at a faster pace. At the upper age scale, the respiratory disease death rates are still high, even compared to the cancer death rates, and particularly among male cohorts. This therefore presents another major opportunity for future mortality reductions. Moderate declines are also expected in the accident and other death rates.

For the advanced cohorts, the proportional make up of the pattern of cause of death is expected to stay mostly unchanged as a result of across-the-broad declines. The percentage share of cancers and cardiovascular diseases is expected to increase marginally from the current 63 percent for males and 61 percent for females to around 65-70 percent. The percentage share of respiratory deaths is likely to stay at around 10 percent while shares for other cause groups drop marginally.

14.1.3 Future Synthetic Mortality Trends

Abridged synthetic life tables for ages 65 years and over by census years from 2001 to 2026 are constructed from cohort based mortality projections. Life expectancies at ages

65 and 85 years from 1986 to 2026, computed from synthetic life tables, are shown in Table 14.2 for comparisons. Over the next 30 years non-Maori life expectancy at age 65 years is expected to increase by 3.7 years for males and 3.5 years for females from their respective 1996 levels. The increases in synthetic life expectancy are lower than those in the cohort series (see Table 14.1), though the synthetic values are consistently higher because of persistent mortality declines at late adult ages. At age 85 years, the projected increases of 1.0 year for males and 1.3 years for females are very similar to those expected from the cohort trends. Life expectancy at age 85 years from the two series, cohort and synthetic, are this way because of the small number of survivors and the relatively high levels of mortality operating at these very old ages.

Table 14.2 Observed and projected non-Maori synthetic life expectancy at ages 65 and 85 years, by gender, by census years, 1986 to 2026

	Observed			Projected					
	1986	1991	1996	2001	2006	2011	2016	2021	2026
				Males					
At age 65	13.7	14.9	15.8	16.6	17.4	18.0	18.6	19.1	19.5
At age 85	4.6	5.0	5.1	5.3	5.5	5.7	5.8	6.0	6.1
				Females					
At age 65	17.5	18.6	19.3	20.1	20.8	21.5	22.0	22.5	22.8
At age 85	5.6	6.1	6.2	6.5	6.8	7.0	7.2	7.4	7.5

Sources: Calculated by the author from cohort mortality projections

It may seem paradoxical that for both genders, the projected gradual accelerating increases of cohort life expectancy at age 65 years in Table 14.1 produce a trend of gradual decelerating increases in the synthetic series in Table 14.2. This is because projected inter-cohort mortality declines are greater among the advanced cohorts than for the younger generation of cohorts. The impact of mortality declines on life expectancy will be most significant at younger older ages, even over the more recent future. As the advanced cohorts grow older, moving diagonally across the synthetic years, the impact of mortality declines on life expectancy decreases.

The synthetic survival curves at ages 65 years and over shown in Figures 14.3a and 14.3b are more convex in shape compared to the cohort survival curves in Figures 14.2a and 14.2b. The greater convexity of the synthetic curves, translates into a greater degree of mortality compression, and are achieved by the cumulative effect of gains in

survivorship at early ages in the younger cohorts, with gains at older ages in the older cohorts.

Figure 14.3a Observed and projected non-Maori male synthetic survival curves at ages 65 years and over, by census years, 1996 to 2026

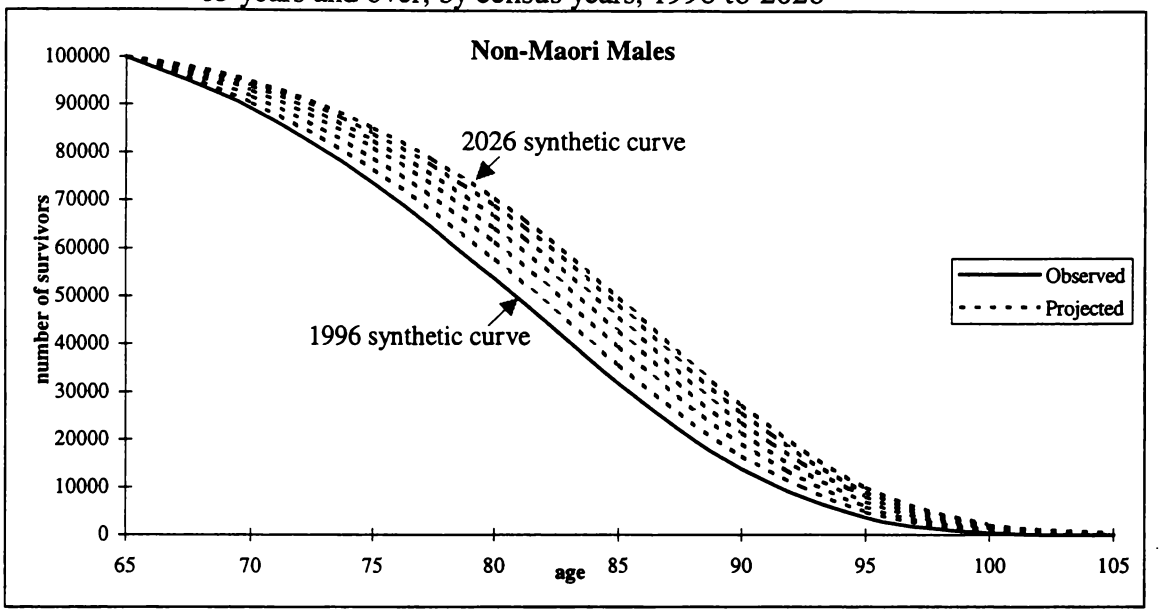
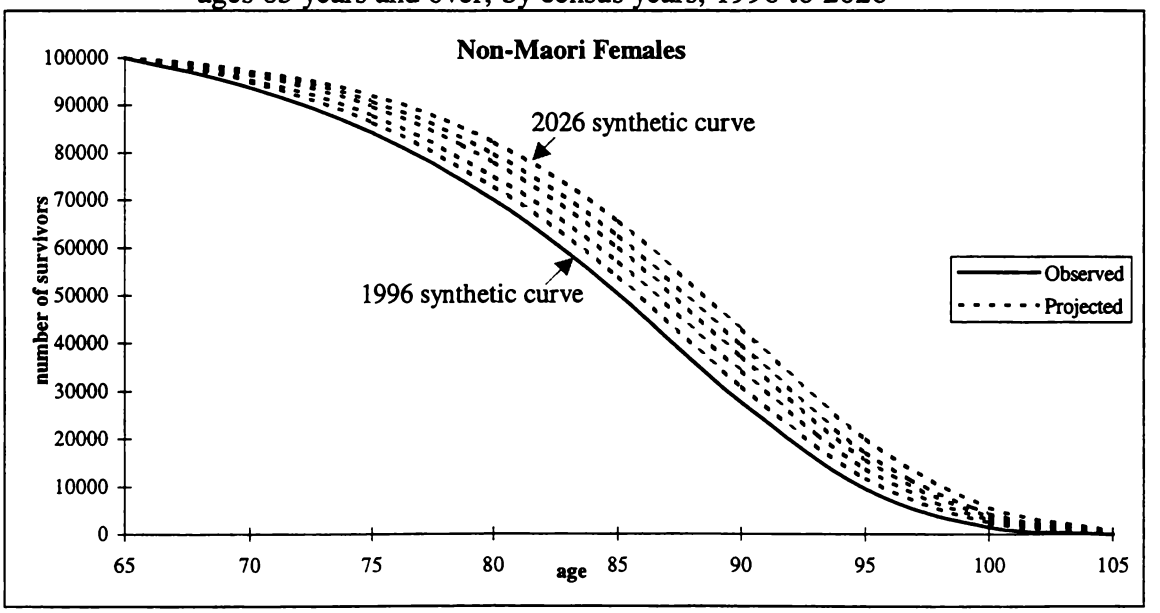


Figure 14.3b Observed and projected non-Maori female synthetic survival curves at ages 65 years and over, by census years, 1996 to 2026



Sources: Calculated by the author from cohort mortality projections

Extension of the tail of the survival curves, which produced absolute mortality expansion in the past, is still noticeable, especially for females. Considerable gains at less advanced ages, expanding the first three-quarters of the survival curves will, however, more than off-set the extension of the tail. Consequently, the survival curves at ages 65 years and

over projected for 2001 to 2026 are becoming more rectangularised. The synthetic trend of relative mortality compression started much earlier in the 1980s (see Section 11.3.2 and Figure 11.6) is projected to strengthen considerably for both males and females over the projected period. In addition, absolute mortality compression among females started during the last inter-censal period is expected to continue at a moderate pace, sustained by the female cohort absolute compression trend in the younger cohorts. While for males, absolute expansion is still expected between 1996 and 2011, followed by a weak trend of absolute compression.

14.2 Future Scenarios of Morbidity Changes

The focus of this section turns to the future morbidity trends in relation to the projected mortality changes. As encountered throughout this thesis, the analysis of non-Maori morbidity trends, especially cohort trends, is hampered by the general lack of adequate historical morbidity data, which makes it difficult to form a conclusive view on its future evolutionary pathway. Three leading paradigms on the current and future morbidity developments in relation to mortality changes are put forward in the international research arena. These are "compression" (Fries, 1980); "pandemic" (Gruenberg, 1977; Kramer, 1980), later encapsulated by the notion of "expansion" (Olshansky, 1985); and "dynamic equilibrium" (Manton, 1982). All three paradigms and recent developments have been discussed already in details in Chapter Five.

In this section simulations of likely future non-Maori morbidity trends under the projected mortality environment are carried out, incorporating the likely scenarios of morbidity development postulated in the international literature outlined above. It should be pointed out that this is not a forecasting exercise. Rather the purpose is to elaborate the implications of possible morbidity changes in response to the changing mortality environment. Due to the unavailability of cohort time series data, morbidity modelling is restricted to synthetic trends by census years between 2001 and 2026. Even at an international level cohort-based morbidity studies and/or projections of population in developed countries are rare.

The hierarchical series of disability-based health expectancies analysed in Chapter Nine is used to model likely future scenarios of morbidity developments. The analysis of the results of these simulations are also guided by patterns and trends in hospital utilisation investigated in Chapter Nine.

The advantage of using a set of measures corresponding to multiple levels of severity of disability is that it allows two-dimensional modelling to be carried out, including both prevalence and severity dimensions. Four scenarios are reported here, these are:

1. static - no change in disability prevalence rates from the 1996/97 levels for all three levels of disability (ie. changes in Disability-Free Life Expectancy (DFLE), Independent Life Expectancy (ILE) and Active Life Expectancy (ALE) will be driven entirely by changes in projected mortality rates);
2. compression - across-the-broad decreases in age specific disability prevalence rates for all three levels of disability;
3. expansion - across-the-broad increases in age specific disability prevalence rates for all three levels of disability; and
4. dynamic equilibrium - increases in age specific disability prevalence rates for level one disability, constant rates for level two disability and decreases in rates for level three disability.

The process of change is assumed to be systematic. In all cases where changes are assumed, inter-censal changes in age specific disability prevalence rates are set at one percentage point, or 100 per 10,000 as conventionally expressed. This assumed magnitude of changes is generally in line with the experiences of the older population in the United States from longitudinal studies (Crimmins et al, 1997a; 1997b; Manton et al, 1993; 1997a). For reasons of simplicity, prevalence rates for different levels of disability are assumed independent of one another. This assumption of independence offers significant practical advantages, though its effects on overall results is difficult to assess with the available data.

Health expectancies calculated under difference scenarios are converted into expectation of life with varying levels of disability. That is, the difference between ILE and DFLE represents state with mild (level one) disability; the difference between ALE and ILE

represents state with moderate (level two) disability; and the difference between life expectancy and ALE represents state with severe (level three) disability.

14.2.1 Scenario One: Static

Results of morbidity modelling of scenario one, expressed as life expectancy at age 65 years with different levels of disability, are summarised in Table 14.3. As expected, expectation of life in all states of health/disability increases in accordance with increases in life expectancy. These increases are driven purely by the projected mortality declines which vary in magnitude by age group and by birth cohorts, and therefore increases in expectation of life are not equally shared among the different states of health/disability. Expectation of life with moderate disability requiring intermittent assistance has the highest prevalence rates of all levels, and has the largest increase of more than one year over the 30 year period. The increase in expectation of life with moderate disability outpaces that with no disability. In contrast, expectation of life with mild disability requiring no assistance benefits the least from the projected mortality declines.

Table 14.3 Non-Maori expectation of life at age 65 years with varying levels of disability under scenario one of constant disability prevalence rates, by gender, by census years, 1996 to 2026

	1996	2001	2006	2011	2016	2021	2026
			Males				
With no disability	7.6	7.9	8.1	8.4	8.6	8.7	8.8
With level 1 disability	2.3	2.4	2.5	2.6	2.7	2.8	2.8
With level 2 disability	4.3	4.7	4.9	5.2	5.4	5.6	5.8
With level 3 disability	1.5	1.6	1.7	1.8	1.9	2.0	2.1
Total (life expectancy)	15.8	16.6	17.4	18.0	18.6	19.1	19.5
			Females				
With no disability	9.3	9.6	9.8	10.1	10.2	10.3	10.4
With level 1 disability	2.6	2.7	2.8	2.8	2.9	2.9	3.0
With level 2 disability	4.8	5.1	5.3	5.6	5.8	5.9	6.1
With level 3 disability	2.6	2.8	2.9	3.1	3.2	3.3	3.4
Total (life expectancy)	19.3	20.1	20.8	21.5	22.0	22.5	22.8

Sources: Estimated by the author

When expressed as a percentage of total life expectancy at age 65 years, however, the proportion of remaining life spent free of any disability decreases over time, from 48

percent in 1996 to 46 percent in 2026. Conversely, the proportion in state of disability increases. Most of the proportional increases occur in states of moderate to severe disability, due to the relatively high prevalence of levels two and three disability at ages 80 years and over and the greater mortality declines projected at these older ages (see Figures 9.1a, 9.1b, 14.3a and 14.3b). The projected increases in time expected to be spend in a state of severe disability may seem contradictory to the recent trends in hospitalisation expectancy, given the close resemblance of the two measures (see Chapter Nine).

14.2.2 Scenario Two: Compression

Under the most optimistic scenario of morbidity compression, expectation of life at age 65 years free of disability increases by 4.8 years for males and 5.3 years for females over the 30 years (see Table 14.4). By 2026, around two-thirds of the remaining life expectancy at age 65 years can be expected to spend in disability-free state. For both genders, the increases in disability-free life expectancy substantially out-pace the corresponding increases in life expectancy.

