Socioeconomic Gradients in Child Mortality:

New Zealand 1981-1999

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Abstract

Background:

International research has demonstrated the existence of socioeconomic inequalities in child mortality at snapshots in time. However few studies have looked at the time trends of these inequalities. Between 1981 and 1999 New Zealand undertook large social and economic reforms, making time trends of inequalities in child mortality over this period of particular interest.

Objectives:

1. To determine if socioeconomic gradients in child (1-14 years) mortality changed between 1981 and 1999 in New Zealand.
2. To examine what causes of mortality generate socioeconomic gradients.

Methods:

A census-mortality record linkage study design was used. Four retrospective cohorts were created using all children, aged 0-14, alive on census night (1981, 1986, 1991, 1996), subsequently followed up for mortality for 3 years. Weights adjusting for linkage bias were calculated. Socioeconomic position for each child was measured using three household level socioeconomic variables: equivalised income, highest occupational class and maternal education. Age and ethnicity standardised mortality rates for each level of these socioeconomic variables were calculated using person-time denominator. Standardised rate ratios and rate differences were calculated to determine relative and absolute differences across the levels of each socioeconomic factor. In order to facilitate

comparisons over time regression based population measures of inequality (relative and slope indices of inequality) were also calculated.

Results:

All socioeconomic groups experienced mortality declines between 1981 and 1999. Children with parents in lower socioeconomic groups had higher all-cause mortality than children in higher socioeconomic groups at all points in time (e.g. 1996 income rate ratio 1.6 (95%CI 1.2-2.1)). *Trends:* There was evidence of increasing mortality inequality by income over time. For example the relative index of inequality increased from 1.5 in 1981 to 1.8 in 1996 (p trend 0.06). There was no clear change over time by education or occupational class, or for absolute measures of inequality for all three socioeconomic variables. *Causes of death*: Socioeconomic gradients were seen in all causes of death, except cancer.

Conclusions:

Childhood mortality socioeconomic inequalities exist in New Zealand. However trends in these inequalities remain uncertain, although results by income suggest an increase in relative inequality. Public health interventions to reduce socioeconomic mortality inequalities need to eliminate differential exposure to the risk factors for child mortality, particularly injury mortality.

Statistics New Zealand Security Statement

The New Zealand Census-Mortality Study (NZCMS) was initiated by Dr Tony Blakely and his co-researchers from the Wellington School of Medicine, University of Otago. It was approved by the Government Statistician as a Data Laboratory project under the Microdata Access Protocols. This security statement is essentially the same as that provided for the original NZCMS research project.

The NZCMS fully complies with the 1975 Statistics Act.

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Under the Statistics Act 1975 the Government Statistician has legal authority to collect and hold information about people, households and businesses, as well as the responsibility of protecting individual information and limits to the use to which such information can be put. The obligations of the Statistics Act 1975 on data collected under the Act are summarised below.

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This project was carried out under section 21(3B). Under Section 21(3B) the Government Statistician requires an independent contractor under contract to Statistics New Zealand, and any employee of the contractor, to make a statutory declaration of secrecy similar to that required of Statistics New Zealand employees where they will have access to information collected under the Act. For the purposes of implementing the confidentiality provisions of the Act, such contractors are deemed to be employees of Statistics New Zealand.

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Census data

Traditionally, data from the Population Census is published by Statistics New Zealand in aggregated tables and graphs for use throughout schools, business and homes. Recently Statistics New Zealand has sought to increase the benefits that can be obtained from its data by providing access to approved researchers to carry out research projects. Microdata access is provided, at the discretion of the Government Statistician, to allow authoritative statistical research of benefit to the public of New Zealand.

The NZCMS uses anonymous census data and mortality data that are integrated (using a probabilistic linking methodology) as a single dataset for each census year. The NZCMS is the first project for which the census has been linked to an administrative dataset for purposes apart from improving the quality of Statistics New Zealand surveys. The project has been closely monitored to ensure it complies with Statistics New Zealand's strict confidentiality requirements.

Further information
For further information about confidentiality matters in regard to the NZCMS, please contact either:

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Abbreviations

CHE	Crown Health Enterprise,
CI	Confidence Interval
FPP	First Past the Post
ICD	International Classification of Disease
ISCO	International Standard Classification of Occupation
NGO	Non Governmental Organisation
NZCMS	New Zealand Census Mortality Study
NZHIS	New Zealand Health Information Service
NZSCO	New Zealand Standard Classification of Occupation
OECD	Organisation for Economic Co-operation and Development
OR	Odds Ratio
RII	Relative Index of Inequality
RR	Relative Risk or Risk Ratio
SIDS	Sudden Infant death Syndrome
SII	Slope index of Inequality
SNZ	Statistics New Zealand
SRD	Standard Rate Difference
SRR	Standard Rate Ratio
WHO	World Health Organisation

Glossary

Gini coefficient – population measure of income inequality, based on equivalised household income. Value ranges between 0 (perfect equality) and 1 (perfect inequality).

Iwi - Maori tribal group.

Māori - The indigenous people of New Zealand.

Non-Māori/non-Pacific people – Residual (and largest) ethnic group in New Zealand, mainly consists of descendants of European migrants to New Zealand.

NZDep – New Zealand deprivation index. Small area deprivation measure, comprising nine characteristics related to deprivation. There are 1991, 1996 and 2001 versions of the index.

Pacific people – Migrants, and descendants of migrants, from the Pacific Islands.

Pakeha – Māori word used to describe those who are non-Māori.

Pareto estimate – Method used to estimate (unknown) distribution of income within an income bracket. In this thesis the method was used to calculate the midpoint of the unknown income bracket.

PHARMAC – (Pharmaceutical Management Agency). A Crown Entity agency established to manage government expenditure on pharmaceuticals in New Zealand.

Te Reo Māori - Māori language.

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Chapter 1: Introduction

Variations, between groups in a society, in adult mortality are an area of research that has received increasing international attention in the last two decades. These differences are present across ethnic group, gender, and socioeconomic position, with the more deprived groups almost invariably having poorer outcomes in all causes of mortality.

Research into equivalent gradients in children is less well developed. Much of the research regarding socioeconomic position in childhood is focused on this 'exposure' as a life course stage leading to the end point of socioeconomic gradients in adult health and mortality. However the research that has been done points to the existence of gradients in mortality during childhood.

Interval sampling of inequalities in adult health has indicated that these inequalities are dynamic, and ongoing monitoring of gradients is also an area of research that has been developed. While there is an increasing body of work on time trends in adult mortality inequalities, there is very little on children.

The dynamic nature of inequalities raises the questions of how they are generated and perpetuated, and what causes them change over time. The answers to these questions are complex and may be different for children, because the influence of socioeconomic position may be different depending on life stage and mortality causes.

There are a number of reasons why it is of interest to look at trends in socioeconomic gradients in child mortality in New Zealand. Despite substantial reductions in mortality in the last 50 years, New Zealand children have high

overall mortality rates in comparison to other OECD countries. There is also evidence that these comparatively high mortality rates have not always been experienced in New Zealand, as our ranking in international 'league tables' for child death has declined in the last few decades.

While there is already some evidence that socioeconomic gradients in child mortality exist in New Zealand, these are snapshots in time. This thesis will look at whether the overall decline in mortality over the last decades has been experienced by all groups of children or whether some have been preferentially favoured over others.

There is also considerable interest in the effects of the structural changes in the economic and social environment on the health of children. The late 1980s and early 1990s saw considerable economic and social policy changes, driven by