Table 14.4 Non-Maori expectation of life at age 65 years with varying levels of disability under scenario two of compression, by gender, by census years, 1996 to 2026

	1996	2001	2006	2011	2016	2021	2026
			Males				
With no disability	7.6	8.4	9.2	10.0	10.8	11.6	12.4
With level 1 disability	2.3	2.3	2.2	2.1	1.9	1.8	1.6
With level 2 disability	4.3	4.5	4.6	4.7	4.7	4.7	4.6
With level 3 disability	1.5	1.5	1.4	1.3	1.2	1.1	0.9
Total (life expectancy)	15.8	16.6	17.4	18.0	18.6	19.1	19.5
			Females				
With no disability	9.3	10.2	11.1	12.0	12.9	13.7	14.6
With level 1 disability	2.6	2.5	2.3	2.2	2.0	1.8	1.6
With level 2 disability	4.8	4.9	4.9	4.9	4.9	4.8	4.7
With level 3 disability	2.6	2.6	2.5	2.5	2.4	2.2	2.1
Total (life expectancy)	19.3	20.1	20.8	21.5	22.0	22.5	22.8

Sources: Estimated by the author

The largest compression effect occurs for the state of mild disability. Expectation of life with mild disability reduces by 0.7 years for males and 1.0 years for females. Expectation

of life with severe disability is also compressed by around half a year for both genders. While mild and severe disability show direct compression effects, expectation of life with moderate disability fluctuates in a narrow range, increasing slowly for most of the projected period before trending down. This is because the high initial prevalence rates of moderate disability at older ages make the one percentage point reduction relatively less insignificant, and the effects of reduction during the early projected period are overshadowed by the impacts of projected mortality declines and thus the momentum from an ageing structure of the older population.

The compression scenario points to an absolute morbidity compression, with the expected future years spend in disability reducing in numbers. The projected compression of severe disability can draw on recent trends in hospitalisation expectancy for its empirical support (see Sections 9.2.5 and 13.1.4), though the projected rate of decline in life expectation with severe disability is considerably slower than the that for hospitalisation expectancy over the last 15 years. However, given the current morbidity environment, significant reductions in expectation of life with mild disability and the outcome of increases in life expectancy free of disability running in tandem with increases in life expectancy may not seem realistic.

More importantly, what is also revealed under this scenario of change is that expectation of life with moderate disability increases in the short-term future, albeit marginally. This increase comes despite the strong morbidity compression effects at work.

14.2.3 Scenario Three: Expansion

In sharp contrast to the compression scenario, the scenario of morbidity expansion yields significant increases in expectation of life with all levels of disability at the expense of disability-free years (see Table 14.5). By 2026, nearly three-quarters of remaining life expectancy at age 65 years is expected to be spent in some forms of disability. The largest increase is in a state of moderate disability. In fact, the time expected in moderate disability is longer than all other states beyond age 65 years, including the state with no disability.

Table 14.5 Non-Maori expectation of life at age 65 years with varying levels of disability under scenario three of expansion, by gender, by census years, 1996 to 2026

	1996	2001	2006	2011	2016	2021	2026
			Males				
With no disability	7.6	7.4	7.1	6.7	6.3	5.9	5.3
With level 1 disability	2.3	2.6	2.9	3.2	3.4	3.7	4.0
With level 2 disability	4.3	4.8	5.3	5.7	6.2	6.6	6.9
With level 3 disability	1.5	1.8	2.1	2.4	2.7	3.0	3.2
Total (life expectancy)	15.8	16.6	17.4	18.0	18.6	19.1	19.5
			Females				
With no disability	9.3	9.0	8.6	8.1	7.6	7.0	6.3
With level 1 disability	2.6	2.9	3.2	3.5	3.8	4.0	4.3
With level 2 disability	4.8	5.3	5.7	6.2	6.7	7.1	7.4
With level 3 disability	2.6	3.0	3.3	3.8	4.1	4.5	4.8
Total (life expectancy)	19.3	20.1	20.8	21.5	22.0	22.5	22.8

Sources: Estimated by the author

Under the expansion scenario, the magnitude of reduction in disability-free life expectancy of 2.3 years for males and 3.0 years for males appears even less realistic than the results for the compression scenario in Table 14.4. In the case of females, the reduction in DFLE almost rivals the magnitude of increase in total life expectancy. It would appear, therefore, that should the expansion theory hold true, the resulting changes in expectation of life with different levels of disability could not go beyond the ranges of change indicated in Table 14.5.

Despite doubts over the assumptions on the magnitude of changes, the results help to highlight the underlying message that significant increases can be expected in expectation of life with moderate disability requiring intermittent assistance.

14.2.4 Scenario Four: Dynamic Equilibrium

Under the scenario of dynamic equilibrium, changes in mild disability prevalence rates are assumed to be same as that under scenario three (expansion), and the resulting expectation of life with mild disability in Table 14.6 are identical to those in Table 14.5. Similarly, expectation of life with severe disability is the same as that under scenario two in Table 14.4. Changes in mild and severe disability prevalence rates cancel each other

out, leaving the same set of expectations of life with no disability and with moderate disability as under scenario one in Table 14.3.

Table 14.6 Non-Maori expectation of life at age 65 years with varying levels of disability under scenario four of dynamic equilibrium, by gender, by census years, 1996 to 2026

	1996	2001	2006	2011	2016	2021	2026
			Males				
With no disability	7.6	7.9	8.1	8.4	8.6	8.7	8.8
With level 1 disability	2.3	2.6	2.9	3.2	3.4	3.7	4.0
With level 2 disability	4.3	4.7	4.9	5.2	5.4	5.6	5.8
With level 3 disability	1.5	1.5	1.4	1.3	1.2	1.1	0.9
Total (life expectancy)	15.8	16.6	17.4	18.0	18.6	19.1	19.5
			Females				
With no disability	9.3	9.6	9.8	10.1	10.2	10.4	10.4
With level 1 disability	2.6	2.9	3.2	3.5	3.8	4.0	4.3
With level 2 disability	4.8	5.1	5.3	5.6	5.8	5.9	6.1
With level 3 disability	2.6	2.6	2.5	2.5	2.4	2.2	2.1
Total (life expectancy)	19.3	20.1	20.8	21.5	22.0	22.5	22.8

Sources: Estimated by the author

Again, expectation of life with moderate disability increases by the largest magnitude, further reinforcing the important role played by moderate disability in overall morbidity pattern. Expectation of life with mild disability has the second largest increases, higher than that shown by expectation of life with no disability. As explained earlier, increases in expectation of life in a state of mild and moderate disability are driven by two different factors. The former reflects expansion effects in relation to changes in the ruling mortality regime, while the latter shows effects entirely attributable to mortality changes.

This scenario of dynamic equilibrium combines essentially the most acceptable elements of all three previous scenarios, given what is known about recent trends in health expectancy overseas and non-Maori hospitalisation expectancy. Unrealistically sharp rises in DFLE under scenario two are eliminated, and so too is the most pessimistic declines in DFLE under scenario three. The assumed expansion of mild disability can be justified on the ground of flow on effects from reductions in severe disability prevalence rates.

14.3 Summary

Persistent mortality declines at adult ages are projected for the non-Maori population between 1996 and 2026. Most of these mortality decline are sustained by the favourable mortality experiences of the advanced cohorts born between 1901 and 1931. The trend of inter-cohort survivorship improvements at late adult ages, starting much earlier with the complete cohorts, is projected to strengthen among the successive advanced cohorts before easing among the younger generation of cohorts. Cohort life expectancies at birth are expected to increase by around 10 years to 70.4 years for the male cohorts born in 1926-1931 and 76.8 years for the corresponding female cohorts. Cohort survival curves are becoming decisively more rectangularised due to the fact that substantial improvements at younger ages have already taken place. At age 65 years, for both genders another four years are expected to be added to cohort life expectancy.

Translating cohort based mortality projections into cross-sectional patterns, life expectancy at age 65 years is expected to increase at a decelerating pace to 19.5 years for males and 22.8 years for females in 2026. The synthetic survival curves at ages 65 years and over are becoming more rectangularised. The effects of the projected changes in survivorship on the future size and age mix of the older population by census years between 2001 and 2026 are discussed in the next chapter (Fifteen) in the context of policy implications.

Less sophisticated simulations of scenarios of future morbidity changes in relation to projected mortality changes are presented in the second part of the chapter. Not surprisingly, changes in synthetic disability-based health expectancies at age 65 years are mostly driven by the set of assumptions employed. However, the most likely factor, common to several of the scenarios, is that expectation of life with moderate (level two) disability will increase. Depending on the actual theoretical pathway that eventuates, the state of moderate disability has the potential to overtake disability-free life expectancy as the leading state of health/disability at late adult ages (65+ years).

This can be shown by reference to what seems the most realistic scenario of dynamic equilibrium, disability-free life expectancy at age 65 years increases as a result of

continuing mortality reduction, but fails to match up with increases in life expectancy. The remaining life time expected to be in a state of mild to moderate disability increases at a pace faster than the state of disability-free, though this is compensated for, to a lesser extent, by the compression of the most severe forms of disability.

In the next chapter, the policy implications of shifts in future patterns and trends in non-Maori mortality and morbidity will be discussed. Other substantive issues raised in earlier chapters will also be addressed from a policy perspective.

CHAPTER FIFTEEN
CONCLUSIONS: POLICY IMPLICATIONS

Over the last century, the non-Maori population in New Zealand had undergone a dramatic yet complex health transition. The patterns and trends of this transition, especially those in the last half century, have been explored in this thesis using macro-level data and measures. A long history of cohort exposure to the risk of mortality was constructed and analysed for the purpose of understanding the accumulated cohort effects from earlier exposure to mortality and morbidity. The empirical research in this thesis examined different theoretical perspectives on possible future trends in developed countries with low mortality. This has been a population health approach to the analysis of heterogeneity among the older population, employing differing methods from those often reliant on micro level data.

This chapter first goes back to the overall premise of the thesis and outlines some substantive, methodological and theoretical objectives addressed in earlier chapters. The focus of the present chapter then moves forward from historical trends to their implications for future changes. The discussion permits the thesis to address policy level questions, thereby providing a context in which policy responses to future changes in population health can be formulated.

15.1 An Overview of the Thesis

The empirical focus of the present study has been on recent mortality trends at older ages and their implications for morbidity changes. To view these issues from a wider demographic perspective and to establish a theoretical framework for their empirical analysis, in the earlier part of the thesis various transition theories and empirical observations were reviewed. Common features of the century long health trends exhibited by populations in developed countries, including non-Maori in New Zealand, were tracked using as a framework the demographic transition model, which maps the shift from high levels of mortality and fertility to the current low levels of mortality and fertility. In doing so, the role of population health trends was then identified as a constituent of the wider demographic transition. The underlying arguments developed as

a result of this review emphasised the need for dynamic multi-directional approaches in studying the processes of changes in population health, and recognised that the exact timing of change is of secondary importance to the process itself.

Another major theoretical extension of the present study was to forge micro-macro linkages between individual senescence and population health. The underlying biological process governing the subject matter of this thesis was first explored using micro-level explanations from a range of disciplines. Demographic interpretations of these micro-level explanations of individual differences in the senescence process were further elaborated through population level modelling, which then led to the introduction of the notion of population heterogeneity, a phenomenon increasingly discussed in the demographic literature. It was argued that cross-sectional views employed in most conventional demographic analyses of health collapse multiple birth cohorts at one point in time, and thus tend to make arbitrary assumptions about the levels of heterogeneity. A theoretical justification for a cohort approach to the analysis of population health patterns and trends is therefore strong, and it is this strategy which guided the analytical approach in the later part of this thesis.

Even those using a cohort approach have still made assumptions about heterogeneity without providing an empirical base for this. The present thesis has, instead, developed an empirical method for identifying heterogeneity and has shown inter-cohort differences in this property.

The background to the more recent mortality and morbidity trends was investigated as part of the theoretical component of this thesis. Alternative theories on mortality and morbidity at older ages were examined, with reference to macro and micro level understandings of population health outlined in earlier chapters. To guide the empirical case study on non-Maori, an integrated framework was developed which incorporates complementary elements of the various theories on the future evolution of population health. The notion of compression of mortality and its corollary, the rectangularisation of the survival curve, was identified as an important anchor point for empirical elaborations since it could be subjected to rigorous testing with available data, yet the concept has far-reaching implications. It was also postulated in the integrated framework that the three

leading theories on morbidity, expansion, dynamic equilibrium and compression reflect different stages of the health transition, and can be reconciled theoretically and empirically.

Translating these theoretical issues into analytical objectives, a framework for the analysis of non-Maori health patterns and trends was developed. This framework brings together major methodological advancements in at least three different directions. First, mortality, a fundamental determinant of population behaviour, was analysed with data spanning over a century. Patterns and trends in morbidity were then analysed in relation to the changing levels of mortality. Second, a population-based true cohort approach, supported by available data, forms the core of the this framework, complementing the more conventional synthetic analyses. Theoretical issues such as population heterogeneity and its determinants could then be addressed from a cohort perspective. Third, on the measurement front, a popular tool for analysing population health used in many other countries, known as health expectancy, was applied in New Zealand for the first time with a total population focus. The technique was then refined to construct a new measure termed "hospitalisation expectancy" which integrates data on hospital utilisation and mortality to yield a summary measure of population health. Backed up by the available historical data on hospital utilisation in New Zealand, hospitalisation expectancy was able to be calculated back in time to provide some valuable insights into historical trends in population health.

The empirical sequence of thesis started with a detailed investigation into the longer-term non-Maori mortality trends, which also set the context for the subsequent synthetic and cohort analyses. Shifts in the force of mortality from younger to older ages were clearly demonstrated to have occurred throughout the twentieth century, although the patterns of these shifts were far from smooth as evident by periods of fluctuations and even reversals in mortality trends. Based on these patterns, five distinct periods in which major changes in non-Maori mortality occurred were identified. The impacts of mortality changes at younger ages had slowly transformed from a key determining factor in overall level of mortality in the early years, essentially to a supporting role of enhancing survivorship and longevity once low levels were reached in the middle of the twentieth century. In contrast, mortality changes at older ages have progressively overtaken

declines at younger ages as the most dominant force. Fluctuations in old age mortality trends in the middle of the twentieth century were principally responsible for the distinctive periods of mortality change. Cohorts born around the turn of the twentieth century contributed directly to this shift process. Declines in their infant and childhood mortality and fluctuations in old age mortality were the most dominant forces in the respective time periods. Over the most recent period since the early 1970s, unprecedented mortality declines in old age mortality have taken place, producing significant increases in life expectancy at birth and raising questions whether or not life expectancy itself is moving closer to a biological limit. This latter question has generated the idea of the compression of mortality.

Accompanying the mortality changes as factors defining population health dynamics have been the changing patterns of morbidity. Thus, patterns and trends in non-Maori morbidity were analysed in this thesis in relation to levels of mortality. Following improvements in survivorship the force of morbidity had also shifted up the age scale, though not to the same extent as that occurring for mortality. Moreover, while the force of mortality was becoming increasingly concentrated around a few selected chronic degenerative diseases, the patterns for causes of morbidity became more dispersed, with non-lethal conditions becoming more heavily represented.

The diverging patterns for causes of mortality and morbidity highlight the question whether, as a result of mortality declines, morbidity is becoming more protracted or is becoming limited to the last few years of life. To help investigate this issue, disability-based health expectancies and hospitalisation expectancies were constructed. Notwithstanding limitations in the morbidity data, comparisons between expectation of life with varying levels of severity of disability from cross-sectional observations indicate that the more severe forms of morbidity are being compressed into a narrower proportion of the life span. This inference was further supported by patterns found in hospitalisation expectancies. In addition, a time series analysis of hospitalisation expectancies, albeit that this was limited to the last few decades, suggests that compression of severe morbidity has indeed been occurring.

This finding has important theoretical as well as policy implications, the latter of which will be discussed later (see below 15.2.2). A major theoretical implication is that deterministic views about the future of population health may be unrealistic. Above all the assumption that improved survivorship will be accompanied by longer periods of illness and a decreased quality of life must be refined and nuanced. There may be increased durations of illness overall, but this thesis shows that the duration of severe illness for surviving members of cohorts may well remain the same or decrease — that is that more of the remaining years of life will be spent outside hospital than was true in the past. Of course, this may be occasioned by resort to chemo-therapeutics and other bio-medical procedures. But arguably, it is better (that is, there is an improved quality of life) to be in a non-institutional setting, albeit with a major illness held at bay and not "cured", than to be hospitalised, and thus, by definition, dependent.

The issue of compression of mortality was investigated in this thesis first from the viewpoint of a period phenomenon between 1971 and 1996. A more rigorous definition of the concept was formulated so that its objective measurement could be made possible, including two empirical measures developed by the present author. It was found that, when mortality at all ages was considered, mortality compression was occurring among non-Maori. Focusing at ages 65 years and over, on what is mainly chronic diseases mortality, survivorship patterns were seem to be changing in rather complex ways. The momentum of mortality reductions generally persisted to the very old ages, thus stretching the tail of the survival curve. The resultant trend was a simultaneous mortality compression in a more relaxed relative sense and expansion in a more robust absolute sense. This finding indicates that life expectancy has yet to reach its biological limits. Further improvements in survivorship at the very old ages can be realistically expected before eventual absolute compression takes place.

A cohort analysis of observed mortality and morbidity patterns and trends was carried out in the present study. This was done by way of constructing mortality histories by birth cohort and comparing inter-cohort trends. Because of the lack of availability of morbidity data and the quality of those available data, the construction of morbidity profiles by cohort was generally not possible.

Patterns observed in inter-cohort mortality trends suggest that a strong cohort effect was operating among cohorts born before the turn of the twentieth century. An inverse selection effect was detected among these cohorts in which significantly improved survivorship at younger ages was off-set by mortality fluctuations at late adult ages. A synthesis carried out in the latter part of the thesis, correlating survivorship changes at younger ages with those at middle to late adult ages, further highlighted this inverse selection effect for both genders. The same analysis identified the fact that reduced communicable disease risks at infancy and early childhood were strongly related to increased chronic respiratory mortality risks at older ages. For these earlier cohorts, exposure to the risk of communicable diseases at younger ages was found to be a key determinant of cohort heterogeneity at older ages.

The issue of mortality compression at older ages was investigated in this thesis also from a cohort perspective, using as a basis an analysis at 65 years and over. Relative compression effects observed in this thesis using synthetic data were generally not supported by cohort data. Instead, mortality expansion was the more dominant feature in non-Maori cohort mortality trends. This is because inter-cohort survivorship improvements so far at older ages have been concentrated at more advanced ages, giving rise to a more widely stretched tail of the survival curve. However, it was also discovered that some important baselines have been laid for mortality compression, in that the momentum of mortality reductions, which had started from the very old ages had progressively spread with successive birth cohorts to less advanced ages. The effect will be to shift the majority of deaths, instead of an extreme minority, to an even older age, and produce relative compression in the process. This inference of future mortality development was then used to prepare projections of non-Maori mortality changes by cohorts between 1996 and 2026.

15.2 Policy Implications of Future Evolution of Population Health

For population health research to be effective it must have direct applied values in the social policy arena, the medium by which issues affecting the wider population can be addressed. The rest of this chapter takes a prospective stand-point, focusing on the

implications for the formulation of future health and related social policies in response to the various issues identified earlier in the present study.

The discussion is organised into five sub-sections. Substantive issues raised in the earlier chapters are, however, not necessarily confined to any particular one of these themes. The first four sub-sections deal with central themes emerged from the key findings of this thesis and their implications for the health and the wider social sectors. Although these emerging issues are discussed separately, their close inter-relationships must be stressed. The fifth sub-section outlines a research agenda for these issues.

15.2.1 Future Mortality Changes and Population Dynamics

As a central theme of this thesis, a major inference of future non-Maori mortality changes from the study of mortality compression is that future increases in life expectancy at birth can be realistically expected. However, what is also clear from the heated theoretical debate on the compression of mortality is that the rapid increase in life expectancy enjoyed by many developed countries may not continue at its current pace. Both of these conclusions are certainly substantiated by the results of cohort-based mortality projections of non-Maori in the last chapter (Fourteen). Projected life expectancy at age 65 years measured in synthetic life tables is expected to increase at a slowly decelerating pace (see Table 14.2).

The realisation that we may not yet be on the fringe of the limits to life expectancy has important implications for future policy setting. Though reductions in old age mortality will have an increasingly minimal effect, from an analytical point of view, on life expectancy at birth, their possible impacts on population health dynamics need to be carefully assessed. This is because the base number in any cohort surviving to middle or older adult ages is now very large, so that even a small improvement in survivorship at older ages will produce a significant increase in the expected size of future older populations. Because of what is essentially a size effect, increases in life expectancy at more advanced ages can be expected to be accompanied by concomitant shifts in patterns of social expenditure on the older populations. The economic and social implications of further reductions in old age mortality cannot therefore be underestimated. Policy decisions on service provision,

capital and infrastructure investments being made today will have implications for health programmes well into the twenty-first century, yet these tough decisions have to be undertaken now.

Some valuable lessons can be drawn from the past. An overly conservative outlook on mortality changes during the mid 1950s to the early 1970s, under a climate of stagnated life expectancy trends, was guided by a belief in a fixed life expectancy, and directly contributed to under-development of community-based home health care and prevention strategies for chronic disease which were viewed as "natural" concomitants of ageing (Manton, 1991). An even more prominent example was provided by the systematic under-estimates in projections of the United States population which later led to dramatic changes in tax policy and policies on eligibility for Social Security, in order to ensure solvency of the Social Security system.

Underlining these policy concerns is the role of mortality in an ageing population. The issue of population ageing was introduced in this thesis at the very start in Chapter One. This is also an issue currently attracting very strong interest from other quarters including policy, for its ramifications are long lasting. The ageing of the non-Maori population in New Zealand, a major driving force of demographic changes in the early twenty-first century, is, however, not a simple monotonic process (Pool, 1999a). This is because for this population the process does not follow a simple progression towards ageing, the end point of change, albeit the factor which tends to capture the attention of politicians. Instead, the process is significantly influenced by population waves created by what are termed "disordered cohort flow" (Rowland, 1996). As identified in earlier chapters, population ageing is largely a function of levels of fertility, which was most vividly demonstrated by the "baby boomer" cohorts, but also by the return to replacement levels around 1990.

Mortality, on the other hand, operates as an important mechanism regulating the size of the flow of these "disordered cohorts". Given the current low levels of mortality at most ages, this factor is generally less important than birth cohort sizes which, in any case, were determined by fertility trends far back in the twentieth century, well before population ageing became a dominating factor for public policy. A more important

function of mortality in an ageing population is, however, its selection effect in determining the composition of population groups in terms of their susceptibility to ill health. The implications of increased heterogeneity among the older population is discussed as a major theme below.

Extending from the idea of rectangularisation of the life table survival curve and the ageing of the population, rectangularisation of the populations with low levels of mortality is said to be occurring (Levy, 1998). A rectangular population pyramid certainly raise some interesting social policy issues in terms of the caring capacity and inter-generational relationships. While the demand for health and support services by the older populations is likely to increase, the future supply of care givers and caring capacity is a function of the changing population dynamics and thus requires thorough consideration. Moreover, the supply of care givers cuts across a wide range of occupations, from medical specialists to persons giving support for daily living. Adequate supplies of these many professions in the future will entail more proactive policies on education and training to be in place. Future employment policies, including those on issues affecting female participation in the labour force, will also have an impact.

However, it should be noted that often buried in the ageing population debate is the fact that nowadays most of the older people are healthy and continue to be productive and caring for themselves into very old age. The younger members of the older population often care for the older members of their peer group, and therefore provide the caring capacity themselves (Arber, 1996; Pool, 1999b). Viewing the issue of ageing from a strictly fiscal perspective tends to overlook the roles and the contributions of the older generation to the society, and risks seeing the old as merely being contributors to fiscal burden.

15.2.2 The Compression of Severe Morbidity and the Impacts on Health Services

A major policy concern in the context of a significant increase in life expectancy at older ages in the more recent period is whether or not increases in life expectancy would result in poorer overall health for the older population because the individuals living longer will

be burdened with co-morbidity. This in turn would mean increased demand for health and support services and reductions in quality of life for surviving elderly. But against this, results of the analyses presented in this thesis indicate that the majority of the population, even at older ages, are expected to stay healthy and independent. Greater longevity is not necessarily being accompanied by longer periods of hospitalisation, and thus will mean lower demand for services, and a better quality of life.

Clearly, the pattern of a compression of disability of the most severe forms into a fraction of life expectancy and a related compression trend for hospitalisation expectancy over the most recent period have major implications for health and support service provisions. These implications are heightened by the fact that in many developed countries rationing of health resources, especially those for secondary and tertiary services, is gradually being brought to the forefront of public debate. In fact, the reduced incidence of intensive intervention with advancing ages, as noted in Chapter Five, suggests that high-cost medical services may already be allocated to the elderly in a more rational manner than is generally assumed. The compression of hospitalisation expectancy in the context of enhanced longevity may help to shed some valuable insights on this rationing debate (see also Section 15.2.3 below on the issue of equity). The period of the entire life span that can be expected to be spent in secondary or tertiary hospital care is declining, partly reflecting configurational changes in health service provision. Overall, future older populations can expect to be healthier than the groups currently at these ages, yet the focus of their needs for health and support services is also likely to shift away from intensive interventions to one focusing on prevention and care in the community or in institutions.

The issue of community-based preventative care is sustained by results of a synthesis of future mortality and morbidity trends carried out in this thesis. This points to a likely increase in the duration in states of moderate disability and hence increments in the numbers of people affected. Under the most realistic scenario modelled here, at age 65 years expectation of life with moderate disability has the potential to overtake disability-free life expectancy as the leading state of health. Longer-term policies with a strong community-based health focus aiming at addressing this issue need to be implemented soon before this trend eventuates.

The compression of severe morbidity and hospitalisation also has implications beyond the more immediate purpose of service planning. As part of the underlying arguments of the pandemic of morbidity theory (reviewed Chapter Five), it is argued that in the past bio-medical research resources were disproportionately funnelled into studies on treatment, with less going towards disease prevention (Gruenberg, 1977). Though available data in New Zealand do not permit an analysis of health expectancies by cause, results from overseas studies suggest that the majority of years in which there is loss of independence are likely to be accounted for by a relatively limited number of chronic disabling conditions, a substantial proportion of which can be prevented or postponed (Khaw, 1997). Shifts in the policy focus and the concomitant shifts in resources for research and development are therefore important.

For the population as a whole, a greater focus is needed on prevention and health maintenance. A social and policy framework which helps individuals to fully realise their potential in attaining physical, mental and social well-being is important (Tu, 1999). A case in point are those people who were identified in the 1996/97 Disability Surveys as having a disability requiring limited assistance (level one disability). Catering for the needs of this group of people, estimated to be some 300,000 in numbers (or 83 per 1,000 people) in the total population (Health Funding Authority and Ministry of Health, 1998), represents a great opportunity for health maintenance and a potential for health gain. Though the majority of these people are currently not captured in the health care system, policies designed to help them manage their conditions are crucial as preventative measures, to ensure that they do not worsen and thus require more support at a greater cost.

15.2.3 The Changing Patterns of Causes of Mortality and Morbidity

The incidence of non-fatal conditions requiring hospitalisation increased significantly over the recent decades and accounted for a disproportionately larger share of hospital utilisation trends. In contrast, the causes of mortality became more and more compressed into a few major categories, mainly cancers and cardiovascular diseases.

These diverging patterns of causes of mortality and causes of morbidity identified in this study present major implications for policy makers. The possibility of finding a single solution by which to arrest both mortality and morbidity simultaneously seems remote. Some forms of prioritisation between enhancing longevity and improving quality of life, and the various means by which to achieve these will be required. Indeed, some trade-offs may have to be made (see Section 15.2.6 for a discussion on health and hospitalisation expectancies as an analytical tool in this line of policy research).

Competing needs for the same health resources will arise between different components of various socio-demographic sub-groups, between geographic locations, between sufferers from different diseases and conditions, and even between service providers. The maintenance of equity, defined in terms of equal access for equal need (Sceats et al, 1995), will be a central issue for policy makers. Improving and balancing access to health services so as to accommodate the changing, and sometimes conflicting patterns of need will be an ongoing challenge to the health care system for the years to come.

The diverging patterns of mortality and morbidity also raise an important question: are people more likely to present with less lethal causes than previously (because expectations have changed), or will it be more typical that they survive and, indeed, avoid more lethal outcomes, and thus will be hospitalised for less lethal causes? The answer to this question is not likely to be clear-cut, though it will have significant influences on future directions of policy setting.

This thesis has gone some way to address this issue. The compression of severe morbidity accompanying increasing longevity lends empirical support for the latter case. Significant declines in old age mortality in the recent decades and the persistent mortality differentials between population groups suggest that medical and public health interventions have substantially improved survival at older ages (Manton and Vaupel, 1995).

In contrast, issues surrounding changing health perceptions among the population at large, and the impacts these have are beyond the scope of this thesis. What is clear is that planning for future health services for the older population will be increasingly geared

towards less lethal chronic conditions, with a greater emphasis on the preventative aspects of care.

Furthermore, as noted in Chapter Nine, future policy agenda on further lowering levels of mortality may not be effective in reducing levels of morbidity. Solution to this potential conflict will set the stage for a profound paradigm shift of the health sector as a whole, from disease care to health care, from disease/condition-specific interventions to health maintenance and health gain. There is an increasing prevalence of co-morbidity at older ages and many major forms of chronic morbidity have common environmental and behavioural determinants which play an important part in shaping their etiology. As a result, policies aiming at arresting individual diseases are likely to be increasingly less effective in improving population health status than those focusing on risk factor modification and targeting a multitude of conditions.

15.2.4 Increasing Heterogeneity in Older Populations

From the preceding discussion in this thesis on the variability of the ageing process and its spectrum of effects, it is apparent that the older population constitutes an heterogeneous group, demonstrating a broad range of disabilities and capacities to benefit from health care services. Increased levels of heterogeneity at older ages are explained by reduced mortality risks at younger ages, favouring those who would not have been able to survive to older ages under a high mortality regime. In light of the findings from the present study, it can be argued that this trend is set to continue and intensify into the early twenty-first century. An adequate understanding of this subtle yet critical relationship between patterns of exposure to risk at different life cycle stages is therefore important for decision making.

The increasing diversity among the older populations in terms of their health status, and health and support service needs, points to a "natural evolution" of the mix of services required (Tu, 1999:10) which carries far-reaching implications for the health sector. A better understanding of this diversity of need among the older population helps to sharpen the focus of contemporary health policy, which has often been captured by

ageing *per se*, and the "looming crisis" in health care. More adequate identification of high risk groups within the older population is needed, particularly in view of intense competition for limited health resources. An increased knowledge of population health dynamics will better prepare policy makers today to anticipate changes in population structure and other determinants of heterogeneity. The majority of cohorts which will be affected by short to medium term policy changes in the future are already born today, and historic time series information on their patterns of exposure to risk is generally available, albeit limited in some cases. The study of the effects on health and other social processes of increased heterogeneity among these cohorts should become a high priority in informing future decision making processes.

Furthermore, and following on from the earlier point on a shift from disease oriented care to health care, the increased diversity of health needs among the older population provides strong support for individualised care. While at present the health system must find ways to cope with the needs of a diverse group of individuals, health policies will be increasingly challenged in the future to respond quickly to the rapid changes in needs that characterised this extremely heterogeneous group. For example, more frequent transitions between acute care and support and rehabilitation services can be expected among the frail elderly. Integrated networks of health care which can facilitate smooth transitions and provide the necessary system-wide support for administering appropriate levels of individual care will be beneficial to the older members of the society.

The key to a rapid policy responses to the changing needs of the older population is by adopting a dynamic outlook on the health system. Inherent in the idea of increased population heterogeneity is the notion of change, of dynamism in patterns of needs and thus in system responses. The existence of dynamic properties both in terms of population health and health needs was identified very early in this thesis. However, recognition of changing needs in the population are not always met by dynamic responses from the policy sector. One of the more recent example of similar failures in New Zealand was the stream of work on a sustainable funding pathway at the Ministry of Health in consultation with the Crown Company Monitoring Advisory Unit and the Treasury, which was commissioned on the eve of the 1996 general election by the then Minister of Health. This purely financial model had the potential of effectively

underscoring all health policy setting over the period from 1996 to 2000, but unfortunately it was based on a most distinctly static view of the system. Changes were only exemplified by the four variables included in its expenditure projections: these were population size and age structure, price inflation, technology and intensity of service use ("volume growth per capita"). Results from assumed changes in these "environmental drivers" were combined with the current funding levels to derive a sustainable funding package. Implicit in this package were, however, the assumptions that the current service configurations as at 1996 were adequate and effective in meeting the needs of the population, and were to be "sustained". But importantly, it was also implicitly assumed in that the same mix of services will be equally adequate and effective in the short to medium future.

A similar reliance on a macro-economic management framework in directing health policy decisions can also be found in policy discussions overseas (for example, see Getzen, 1992). It is clear from the preceding discussions in this chapter, however, that for future policy setting to be effective, financial sustainability and/or macro-economic considerations cannot be treat as a separate entities from the multiplicity of health needs of a heterogeneous population and the complexity of the health system.

15.2.5 Research Agenda to Inform Policies Affecting Future Population Health

Effective determination of public health priorities and planning for health and other social services requires reliable information on the health status of the population. As advocated by the WHO programme "Health for All", this information is required for assessing the health status of populations in both a national and an international context, and also for understanding and monitoring the positive as well as negative determinants of well-being and their dynamics (Nayar, 1999).

It was noted in Chapter Seven, that there is a dearth of morbidity data in New Zealand by comparison with some other developed countries (Johnstone et al, 1998; Katzenellenbogen and Cheung, 1998). Improved availability of population-based health and related data, from either routinely collected statistics or population-based surveys, is

required. Innovations in collating health data, such as, for example, improving the efficiency of transfer of administrative records to a centrally administered population health data set (through the National Health Index), and linking these processes and information series to survey design and to survey data, and developing procedures to combine multiple data sources (including the censuses), should be considered as high priorities.

Health expectancy type measures which integrate levels of mortality and other health-related dimensions have been validated in numerous overseas studies, and have been proven to be a useful analytical tool and a highly relevant indicator for policy decision making. Health expectancies calculated for different demographic sub-groups can be compared directly and over time, independent of size and age compositional effects. The ability to make direct comparisons ensures health expectancy measures as particularly useful in establishing public health priorities, when potential benefits from the competing programmes can be calculated and compared, leaving policy planning with the necessary information to make an informed choice.

The major application of the health expectancy methodology in New Zealand was reported extensively in this thesis. The versatility of health expectancy as a tool for policy research has also been demonstrated elsewhere by the present author (Cheung, 1999b). The vital information will be the way in which the relationships between life expectancy and health expectancy are changing, corresponding to scenarios of expansion or compression of morbidity. To take an example, insights into the relative impacts of future trends in disability and mortality on population health status may be gained by examining the elasticity of health expectancy (Cheung, 1999b). This is done by analysing the sensitivity of the health expectancy indices to a small (one percent) change in mortality and/or disability. The results of this analysis point to a greater effectiveness of strategies aimed at disability prevention than those aimed at postponing deaths, should maximising independent years of life be the policy goal. For females, the former would be twice as effective as the latter.

To realise fully the potential of health expectancy as a policy indicator and its inherent analytical strengths, drawn from life table techniques, further developments are required

in a number of areas. First, because of a lack of available data, health expectancies calculated for New Zealand thus far are merely cross-sectional observations. Repeated disability surveys using compatible survey instruments and methodology are needed in the future, preferably over regular intervals, to give comparable measures at different points in time. More detailed information on the causes of disability would be valuable for calculating cause-specific and/or cause-deleted health expectancy. This information is of direct policy value in informing the prioritisation process. However, given the relatively small population size in New Zealand and the likely high per capita cost in obtaining this information, findings from overseas studies may need to be drawn upon and interpolated into New Zealand data to provide imputations which would be of value for policy analyses in this area.

A simple two stage division is used in the computation of most health expectancies found in the literature. A person is either healthy or unhealthy depending on the definition of ill-health employed. Such a simple, and sometimes arbitrary, threshold does not adequately reflect the full spectrum of health. An indicator that includes all levels of ill-health / disability rather than just a single dependency threshold would provide a more precise measure of population health status. Development of this type of instruments is, however, in its infancy, and unavoidably involves subjective judgements on the relative values of different states of health.

A major extension of the health expectancy technique demonstrated in this thesis is the calculation of hospitalisation expectancies. Using available hospital data dating back to the middle of the twentieth century, a time series data set employing an integrated measure of population health was developed. Notwithstanding the inherent problems with using hospital utilisation data, the strength of this methodological extension is its applicability at sub-national levels. In another exercise and as a test of its analytical power and utility, hospitalisation expectancy was calculated by the present author for a population of around 137,000 for one New Zealand region. The results obtained were intuitively reasonable, suggesting that the methodology is robust also at the sub-national level. The wide applicability of this methodology is a particular advantage in many developing countries where population wide surveys on health are not available, but where hospital records might be, say for a capital city, as well as an estimate of the size

and structure of that population, plus life tables indirectly estimated by the so-called non-conventional techniques (Pool et al, 1999). Results of hospitalisation expectancies calculated using local data can be compared with those from other countries which have multiple-level sets of health expectancies calculated based on hospital utilisation as well as survey data. Techniques similar to indirect standardisation can then be applied to infer health expectancies with different thresholds for countries or areas where survey data are lacking.

There are two other very important differences between health and the hospitalisation expectancies developed in this thesis and alluded to in the last paragraph: the difference between sample survey data from sometimes irregular, or even once-off, surveys and population data from routinely collected sources. The use of population-based data for the hospitalisation expectancies means that numbers in cells are typically very large and thus problems akin to sampling error are avoided. Moreover, continuous and more timely hospitalisation expectancy series can be built up, as was demonstrated in this thesis.

The hospitalisation expectancy methods can provide planners with the tools and a sound evidence-base to plan services to meet emerging needs of the population, as well as to undertake theoretically robust yet empirically based analyses. For example, the ratio of persons-years exposure at ages x to x+n partitioned into the state of hospital stay (${}_nL^h_x$) to the number of survivors at that age (l_x) gives an estimated number of hospital days over the next n-years period. This ratio takes into account of both the incidence and the duration dimensions of hospitalisation, and the levels of mortality. This latter factor is particularly importantly when dealing with older ages where both the prevalence of hospitalisation and mortality levels are high. Ratios derived for each demographic sub-group can then be applied to the corresponding numbers in the population to estimate the number of hospital days.

Conventional analyses based on hospital admission and discharge rates alone, typically multiplying hospital rates by the numbers of population, effectively assume everyone in the population will survive to the end of the projected period, and thus artificially inflate the number of hospital days. The hospitalisation expectancy-based projection

methodology outlined above incorporates this mortality factor, and thus gives a far more reliable estimate of potential hospital use for financial and service planning. Moreover, instead of segregating the effects of mortality on population needs from those needs directly measured from utilisation data, as occurs in some other needs assessments tools such as QALYs, the analysis using hospitalisation expectancy integrates these two crucial components of population needs and their interactions at every age group. The assessment of the impacts of changing patterns of morbidity and mortality on hospital use is made possible with this measure.

15.3 Conclusion

Population health status is an increasingly major concern especially in societies with low levels of mortality. The noble sentiment of "good health for all" has been a celebrated political platform for at least two decades though its attainment is much difficult to realise — the slogan would have lost its political appeal if success had been achieved. This point merely highlights the difficulties in setting a realistic policy goal which also appeals to the general public.

This thesis has addressed, from both a theoretical and a methodological perspective, contemporary issues, predominantly of theoretical origin, in population health, and has demonstrated their policy relevance. Considerable knowledge about future population health dynamics can be gained from scientific studies on mortality and morbidity patterns and trends. Patterns and trends identified for the non-Maori population from the present study tend to support one of two diametrically opposing views on future evolution of population health. In the words of Hayward and Heron: "...'long life in good health' [an apparent rebuff to Verbrugge's longer life but worsening health (1984)] is not simply a theoretical construct but an empirical fact for a significant number of individuals in the population." (1999:89). Deriving policy mechanisms to achieve this goal is fundamental if good health is ever to be attainable for all.

APPENDICES

APPENDIX ONE:

Non-Maori Life Expectancies at Selected Ages from Synthetic Life Tables, by Gender, 1881-1996

APPENDIX ONE:
NON-MAORI LIFE EXPECTANCIES AT SELECTED AGES FROM SYNTHETIC LIFE TABLES, BY GENDER, 1881-1996

Non-Maori Males

Calendar year	Life expectancy at age					
	0	1	15	45	65	85
1881	52.9	57.7	48.0	24.9	12.4	
1886	54.0	59.0	48.6	25.5	12.4	
1891	55.2	59.9	49.2	25.3	11.9	
1896	57.4	61.8	50.4	26.2	12.4	
1901	57.6	61.8	50.6	26.0	12.0	
1906	58.8	63.0	51.5	26.3	12.1	3.9
1911	60.5	63.7	51.9	26.5	12.2	3.8
1916	60.7	63.3	51.7	27.1	12.5	4.0
1921	61.9	64.3	52.5	27.1	12.6	3.7
1926	63.7	65.6	53.4	27.3	12.5	3.9
1931	65.3	66.7	54.2	27.9	12.9	3.8
1936	65.5	66.9	54.4	27.8	12.8	3.9
1941	65.6	66.8	54.1	27.3	12.5	3.6
1945	66.7	67.9	55.0	27.8	12.7	3.9
1951	68.3	69.0	55.8	28.1	12.9	3.9
1956	68.9	69.5	56.1	28.3	12.9	4.1
1961	69.2	69.7	56.3	28.2	12.8	3.9
1966	68.7	69.0	55.6	27.7	12.6	4.2
1971	69.1	69.3	55.8	27.9	12.6	4.0
1976	69.4	69.5	56.0	28.2	12.9	3.9
1981	70.8	70.7	57.1	29.1	13.4	4.4
1986	71.4	71.2	57.6	29.7	13.7	4.6
1991	73.4	72.9	59.3	31.4	14.9	5.0
1996	75.3	74.7	61.0	32.8	15.8	5.1

Non-Maori Females

Calendar year	Life expectancy at age					
	0	1	15	45	65	85
1881	56.4	60.5	50.6	27.8	13.9	
1886	57.1	61.5	50.9	27.8	13.7	
1891	58.1	61.9	50.9	27.5	13.1	
1896	59.9	63.5	52.2	28.2	13.7	
1901	59.9	63.3	52.0	27.6	13.1	
1906	61.2	64.4	52.8	28.1	13.1	4.1
1911	63.2	65.7	53.6	28.3	13.2	4.4
1916	64.3	66.1	54.4	29.0	13.8	4.3
1921	64.3	66.1	54.1	28.7	13.5	4.2
1926	66.3	67.8	55.5	29.1	13.7	4.3
1931	67.8	68.8	56.0	29.5	13.8	4.1
1936	68.5	69.5	56.7	29.7	13.9	4.3
1941	69.1	69.9	56.9	29.5	13.7	4.2
1945	70.3	71.0	57.9	30.2	14.4	4.5
1951	72.4	72.9	59.5	31.1	14.8	4.2
1956	73.9	74.2	60.7	32.0	15.3	4.6
1961	74.5	74.7	61.2	32.4	15.5	4.5
1966	74.8	74.9	61.3	32.6	15.9	4.7
1971	75.2	75.2	61.6	32.8	16.0	4.7
1976	75.9	75.8	62.2	33.5	16.7	5.1
1981	76.9	76.6	63.0	34.2	17.2	5.5
1986	77.4	77.1	63.4	34.6	17.5	5.6
1991	79.2	78.7	64.9	36.0	18.6	6.1
1996	80.6	80.0	66.2	37.1	19.3	6.2

APPENDIX TWO:

Method for Estimating Life Cycle Stage Contribution to the Increases in Life Expectancy at Birth

APPENDIX TWO
METHOD FOR ESTIMATING LIFE CYCLE STAGE CONTRIBUTION TO THE INCREASES IN LIFE EXPECTANCY AT BIRTH

The contribution at each life cycle stage to the increases in life expectancy at birth (e_0), expressed as percentage of the overall increases in e_0, is calculated using the following method.

1. Let years x and y denote the beginning and the end of the time interval.
2. The difference in e_0 between synthetic life table of year x and synthetic life table of year y, representing the overall increases in life expectancy at birth, is calculated and is used as the denominator in the subsequent percentage calculations.
3. For each life cycle stage, a new e_0 for year x is estimated by substituting the corresponding ${}_nP_x$ from synthetic life table of year x with that from year y, and p_x's at all other ages are kept as the same at year x.
4. The difference between the estimated new e_0 (calculated specificly for each life cycle stage) and the old e_0 for year x, representing the increases in life expectancy at birth attributable to imprvements in survivorship at the respective life cycle stage alone, is then calculated to be the numerator.
5. Percentage contribution to the increases in e_0 through mortality reductions at each life cycle stage is calculated by dividing the numerator from step 4 by the denominator from step 2.

APPENDIX THREE:

Construction of Health and Hospitalisation Expectancies

APPENDIX THREE
CONSTRUCTION OF HEALTH AND HOSPITALISATION EXPECTANCIES

Disability-based health expectancies are caluclated by the following steps, using the Sullivan's observed prevalence life table method (1971):

1. Observed disability prevalence rates are extracted from the 1996/97 Disability Surveys by three levels of disability (Levels 1, 2 and 3), by age group (<5, 5-9, 10-14, 15-24, 25-29, 30-34, 35-39, ..., 80+), by gender and by ethnicity (total, Maori and non-Maori).

2. Level specific prevalence rates are re-organised into reverse incremental levels of disability, i.e., Level 3, Levels 2+3, Levels 1+2+3 (see Figures 9.1a and 9.1b).

3. An actuarial technique, known as graduation, is applied to the observed rates to smooth out random fluctuations between age groups and to estimate rates by five-year age group at ages 80 years and above. It was found a third-degree polynomial function has the best overall fit[1], and the best fit at the adult age range.
   Let ${}_n\delta_x^i$ denotes the graduated disability prevalence rate at age group x to x+n with level i or above disability, x = 0, ..., 100; i = 1,2 or 3.

4. Complete life tables from Statistics New Zealand for the non-Maori population, by gender, are abridged with age groups: <1, 1-4, 5-9, 10-14, ..., 100+.

5. At each age group, life table person-years exposure between exact ages x and x+n (${}_nL_x$) either with level i or above disability (denoted by ${}_nL^i{}_x$) or without level i or above disability (denoted by ${}_nL^{-i}{}_x$) are partitioned by:

   $${}_nL^i{}_x = {}_nL_x * {}_n\delta_x^i; \qquad {}_nL^{-i}{}_x = {}_nL_x - {}_nL^i{}_x$$

---

[1] The coefficient of determination, R^2, is also calculated which measures the adherence of data to the fitted curve. An R^2 value of 1 indicates perfect adherence of data. The R^2 calculated are 0.95 or more in all cases, indicating an excellent fit with at least 95% of the variations accounted for by the fitted curve.

6. Life table total person-years exposure at exact age x (T_x) either with or without level i or above disability (denoted by ${}^{i}T_x$ and ${}^{-i}T_x$ respectively) are calculated by:

$$ {}^{i}T_x = \Sigma_n {}^{i}L_x \quad ; \quad {}^{-i}T_x = \Sigma_n {}^{-i}L_x = T_x - {}^{i}T_x $$

7. Life expectancy at exact age x either with or without level i or above disability (denoted by ${}^{i}e_x$ and ${}^{-i}e_x$ respectively) are calculated by:

$$ {}^{i}e_x = {}^{i}T_x / l_x \quad ; \quad {}^{-i}e_x = {}^{-i}T_x / l_x = e_x - {}^{i}e_x $$

Hospitalisation expectancies are calculated by following the same procedures. Prevalence rates of hosptialisation by five-year age group, by gender, are estimated as the product of age-gender-specific discharge rate and age-gender-specific average length of stay (expressed in years to match with the life table ${}_nL_x$ function). The final results of hospitalisation expectancies (${}^{h}e_x$) are then converted into number of days.

APPENDIX FOUR:

Non-Maori Disability-Based Health Expectancies and Standard Errors, at Five-Year Age Interval, by Gender, 1996/97

APPENDIX FOUR:
NON-MAORI DISABILITY-BASED HEALTH EXPECTANCIES AND STANDARD ERRORS, AT FIVE-YEAR AGE INTERVAL, BY GENDER, 1996/97

Age	Non-Maori Males				Non-Maori Females			
	life exp.*	disability-free life exp. (DFLE)	independent life exp. (ILE)	active life exp. (ALE)	life exp.	disability-free life exp. (DFLE)	independent life exp. (ILE)	active life exp. (ALE)
0	75.3	58.7	65.6	73.1	80.6	62.0	68.9	76.9
1	74.7	58.1	65.1	72.5	80.0	61.4	68.3	76.3
5	70.9	54.6	61.5	68.7	76.1	57.7	64.4	72.4
10	65.9	50.2	56.9	63.8	71.1	53.2	59.7	67.5
15	61.0	45.8	52.3	59.0	66.2	48.7	55.1	62.7
20	56.3	41.5	47.8	54.4	61.4	44.3	50.5	58.0
25	51.7	37.3	43.4	49.9	56.5	39.9	46.0	53.3
30	47.0	33.1	39.0	45.3	51.6	35.5	41.4	48.5
35	42.3	28.9	34.4	40.6	46.8	31.3	36.9	43.7
40	37.5	24.8	29.9	35.8	41.9	27.1	32.4	39.0
45	32.8	20.8	25.4	31.2	37.1	23.1	28.0	34.3
50	28.2	17.0	21.1	26.6	32.4	19.3	23.6	29.6
55	23.8	13.5	17.0	22.2	27.9	15.7	19.5	25.1
60	19.6	10.4	13.3	18.1	23.5	12.3	15.6	20.8
65	15.8	7.6	9.9	14.3	19.3	9.3	11.9	16.7
70	12.4	5.3	7.1	11.0	15.4	6.7	8.7	13.0
75	9.4	3.5	4.8	8.1	11.9	4.5	5.9	9.6
80	7.0	2.1	2.9	5.8	8.8	2.8	3.7	6.8
				Standard Error				
0		0.7198	0.7406	0.5855		0.7319	0.7650	0.7716
1		0.7212	0.7422	0.5869		0.7328	0.7676	0.7736
5		0.7129	0.7345	0.5809		0.7238	0.7638	0.7694
10		0.7076	0.7291	0.5737		0.7166	0.7573	0.7622
15		0.7025	0.7240	0.5637		0.7081	0.7469	0.7405
20		0.6425	0.6532	0.5128		0.6660	0.6930	0.6662
25		0.5745	0.5793	0.4627		0.6182	0.6327	0.5789
30		0.5421	0.5579	0.4453		0.5812	0.6009	0.5407
35		0.5043	0.5283	0.4292		0.5458	0.5679	0.5126
40		0.4695	0.5024	0.4177		0.5066	0.5371	0.4936
45		0.4346	0.4775	0.4092		0.4635	0.4992	0.4599
50		0.3843	0.4345	0.3968		0.4208	0.4686	0.4438
55		0.3353	0.3822	0.3820		0.3636	0.4215	0.4218
60		0.2605	0.3181	0.3546		0.2928	0.3420	0.3770
65		0.2006	0.2546	0.3282		0.2005	0.2490	0.3300
70		0.1596	0.2057	0.2858		0.1547	0.1972	0.2884
75		0.1205	0.1576	0.2573		0.1150	0.1449	0.2353
80		0.0535	0.0882	0.2458		0.0572	0.0781	0.1827

* exp. = expectancy

APPENDIX FIVE:

Non-Maori Hospitalisation Expectancies, at Five-Year Age Interval, by Gender, 1996-98

APPENDIX FIVE:
NON-MAORI HOSPITALISATION EXPECTANCIES, AT FIVE-YEAR AGE INTERVAL, BY GENDER, 1996-98

Age	Hospitalisation Expectancy (in days)	
	non-Maori males	non-Maori females
0	62.5	68.9
1	59.3	66.2
5	58.3	65.5
10	57.6	64.9
15	56.8	64.2
20	56.0	63.4
25	55.0	62.4
30	54.0	61.5
35	53.1	60.4
40	52.2	59.4
45	51.3	58.2
50	50.3	56.9
55	49.2	55.5
60	47.7	53.7
65	45.6	51.4
70	42.8	48.2
75	39.5	44.2
80	35.1	38.5
85	30.2	30.3
90	23.4	21.3

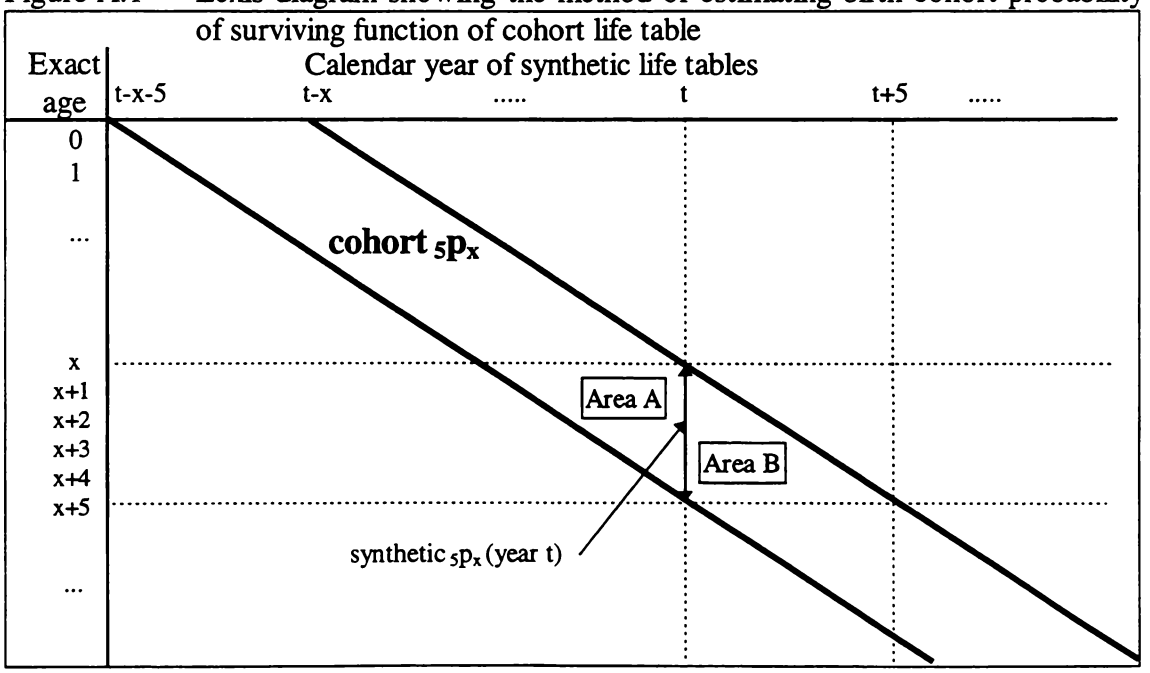
APPENDIX SIX:

Construction of Non-Maori Cohort Life Tables

APPENDIX SIX:
CONSTRUCTION OF NON-MAORI COHORT LIFE TABLES

Cohort life tables are constructed based on the abridged synthetic life tables analysed in Chapter Eight. The probability of each birth cohort surviving from exact age x to x+5 is estimated from synthetic life tables using the method illustrated in Figure A.1. For cohort born in the five years from t-x-5 to t-x, the probability of surviving between exact ages x and x+5 is estimated by the corresponding probability of surviving between exact ages x and x+5 from the synthetic life table in year t. It is assumed that the birth cohort entering age group x to x+5 and exposed to the risk of dying between years t-5 and t (denoted by the triangular area A) experienced the same age specific survival rate ($_5p_x$) as estimated from the synthetic life table in year t. Similarly, those leaving age group x to x+5 and exposed to the risk of dying between years t and t+5 (denoted by the triangular area B) are assumed to experience the same age specific survival rate ($_5p_x$) estimated for the synthetic life table in year t.

Figure A.1 Lexis diagram showing the method of estimating birth cohort probability of surviving function of cohort life table



Sources: Derived by the author

The estimation of cohort survivorship for the first year of life is a minor exception to this formula. It is taken from the average for years t-x+5 and t-x of the probability of surviving from birth to exact age one, assuming a linear trend in infant mortality rate during the interval.

This method of estimating cohort survivorship is much simpler than some other approaches, such as the "extinct cohort" method (see Kannisto, 1988), which follows through the mortality experience of a birth cohort from birth to death year after year. The major drawback of the simpler approach used here is that, rather than making direct measurements, the mortality experience during the inter-censal period is approximated from that around the census year. With some minor exceptions, and one major exception (influenza 1917-19), of epidemics in the early years, large annual fluctuations in non-Maori mortality rates have generally disappeared over the last century. There is, however, also the impact noted earlier of World War One deaths of males aged 15 to 44 years. This simpler approach is adequate for the purposes of this thesis of studying a long term demographic phenomenon.

APPENDIX SEVEN:

Non-Maori Cohort Life Tables, by Five-Year Birth Interval, by Gender, 1871-1961

APPENDIX SEVEN: NON-MAORI COHORT LIFE TABLES, BY FIVE-YEAR BIRTH INTERVAL, BY GENDER, 1871-1961

Cohort Born 1871-76

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.88670	92,069	5,445,933	54.46
1-4	88,670	0.94068	344,158	5,353,864	60.38
5-9	83,409	0.97942	412,756	5,009,706	60.06
10-14	81,693	0.98804	406,021	4,596,950	56.27
15-19	80,716	0.98165	399,875	4,190,929	51.92
20-24	79,234	0.97754	391,723	3,791,054	47.85
25-29	77,455	0.97820	383,052	3,399,331	43.89
30-34	75,766	0.97768	374,601	3,016,279	39.81
35-39	74,075	0.97292	365,357	2,641,678	35.66
40-44	72,068	0.96748	354,483	2,276,321	31.59
45-49	69,725	0.95792	341,289	1,921,838	27.56
50-54	66,791	0.94774	325,226	1,580,549	23.66
55-59	63,300	0.92996	305,417	1,255,323	19.83
60-64	58,867	0.88785	277,828	949,906	16.14
65-69	52,265	0.82377	238,297	672,078	12.86
70-74	43,054	0.73757	187,024	433,780	10.08
75-79	31,756	0.63064	129,455	246,756	7.77
80-84	20,026	0.49214	74,705	117,301	5.86
85-89	9,856	0.30176	32,075	42,596	4.32
90-94	2,974	0.19687	8,899	10,522	3.54
95-99	585	0.05432	1,543	1,623	2.77
100+	32		80	80	2.50

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.90460	93,322	5,768,920	57.69
1-4	90,460	0.94383	351,676	5,675,599	62.74
5-9	85,378	0.98333	423,333	5,323,923	62.36
10-14	83,955	0.98876	417,415	4,900,590	58.37
15-19	83,011	0.98185	411,288	4,483,175	54.01
20-24	81,504	0.97884	403,210	4,071,887	49.96
25-29	79,780	0.97724	394,360	3,668,677	45.99
30-34	77,964	0.97612	385,167	3,274,317	42.00
35-39	76,103	0.97412	375,589	2,889,150	37.96
40-44	74,133	0.97373	365,796	2,513,561	33.91
45-49	72,185	0.96562	354,723	2,147,765	29.75
50-54	69,704	0.95537	340,742	1,793,042	25.72
55-59	66,593	0.94123	323,180	1,452,300	21.81
60-64	62,679	0.91265	299,708	1,129,120	18.01
65-69	57,204	0.85900	265,856	829,412	14.50
70-74	49,138	0.79073	219,984	563,556	11.47
75-79	38,855	0.69370	164,523	343,571	8.84
80-84	26,954	0.56192	105,249	179,049	6.64
85-89	15,146	0.38006	52,256	73,800	4.87
90-94	5,756	0.23124	17,719	21,544	3.74
95-99	1,331	0.07451	3,576	3,825	2.87
100+	99		249	249	2.51

Cohort Born 1876-81

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.89356	92,549	5,657,668	56.58
1-4	89,356	0.95699	349,739	5,565,119	62.28
5-9	85,513	0.98340	424,017	5,215,381	60.99
10-14	84,094	0.99019	418,407	4,791,363	56.98
15-19	83,269	0.98472	413,164	4,372,956	52.52
20-24	81,996	0.98065	406,015	3,959,792	48.29
25-29	80,410	0.98128	398,285	3,553,777	44.20
30-34	78,905	0.97904	390,388	3,155,492	39.99
35-39	77,251	0.97162	380,772	2,765,104	35.79
40-44	75,058	0.96830	369,342	2,384,332	31.77
45-49	72,679	0.96152	356,402	2,014,990	27.72
50-54	69,882	0.94953	340,592	1,658,588	23.73
55-59	66,355	0.92717	319,693	1,317,995	19.86
60-64	61,522	0.88381	289,742	998,302	16.23
65-69	54,374	0.83039	248,816	708,560	13.03
70-74	45,152	0.74828	197,345	459,744	10.18
75-79	33,786	0.63108	137,770	262,399	7.77
80-84	21,322	0.47893	78,833	124,629	5.85
85-89	10,212	0.33287	34,027	45,796	4.48
90-94	3,399	0.18256	10,049	11,770	3.46
95-99	621	0.05285	1,633	1,720	2.77
100+	33		85	87	2.65

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.91094	93,765	6,012,811	60.13
1-4	91,094	0.95899	356,902	5,919,045	64.98
5-9	87,358	0.98677	433,898	5,562,143	63.67
10-14	86,202	0.99042	428,943	5,128,245	59.49
15-19	85,376	0.98435	423,538	4,699,301	55.04
20-24	84,040	0.98158	416,329	4,275,763	50.88
25-29	82,492	0.97814	407,952	3,859,434	46.79
30-34	80,689	0.97816	399,038	3,451,482	42.78
35-39	78,926	0.97724	390,141	3,052,445	38.67
40-44	77,130	0.97308	380,459	2,662,303	34.52
45-49	75,053	0.96791	369,246	2,281,844	30.40
50-54	72,645	0.96056	356,062	1,912,598	26.33
55-59	69,780	0.94385	339,104	1,556,536	22.31
60-64	65,861	0.91105	314,661	1,217,433	18.48
65-69	60,003	0.87412	281,132	902,772	15.05
70-74	52,450	0.81028	237,372	621,640	11.85
75-79	42,499	0.71199	181,895	384,268	9.04
80-84	30,259	0.56187	118,152	202,373	6.69
85-89	17,002	0.39374	59,240	84,221	4.95
90-94	6,694	0.23080	20,598	24,981	3.73
95-99	1,545	0.06458	4,112	4,382	2.84
100+	100		262	270	2.71

Cohort Born 1881-86

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.89984	92,989	5,773,217	57.73
1-4	89,984	0.96419	353,490	5,680,228	63.13
5-9	86,761	0.98539	430,637	5,326,739	61.40
10-14	85,494	0.99075	425,491	4,896,102	57.27
15-19	84,703	0.98566	420,477	4,470,610	52.78
20-24	83,488	0.98217	413,718	4,050,133	48.51
25-29	81,999	0.98284	406,478	3,636,415	44.35
30-34	80,592	0.97663	398,252	3,229,937	40.08
35-39	78,709	0.97400	388,427	2,831,686	35.98
40-44	76,662	0.97386	378,301	2,443,259	31.87
45-49	74,658	0.96526	366,809	2,064,958	27.66
50-54	72,065	0.95098	351,494	1,698,149	23.56
55-59	68,532	0.92135	329,187	1,346,655	19.65
60-64	63,142	0.88355	297,329	1,017,469	16.11
65-69	55,789	0.82684	254,795	720,139	12.91
70-74	46,129	0.74321	201,030	465,344	10.09
75-79	34,283	0.62749	139,490	264,314	7.71
80-84	21,513	0.47943	79,566	124,824	5.80
85-89	10,314	0.31851	33,997	45,258	4.39
90-94	3,285	0.16870	9,598	11,262	3.43
95-99	554	0.09706	1,520	1,664	3.00
100+	54		140	144	2.67

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.91539	94,077	6,174,021	61.74
1-4	91,539	0.96718	360,146	6,079,944	66.42
5-9	88,534	0.98663	439,712	5,719,797	64.61
10-14	87,350	0.99174	434,949	5,280,086	60.45
15-19	86,629	0.98520	429,940	4,845,137	55.93
20-24	85,347	0.98229	422,956	4,415,197	51.73
25-29	83,835	0.98114	415,223	3,992,241	47.62
30-34	82,254	0.97965	407,084	3,577,018	43.49
35-39	80,580	0.97672	398,209	3,169,934	39.34
40-44	78,704	0.97897	389,381	2,771,725	35.22
45-49	77,048	0.96984	379,433	2,382,345	30.92
50-54	74,725	0.96194	366,514	2,002,911	26.80
55-59	71,881	0.94240	349,052	1,636,397	22.77
60-64	67,740	0.92043	325,225	1,287,346	19.00
65-69	62,350	0.88285	293,489	962,121	15.43
70-74	55,046	0.82342	250,929	668,632	12.15
75-79	45,326	0.71460	194,289	417,703	9.22
80-84	32,390	0.58382	128,249	223,414	6.90
85-89	18,910	0.39662	66,025	95,165	5.03
90-94	7,500	0.24174	23,283	29,140	3.89
95-99	1,813	0.14015	5,168	5,857	3.23
100+	254		667	689	2.71

Cohort Born 1886-91

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.90233	93,163	5,869,814	58.70
1-4	90,233	0.96747	355,060	5,776,651	64.02
5-9	87,298	0.98968	434,237	5,421,591	62.10
10-14	86,397	0.99145	430,139	4,987,354	57.73
15-19	85,659	0.98746	425,607	4,557,215	53.20
20-24	84,584	0.98515	419,782	4,131,607	48.85
25-29	83,328	0.97911	412,290	3,711,825	44.54
30-34	81,587	0.97930	403,715	3,299,536	40.44
35-39	79,899	0.97825	395,150	2,895,820	36.24
40-44	78,161	0.97499	385,918	2,500,671	31.99
45-49	76,206	0.96697	374,738	2,114,753	27.75
50-54	73,689	0.94746	358,767	1,740,015	23.61
55-59	69,818	0.92830	336,573	1,381,248	19.78
60-64	64,812	0.88786	305,889	1,044,674	16.12
65-69	57,544	0.82689	262,815	738,786	12.84
70-74	47,582	0.74333	207,379	475,971	10.00
75-79	35,369	0.61699	142,979	268,592	7.59
80-84	21,823	0.47407	80,420	125,613	5.76
85-89	10,345	0.30447	33,738	45,193	4.37
90-94	3,150	0.20274	9,471	11,455	3.64
95-99	639	0.11664	1,783	1,984	3.11
100+	74		195	202	2.71

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.91789	94,252	6,321,164	63.21
1-4	91,789	0.97277	362,155	6,226,912	67.84
5-9	89,289	0.98958	444,118	5,864,757	65.68
10-14	88,358	0.99106	439,816	5,420,639	61.35
15-19	87,568	0.98691	434,975	4,980,823	56.88
20-24	86,422	0.98526	428,924	4,545,849	52.60
25-29	85,148	0.98046	421,578	4,116,925	48.35
30-34	83,484	0.98000	413,245	3,695,346	44.26
35-39	81,814	0.98163	405,313	3,282,102	40.12
40-44	80,311	0.98048	397,635	2,876,789	35.82
45-49	78,743	0.97234	388,271	2,479,154	31.48
50-54	76,565	0.96277	375,700	2,090,884	27.31
55-59	73,715	0.94439	358,326	1,715,183	23.27
60-64	69,616	0.92343	334,752	1,356,857	19.49
65-69	64,285	0.89015	303,772	1,022,105	15.90
70-74	57,223	0.82833	261,558	718,334	12.55
75-79	47,400	0.73539	205,642	456,776	9.64
80-84	34,857	0.58917	138,484	251,133	7.20
85-89	20,537	0.44557	74,218	112,649	5.49
90-94	9,151	0.29267	29,571	38,431	4.20
95-99	2,678	0.15210	7,713	8,860	3.31
100+	407		1,094	1,146	2.81

Cohort Born 1891-96

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.90958	93,218	5,961,444	59.61
1-4	90,958	0.97409	359,118	5,868,226	64.52
5-9	88,601	0.98829	440,412	5,509,108	62.18
10-14	87,564	0.99186	436,037	5,068,696	57.89
15-19	86,851	0.98894	431,853	4,632,658	53.34
20-24	85,890	0.97602	424,302	4,200,805	48.91
25-29	83,830	0.98314	415,618	3,776,503	45.05
30-34	82,417	0.98274	408,529	3,360,885	40.78
35-39	80,995	0.98064	401,053	2,952,356	36.45
40-44	79,426	0.97521	392,209	2,551,303	32.12
45-49	77,457	0.96793	381,075	2,159,094	27.87
50-54	74,973	0.95358	366,163	1,778,019	23.72
55-59	71,492	0.92611	344,255	1,411,857	19.75
60-64	66,210	0.88689	312,326	1,067,602	16.12
65-69	58,721	0.82816	268,378	755,276	12.86
70-74	48,630	0.72952	210,268	486,898	10.01
75-79	35,477	0.61503	143,241	276,630	7.80
80-84	21,819	0.49784	81,704	133,389	6.11
85-89	10,862	0.35756	36,866	51,685	4.76
90-94	3,884	0.23050	11,948	14,819	3.82
95-99	895	0.13868	2,549	2,871	3.21
100+	124		318	322	2.60

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.92517	94,388	6,476,551	64.77
1-4	92,517	0.97476	365,398	6,382,163	68.98
5-9	90,182	0.99054	448,777	6,016,765	66.72
10-14	89,329	0.99207	444,873	5,567,988	62.33
15-19	88,620	0.98899	440,663	5,123,115	57.81
20-24	87,645	0.98479	434,891	4,682,452	53.43
25-29	86,312	0.98078	427,412	4,247,561	49.21
30-34	84,653	0.98353	419,781	3,820,149	45.13
35-39	83,259	0.98290	412,737	3,400,368	40.84
40-44	81,835	0.97957	404,999	2,987,631	36.51
45-49	80,164	0.97367	395,543	2,582,632	32.22
50-54	78,053	0.96204	382,859	2,187,089	28.02
55-59	75,090	0.95025	366,113	1,804,230	24.03
60-64	71,355	0.93259	344,749	1,438,117	20.15
65-69	66,545	0.89882	315,893	1,093,368	16.43
70-74	59,812	0.83660	274,627	777,475	13.00
75-79	50,039	0.73797	217,414	502,847	10.05
80-84	36,927	0.62881	150,368	285,433	7.73
85-89	23,220	0.48920	86,448	135,065	5.82
90-94	11,359	0.29818	36,866	48,617	4.28
95-99	3,387	0.18849	10,064	11,751	3.47
100+	638		1,650	1,687	2.64

Cohort Born 1896-01

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.91535	93,651	6,072,558	60.73
1-4	91,535	0.97216	361,042	5,978,907	65.32
5-9	88,986	0.98875	442,430	5,617,864	63.13
10-14	87,986	0.99192	438,150	5,175,434	58.82
15-19	87,275	0.98807	433,771	4,737,283	54.28
20-24	86,234	0.98459	427,848	4,303,513	49.91
25-29	84,905	0.98549	421,446	3,875,665	45.65
30-34	83,673	0.98568	415,371	3,454,219	41.28
35-39	82,475	0.98234	408,734	3,038,848	36.85
40-44	81,018	0.97844	400,724	2,630,113	32.46
45-49	79,271	0.97168	390,744	2,229,389	28.12
50-54	77,026	0.95535	376,534	1,838,645	23.87
55-59	73,587	0.92934	354,937	1,462,111	19.87
60-64	68,388	0.88544	322,352	1,107,174	16.19
65-69	60,553	0.81941	275,427	784,822	12.96
70-74	49,618	0.73568	215,302	509,395	10.27
75-79	36,503	0.63100	148,841	294,093	8.06
80-84	23,033	0.51053	86,981	145,252	6.31
85-89	11,759	0.37633	40,461	58,271	4.96
90-94	4,425	0.27319	14,086	17,810	4.02
95-99	1,209	0.11291	3,364	3,724	3.08
100+	137		353	360	2.64

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.92925	94,694	6,612,674	66.13
1-4	92,925	0.97362	366,798	6,517,979	70.14
5-9	90,474	0.99134	450,410	6,151,181	67.99
10-14	89,690	0.99439	447,193	5,700,771	63.56
15-19	89,187	0.98996	443,694	5,253,579	58.91
20-24	88,291	0.98488	438,117	4,809,885	54.48
25-29	86,956	0.98635	431,811	4,371,768	50.28
30-34	85,769	0.98393	425,397	3,939,957	45.94
35-39	84,390	0.98520	418,829	3,514,560	41.65
40-44	83,142	0.98266	412,103	3,095,731	37.23
45-49	81,700	0.97350	403,086	2,683,627	32.85
50-54	79,535	0.96748	391,206	2,280,541	28.67
55-59	76,948	0.95803	376,666	1,889,335	24.55
60-64	73,719	0.93593	356,784	1,512,670	20.52
65-69	68,995	0.89712	327,230	1,155,886	16.75
70-74	61,897	0.83985	284,703	828,655	13.39
75-79	51,984	0.76166	228,947	543,952	10.46
80-84	39,594	0.64715	163,045	315,005	7.96
85-89	25,624	0.49048	95,479	151,960	5.93
90-94	12,568	0.34312	42,200	56,482	4.49
95-99	4,312	0.15591	12,462	14,281	3.31
100+	672		1,763	1,820	2.71

Cohort Born 1901-06

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.91819	93,864	6,194,483	61.94
1-4	91,819	0.97721	363,093	6,100,619	66.44
5-9	89,727	0.99051	446,507	5,737,526	63.94
10-14	88,876	0.99232	442,671	5,291,020	59.53
15-19	88,193	0.98986	438,728	4,848,349	54.97
20-24	87,299	0.98670	433,591	4,409,621	50.51
25-29	86,138	0.98687	427,862	3,976,030	46.16
30-34	85,007	0.98759	422,399	3,548,168	41.74
35-39	83,952	0.98504	416,622	3,125,769	37.23
40-44	82,697	0.98292	409,952	2,709,147	32.76
45-49	81,284	0.97333	401,001	2,299,195	28.29
50-54	79,116	0.95731	387,137	1,898,193	23.99
55-59	75,738	0.92957	365,357	1,511,056	19.95
60-64	70,404	0.88093	331,065	1,145,699	16.27
65-69	62,022	0.81869	281,995	814,634	13.13
70-74	50,776	0.73655	220,440	532,639	10.49
75-79	37,399	0.64528	153,831	312,199	8.35
80-84	24,133	0.52340	91,911	158,368	6.56
85-89	12,631	0.42275	44,927	66,457	5.26
90-94	5,340	0.26875	16,937	21,530	4.03
95-99	1,435	0.13588	4,075	4,593	3.20
100+	195		505	518	2.66

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.93264	94,948	6,752,309	67.52
1-4	93,264	0.97701	368,769	6,657,361	71.38
5-9	91,120	0.99179	453,731	6,288,592	69.01
10-14	90,372	0.99344	450,379	5,834,861	64.56
15-19	89,779	0.98981	446,610	5,384,483	59.97
20-24	88,865	0.98763	441,575	4,937,873	55.57
25-29	87,766	0.98675	435,921	4,496,297	51.23
30-34	86,603	0.98720	430,243	4,060,376	46.89
35-39	85,494	0.98597	424,473	3,630,133	42.46
40-44	84,295	0.98462	418,233	3,205,660	38.03
45-49	82,999	0.97878	410,589	2,787,427	33.58
50-54	81,237	0.97144	400,385	2,376,837	29.26
55-59	78,917	0.96038	386,767	1,976,453	25.04
60-64	75,790	0.93467	366,570	1,589,686	20.97
65-69	70,838	0.90223	336,877	1,223,116	17.27
70-74	63,913	0.84679	295,083	886,238	13.87
75-79	54,120	0.77951	240,769	591,156	10.92
80-84	42,187	0.66045	175,125	350,386	8.31
85-89	27,863	0.53600	106,993	175,261	6.29
90-94	14,934	0.34383	50,173	68,269	4.57
95-99	5,135	0.19504	15,341	18,095	3.52
100+	1,002		2,653	2,754	2.75

Note: Figures below the broken line denote projected numbers

Cohort Born 1906-11

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.92731	94,548	6,317,816	63.18
1-4	92,731	0.98087	367,376	6,223,268	67.11
5-9	90,957	0.98775	452,001	5,855,891	64.38
10-14	89,843	0.99238	447,504	5,403,891	60.15
15-19	89,158	0.99075	443,730	4,956,387	55.59
20-24	88,334	0.98735	438,875	4,512,657	51.09
25-29	87,217	0.98837	433,546	4,073,782	46.71
30-34	86,202	0.98710	428,229	3,640,236	42.23
35-39	85,090	0.98794	422,884	3,212,006	37.75
40-44	84,064	0.98431	417,022	2,789,123	33.18
45-49	82,745	0.97576	408,710	2,372,101	28.67
50-54	80,739	0.95742	395,100	1,963,391	24.32
55-59	77,301	0.92299	371,622	1,568,292	20.29
60-64	71,348	0.88101	335,514	1,196,670	16.77
65-69	62,858	0.83000	287,576	861,155	13.70
70-74	52,172	0.75924	229,460	573,579	10.99
75-79	39,612	0.65780	164,171	344,120	8.69
80-84	26,057	0.55785	101,481	179,949	6.91
85-89	14,536	0.43557	52,168	78,468	5.40
90-94	6,331	0.28619	20,358	26,300	4.15
95-99	1,812	0.14960	5,208	5,942	3.28
100+	271		711	734	2.71

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.94174	95,631	6,958,587	69.59
1-4	94,174	0.98201	373,308	6,862,957	72.88
5-9	92,480	0.98972	460,023	6,489,649	70.17
10-14	91,529	0.99408	456,291	6,029,625	65.88
15-19	90,987	0.99217	453,155	5,573,334	61.25
20-24	90,275	0.98948	448,998	5,120,179	56.72
25-29	89,325	0.98892	444,149	4,671,180	52.29
30-34	88,335	0.98890	439,223	4,227,031	47.85
35-39	87,354	0.98762	434,067	3,787,808	43.36
40-44	86,273	0.98733	428,629	3,353,741	38.87
45-49	85,179	0.98273	422,218	2,925,112	34.34
50-54	83,708	0.97525	413,360	2,502,894	29.90
55-59	81,636	0.95931	399,876	2,089,534	25.60
60-64	78,314	0.93905	379,638	1,689,657	21.58
65-69	73,541	0.90961	351,086	1,310,019	17.81
70-74	66,893	0.85992	311,042	958,933	14.34
75-79	57,523	0.78110	256,136	647,891	11.26
80-84	44,931	0.68951	189,780	391,755	8.72
85-89	30,981	0.54851	119,935	201,975	6.52
90-94	16,993	0.37974	58,616	82,040	4.83
95-99	6,453	0.21332	19,574	23,425	3.63
100+	1,377		3,685	3,850	2.80

Note: Figures below the broken line denote projected numbers

Cohort Born 1911-16

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.93967	95,475	6,465,328	64.65
1-4	93,967	0.97882	371,887	6,369,853	67.79
5-9	91,977	0.98975	457,528	5,997,965	65.21
10-14	91,035	0.99379	453,760	5,540,437	60.86
15-19	90,470	0.99063	450,229	5,086,677	56.23
20-24	89,622	0.98862	445,561	4,636,447	51.73
25-29	88,602	0.98749	440,239	4,190,887	47.30
30-34	87,494	0.98969	435,213	3,750,647	42.87
35-39	86,592	0.98824	430,411	3,315,434	38.29
40-44	85,573	0.98552	424,766	2,885,023	33.71
45-49	84,334	0.97417	416,222	2,460,257	29.17
50-54	82,155	0.95342	401,209	2,044,035	24.88
55-59	78,328	0.92614	377,179	1,642,826	20.97
60-64	72,543	0.88797	342,399	1,265,647	17.45
65-69	64,416	0.83862	296,093	923,249	14.33
70-74	54,021	0.76732	238,680	627,156	11.61
75-79	41,451	0.69969	176,135	388,476	9.37
80-84	29,003	0.59190	115,424	212,341	7.32
85-89	17,167	0.46451	62,853	96,917	5.65
90-94	7,974	0.30331	25,982	34,064	4.27
95-99	2,419	0.16134	7,022	8,082	3.34
100+	390		1,026	1,060	2.72

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.95275	96,457	7,143,603	71.44
1-4	95,275	0.98078	377,439	7,047,146	73.97
5-9	93,444	0.99156	465,248	6,669,708	71.38
10-14	92,655	0.99436	461,970	6,204,459	66.96
15-19	92,133	0.99277	458,997	5,742,489	62.33
20-24	91,466	0.99076	455,219	5,283,492	57.76
25-29	90,621	0.99029	450,907	4,828,272	53.28
30-34	89,741	0.99118	446,728	4,377,366	48.78
35-39	88,950	0.99150	442,859	3,930,637	44.19
40-44	88,194	0.98940	438,631	3,487,778	39.55
45-49	87,259	0.98393	432,789	3,049,147	34.94
50-54	85,857	0.97319	423,529	2,616,359	30.47
55-59	83,555	0.96153	409,739	2,192,829	26.24
60-64	80,340	0.94044	389,739	1,783,090	22.19
65-69	75,555	0.90844	360,482	1,393,351	18.44
70-74	68,637	0.86346	319,758	1,032,870	15.05
75-79	59,266	0.81552	268,996	713,111	12.03
80-84	48,332	0.71872	207,674	444,115	9.19
85-89	34,737	0.57354	136,652	236,441	6.81
90-94	19,923	0.40187	69,825	99,789	5.01
95-99	8,007	0.23244	24,669	29,965	3.74
100+	1,861		5,035	5,296	2.85

Note: Figures below the broken line denote projected numbers

Cohort Born 1916-21

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.94603	95,952	6,617,357	66.17
1-4	94,603	0.98028	374,680	6,521,405	68.93
5-9	92,737	0.99200	461,831	6,146,725	66.28
10-14	91,995	0.99474	458,769	5,684,894	61.80
15-19	91,512	0.99094	455,488	5,226,125	57.11
20-24	90,683	0.98609	450,263	4,770,637	52.61
25-29	89,422	0.98895	444,639	4,320,374	48.31
30-34	88,434	0.99116	440,212	3,875,736	43.83
35-39	87,651	0.99017	436,104	3,435,523	39.20
40-44	86,790	0.98515	430,727	2,999,420	34.56
45-49	85,501	0.97234	421,593	2,568,692	30.04
50-54	83,136	0.95588	406,510	2,147,100	25.83
55-59	79,468	0.93018	383,467	1,740,590	21.90
60-64	73,919	0.89611	350,397	1,357,122	18.36
65-69	66,240	0.84859	306,125	1,006,725	15.20
70-74	56,210	0.80236	253,278	700,600	12.46
75-79	45,101	0.72975	195,033	447,322	9.92
80-84	32,912	0.61879	133,195	252,290	7.67
85-89	20,366	0.48423	75,568	119,094	5.85
90-94	9,862	0.32349	32,630	43,526	4.41
95-99	3,190	0.17469	9,369	10,896	3.42
100+	557		1,473	1,528	2.74

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.95785	96,838	7,328,420	73.28
1-4	95,785	0.98178	379,648	7,231,581	75.50
5-9	94,040	0.99321	468,603	6,851,933	72.86
10-14	93,402	0.99671	466,238	6,383,330	68.34
15-19	93,094	0.99401	464,075	5,917,092	63.56
20-24	92,536	0.99202	460,835	5,453,017	58.93
25-29	91,798	0.99180	457,106	4,992,182	54.38
30-34	91,045	0.99390	453,835	4,535,076	49.81
35-39	90,489	0.99249	450,748	4,081,241	45.10
40-44	89,810	0.99012	446,830	3,630,493	40.42
45-49	88,922	0.98244	440,709	3,183,663	35.80
50-54	87,361	0.97426	431,185	2,742,954	31.40
55-59	85,113	0.96278	417,644	2,311,770	27.16
60-64	81,945	0.94589	398,640	1,894,125	23.11
65-69	77,511	0.91716	371,502	1,495,486	19.29
70-74	71,090	0.88299	334,654	1,123,983	15.81
75-79	62,772	0.83168	287,445	789,329	12.57
80-84	52,206	0.73959	227,043	501,884	9.61
85-89	38,611	0.60145	154,584	274,842	7.12
90-94	23,223	0.42067	82,480	120,258	5.18
95-99	9,769	0.25435	30,635	37,778	3.87
100+	2,485		6,766	7,144	2.87

Note: Figures below the broken line denote projected numbers

Cohort Born 1921-26

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.95224	96,179	6,824,501	68.25
1-4	95,224	0.98524	378,084	6,728,323	70.66
5-9	93,818	0.99307	467,466	6,350,239	67.69
10-14	93,168	0.99464	464,590	5,882,773	63.14
15-19	92,668	0.99199	461,487	5,418,183	58.47
20-24	91,926	0.98758	456,776	4,956,696	53.92
25-29	90,784	0.99244	452,207	4,499,920	49.57
30-34	90,098	0.99210	448,712	4,047,713	44.93
35-39	89,386	0.99037	444,781	3,599,001	40.26
40-44	88,526	0.98436	439,167	3,154,220	35.63
45-49	87,141	0.97441	430,129	2,715,053	31.16
50-54	84,911	0.95662	415,345	2,284,923	26.91
55-59	81,227	0.93596	393,132	1,869,578	23.02
60-64	76,025	0.90532	362,132	1,476,446	19.42
65-69	68,827	0.87258	322,212	1,114,315	16.19
70-74	60,058	0.82618	274,190	792,103	13.19
75-79	49,619	0.75639	217,874	517,913	10.44
80-84	37,531	0.64438	154,289	300,038	7.99
85-89	24,184	0.50212	90,820	145,750	6.03
90-94	12,143	0.33679	40,583	54,930	4.52
95-99	4,090	0.19109	12,178	14,347	3.51
100+	782		2,082	2,169	2.78

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.96175	96,940	7,490,329	74.90
1-4	96,175	0.98541	381,892	7,393,390	76.87
5-9	94,771	0.99408	472,453	7,011,498	73.98
10-14	94,210	0.99594	470,092	6,539,045	69.41
15-19	93,827	0.99540	468,055	6,068,953	64.68
20-24	93,395	0.99310	465,364	5,600,898	59.97
25-29	92,750	0.99519	462,638	5,135,535	55.37
30-34	92,305	0.99558	460,505	4,672,897	50.62
35-39	91,897	0.99319	457,921	4,212,392	45.84
40-44	91,271	0.98875	453,789	3,754,471	41.14
45-49	90,244	0.98252	447,278	3,300,682	36.57
50-54	88,667	0.97591	437,994	2,853,404	32.18
55-59	86,531	0.96338	424,730	2,415,411	27.91
60-64	83,361	0.94637	405,630	1,990,681	23.88
65-69	78,891	0.92874	380,399	1,585,051	20.09
70-74	73,269	0.89769	347,605	1,204,652	16.44
75-79	65,773	0.84419	303,244	857,047	13.03
80-84	55,525	0.75980	244,281	553,803	9.97
85-89	42,188	0.62003	170,864	309,521	7.34
90-94	26,158	0.43392	93,770	138,657	5.30
95-99	11,350	0.26923	36,016	44,886	3.95
100+	3,056		8,373	8,871	2.90

Note: Figures below the broken line denote projected numbers

Cohort Born 1926-31

Males

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.95997	96,797	7,045,328	70.45
1-4	95,997	0.98813	381,707	6,948,530	72.38
5-9	94,857	0.99332	472,700	6,566,824	69.23
10-14	94,223	0.99490	469,914	6,094,124	64.68
15-19	93,743	0.99281	467,027	5,624,210	60.00
20-24	93,068	0.99175	463,423	5,157,183	55.41
25-29	92,301	0.99281	459,844	4,693,760	50.85
30-34	91,637	0.99315	456,614	4,233,916	46.20
35-39	91,009	0.98959	452,675	3,777,302	41.50
40-44	90,061	0.98465	446,850	3,324,628	36.92
45-49	88,679	0.97413	437,660	2,877,777	32.45
50-54	86,385	0.96373	424,091	2,440,118	28.25
55-59	83,252	0.94184	404,153	2,016,026	24.22
60-64	78,410	0.92195	376,748	1,611,873	20.56
65-69	72,290	0.89197	341,923	1,235,125	17.09
70-74	64,480	0.84674	297,693	893,201	13.85
75-79	54,598	0.77878	242,793	595,508	10.91
80-84	42,520	0.66686	177,186	352,715	8.30
85-89	28,355	0.51919	107,691	175,529	6.19
90-94	14,722	0.35084	49,716	67,839	4.61
95-99	5,165	0.20175	15,517	18,122	3.51
100+	1,042		2,605	2,605	2.50

Females

age group	l_x	p_x	$_nL_x$	T_x	e_x
<1	100,000	0.96813	97,450	7,693,534	76.94
1-4	96,813	0.98942	385,203	7,596,084	78.46
5-9	95,789	0.99452	477,632	7,210,881	75.28
10-14	95,264	0.99687	475,576	6,733,249	70.68
15-19	94,966	0.99585	473,845	6,257,674	65.89
20-24	94,572	0.99632	471,988	5,783,829	61.16
25-29	94,223	0.99672	470,345	5,311,841	56.38
30-34	93,915	0.99561	468,542	4,841,496	51.55
35-39	93,502	0.99392	466,089	4,372,955	46.77
40-44	92,934	0.98944	462,213	3,906,866	42.04
45-49	91,952	0.98344	455,952	3,444,653	37.46
50-54	90,429	0.97782	447,132	2,988,700	33.05
55-59	88,424	0.96591	434,583	2,541,568	28.74
60-64	85,409	0.95530	417,501	2,106,985	24.67
65-69	81,591	0.93775	395,258	1,689,484	20.71
70-74	76,512	0.90994	365,333	1,294,226	16.92
75-79	69,621	0.85439	322,762	928,893	13.34
80-84	59,484	0.76972	263,173	606,131	10.19
85-89	45,786	0.63444	187,085	342,958	7.49
90-94	29,048	0.44831	105,177	155,873	5.37
95-99	13,022	0.27860	41,626	50,696	3.89
100+	3,628		9,070	9,070	2.50

Note: Figures below the broken line denote projected numbers

Cohort Born 1931-36

Males

age group	l_x	p_x
<1	100,000	0.96341
1-4	96,341	0.98822
5-9	95,206	0.99503
10-14	94,733	0.99546
15-19	94,303	0.99330
20-24	93,671	0.99141
25-29	92,867	0.99422
30-34	92,330	0.99202
35-39	91,593	0.99027
40-44	90,702	0.98537
45-49	89,375	0.97816
50-54	87,423	0.96385
55-59	84,263	0.95279
60-64	80,284	0.93571
65-69	75,123	0.90364
70-74	67,884	0.85963
75-79	58,356	0.79359
80-84	46,311	0.68406
85-89	31,679	0.53433
90-94	16,927	0.36154
95-99	6,120	

Females

age group	l_x	p_x
<1	100,000	0.97144
1-4	97,144	0.99070
5-9	96,241	0.99557
10-14	95,814	0.99644
15-19	95,473	0.99703
20-24	95,189	0.99738
25-29	94,940	0.99716
30-34	94,670	0.99606
35-39	94,297	0.99373
40-44	93,706	0.98921
45-49	92,695	0.98390
50-54	91,203	0.97775
55-59	89,174	0.97064
60-64	86,556	0.96191
65-69	83,259	0.94768
70-74	78,903	0.91947
75-79	72,549	0.86931
80-84	63,068	0.78266
85-89	49,360	0.64692
90-94	31,932	0.46534
95-99	14,859	

Note: Figures below the broken line denote projected numbers

Cohort Born 1936-41

Males

age group	l_x	p_x
<1	100,000	0.96497
1-4	96,497	0.99026
5-9	95,557	0.99503
10-14	95,082	0.99661
15-19	94,759	0.99414
20-24	94,204	0.99340
25-29	93,582	0.99323
30-34	92,949	0.99330
35-39	92,326	0.99105
40-44	91,500	0.98722
45-49	90,331	0.98092
50-54	88,607	0.97202
55-59	86,128	0.96182
60-64	82,840	0.94628
65-69	78,389	0.91748
70-74	71,920	0.87262
75-79	62,759	0.80948
80-84	50,802	0.69944
85-89	35,533	0.54777
90-94	19,464	

Females

age group	l_x	p_x
<1	100,000	0.97296
1-4	97,296	0.99155
5-9	96,474	0.99651
10-14	96,137	0.99847
15-19	95,990	0.99775
20-24	95,775	0.99770
25-29	95,555	0.99697
30-34	95,265	0.99583
35-39	94,867	0.99410
40-44	94,308	0.99107
45-49	93,466	0.98683
50-54	92,235	0.98103
55-59	90,485	0.97634
60-64	88,344	0.96768
65-69	85,489	0.95429
70-74	81,581	0.92979
75-79	75,854	0.87910
80-84	66,683	0.79341
85-89	52,907	0.65505
90-94	34,657	

Note: Figures below the broken line denote projected numbers

Cohort Born 1941-46

Males

age group	l_x	p_x
<1	100,000	0.96749
1-4	96,749	0.99143
5-9	95,920	0.99671
10-14	95,604	0.99741
15-19	95,357	0.99486
20-24	94,867	0.99243
25-29	94,149	0.99391
30-34	93,576	0.99345
35-39	92,962	0.99179
40-44	92,199	0.98875
45-49	91,162	0.98362
50-54	89,669	0.97762
55-59	87,662	0.96927
60-64	84,968	0.95798
65-69	81,398	0.92911
70-74	75,628	0.88026
75-79	66,572	0.82104
80-84	54,658	0.70674
85-89	38,629	

Females

age group	l_x	p_x
<1	100,000	0.97522
1-4	97,522	0.99294
5-9	96,834	0.99784
10-14	96,624	0.99815
15-19	96,445	0.99790
20-24	96,243	0.99742
25-29	95,995	0.99717
30-34	95,723	0.99598
35-39	95,338	0.99496
40-44	94,858	0.99241
45-49	94,138	0.98809
50-54	93,017	0.98513
55-59	91,633	0.98176
60-64	89,962	0.97564
65-69	87,770	0.96374
70-74	84,587	0.93767
75-79	79,315	0.88824
80-84	70,451	0.79849
85-89	56,254	

Note: Figures below the broken line denote projected numbers

Cohort Born 1946-51

Males

age group	l_x	p_x
<1	100,000	0.97176
1-4	97,176	0.99465
5-9	96,656	0.99743
10-14	96,408	0.99763
15-19	96,180	0.99394
20-24	95,597	0.99248
25-29	94,878	0.99358
30-34	94,269	0.99349
35-39	93,655	0.99217
40-44	92,922	0.99004
45-49	91,997	0.98696
50-54	90,797	0.98101
55-59	89,073	0.97515
60-64	86,859	0.96542
65-69	83,855	0.93813
70-74	78,667	0.88917
75-79	69,949	0.82886
80-84	57,978	

Females

age group	l_x	p_x
<1	100,000	0.97793
1-4	97,793	0.99539
5-9	97,342	0.99837
10-14	97,184	0.99847
15-19	97,035	0.99784
20-24	96,825	0.99730
25-29	96,564	0.99693
30-34	96,268	0.99636
35-39	95,918	0.99489
40-44	95,428	0.99263
45-49	94,725	0.99109
50-54	93,880	0.98767
55-59	92,723	0.98591
60-64	91,417	0.97819
65-69	89,423	0.96702
70-74	86,474	0.94244
75-79	81,496	0.89331
80-84	72,801	

Note: Figures below the broken line denote projected numbers

Cohort Born 1951-56

Males

age group	l_x	p_x
<1	100,000	0.97617
1-4	97,617	0.99504
5-9	97,133	0.99773
10-14	96,912	0.99747
15-19	96,667	0.99293
20-24	95,983	0.99092
25-29	95,112	0.99311
30-34	94,456	0.99324
35-39	93,818	0.99182
40-44	93,050	0.99181
45-49	92,288	0.98986
50-54	91,352	0.98338
55-59	89,834	0.98015
60-64	88,051	0.97120
65-69	85,515	0.94596
70-74	80,894	0.89675
75-79	72,542	

Females

age group	l_x	p_x
<1	100,000	0.98139
1-4	98,139	0.99564
5-9	97,711	0.99825
10-14	97,540	0.99862
15-19	97,405	0.99737
20-24	97,149	0.99711
25-29	96,869	0.99711
30-34	96,589	0.99643
35-39	96,244	0.99556
40-44	95,817	0.99471
45-49	95,310	0.99249
50-54	94,594	0.99000
55-59	93,648	0.98797
60-64	92,522	0.98004
65-69	90,675	0.96914
70-74	87,877	0.94626
75-79	83,154	

Note: Figures below the broken line denote projected numbers

Cohort Born 1956-61

Males

age group	l_x	p_x
<1	100,000	0.98323
1-4	98,323	0.99654
5-9	97,983	0.99865
10-14	97,851	0.99851
15-19	97,705	0.99717
20-24	97,429	0.99668
25-29	97,106	0.99685
30-34	96,800	0.99678
35-39	96,488	0.99645
40-44	96,145	0.99597
45-49	95,757	0.99333
50-54	95,118	0.99227
55-59	94,383	0.99012
60-64	93,450	0.98173
65-69	91,743	0.97122
70-74	89,103	

Females

age group	l_x	p_x
<1	100,000	0.98323
1-4	98,323	0.99654
5-9	97,983	0.99865
10-14	97,851	0.99851
15-19	97,705	0.99717
20-24	97,429	0.99668
25-29	97,106	0.99685
30-34	96,800	0.99678
35-39	96,488	0.99645
40-44	96,145	0.99597
45-49	95,757	0.99333
50-54	95,118	0.99227
55-59	94,383	0.99012
60-64	93,450	0.98173
65-69	91,743	0.97122
70-74	89,103	

Note: Figures below the broken line denote projected numbers

APPENDIX EIGHT:

Indices of Compression of Mortality Calculated Based on Non-Maori Synthetic Life Tables, at Birth and at Age 65 Years, by Gender, 1971-1996

APPENDIX EIGHT:
INDICES OF COMPRESSION OF MORTALITY CALCULATED BASED ON NON-MAORI SYNTHETIC LIFE TABLES, AT BIRTH AND AT AGE 65 YEARS, BY GENDER, 1971-1996

Non-Maori Males

	1970-72	1975-77	1980-82	1985-87	1990-92	1995-97
			At birth			
life expectancy (e_0)	69.1	69.4	70.8	71.4	73.4	75.3
person-year differential (PD)	26.74	26.43	26.20	26.39	25.90	24.08
person-year ratio (PR)	0.2791	0.2759	0.2703	0.2700	0.2609	0.2423
standard deviation (SD)	17.50	17.71	16.97	16.95	16.56	15.73
coefficient of variation (CV)	0.2534	0.2553	0.2398	0.2375	0.2257	0.2088
numerator of entropy (NH)	12.37	12.45	12.07	12.06	11.81	11.13
entropy (H)	0.1791	0.1795	0.1706	0.1689	0.1610	0.1478
			At age 65 years			
life expectancy (e_{65})	12.6	12.9	13.4	13.7	14.9	15.8
person-year differential (PD)	18.95	18.63	19.27	19.68	19.87	18.95
person-year ratio (PR)	0.6008	0.5914	0.5902	0.5890	0.5717	0.5455
standard deviation (SD)	7.38	7.42	7.63	7.73	8.01	8.05
coefficient of variation (CV)	0.5855	0.5759	0.5699	0.5628	0.5375	0.5100
numerator of entropy (NH)	6.85	6.85	7.05	7.15	7.34	7.27
entropy (H)	0.5435	0.5318	0.5267	0.5202	0.4926	0.4606

Non-Maori Females

	1970-72	1975-77	1980-82	1985-87	1990-92	1995-97
			At birth			
life expectancy (e_0)	75.2	75.9	76.9	77.4	79.2	80.6
person-year differential (PD)	23.32	22.93	23.77	23.52	23.08	21.24
person-year ratio (PR)	0.2368	0.2321	0.2363	0.2331	0.2256	0.2086
standard deviation (SD)	16.54	16.62	16.23	15.95	15.14	14.50
coefficient of variation (CV)	0.2201	0.2190	0.2112	0.2060	0.1911	0.1799
numerator of entropy (NH)	11.19	11.28	11.25	11.05	10.67	10.05
entropy (H)	0.1488	0.1487	0.1464	0.1428	0.1346	0.1247
			At age 65 years			
life expectancy (e_{65})	16.0	16.7	17.2	17.5	18.6	19.3
person-year differential (PD)	17.77	17.41	18.71	18.74	18.98	17.66
person-year ratio (PR)	0.5259	0.5107	0.5208	0.5171	0.5050	0.4776
standard deviation (SD)	7.79	8.01	8.33	8.30	8.48	8.39
coefficient of variation (CV)	0.4858	0.4803	0.4837	0.4741	0.4556	0.4339
numerator of entropy (NH)	6.94	7.08	7.39	7.36	7.47	7.26
entropy (H)	0.4332	0.4247	0.4292	0.4204	0.4011	0.3756

APPENDIX NINE:

Indices of Compression of Mortality Calculated Based on Non-Maori Cohort Life Tables for Cohorts Born between 1851 and 1901, at Birth and at Age 65 Years, by Gender

APPENDIX NINE:
INDICES OF COMPRESSION OF MORTALITY CALCULATED BASED ON NON-MAORI COHORT LIFE TABLES FOR COHORTS BORN BETWEEN 1851 AND 1901, AT BIRTH AND AT AGE 65 YEARS, BY GENDER

Non-Maori Males

	1851-61	1861-71	1871-81	1881-91	1891-01
	At birth				
life expectancy (e_0)	-	-	55.5	58.2	60.2
person-year differential (PD)	-	-	40.41	37.72	36.74
person-year ratio (PR)	-	-	0.4214	0.3933	0.3750
standard deviation (SD)	-	-	29.82	28.34	27.37
coefficient of variation (CV)	-	-	0.5373	0.4868	0.4549
numerator of entropy (NH)	-	-	22.05	20.40	19.47
entropy (H)	-	-	0.3973	0.3505	0.3237
	At age 65 years				
life expectancy (e_{65})	12.6	12.8	12.9	12.9	12.9
person-year differential (PD)	18.69	19.49	20.02	20.09	21.19
person-year ratio (PR)	0.5970	0.6029	0.6075	0.6098	0.6217
standard deviation (SD)	7.14	7.28	7.47	7.41	7.66
coefficient of variation (CV)	0.5651	0.5669	0.5769	0.5755	0.5937
numerator of entropy (NH)	6.78	6.99	7.14	7.10	7.40
entropy (H)	0.5370	0.5441	0.5516	0.5518	0.5729

Non-Maori Females

	1851-61	1861-71	1871-81	1881-91	1891-01
	At birth				
life expectancy (e_0)	-	-	58.9	62.5	65.4
person-year differential (PD)	-	-	39.62	37.09	35.86
person-year ratio (PR)	-	-	0.4022	0.3726	0.3541
standard deviation (SD)	-	-	29.95	28.81	28.07
coefficient of variation (CV)	-	-	0.5086	0.4613	0.4290
numerator of entropy (NH)	-	-	21.72	20.32	19.43
entropy (H)	-	-	0.3688	0.3254	0.2969
	At age 65 years				
life expectancy (e_{65})	13.8	14.1	14.8	15.7	16.6
person-year differential (PD)	19.07	19.86	19.94	20.05	21.03
person-year ratio (PR)	0.5807	0.5848	0.5748	0.5617	0.5592
standard deviation (SD)	7.42	7.72	7.94	8.08	8.49
coefficient of variation (CV)	0.5379	0.5473	0.5378	0.5164	0.5119
numerator of entropy (NH)	7.00	7.30	7.42	7.53	7.83
entropy (H)	0.5080	0.5175	0.5024	0.4808	0.4723

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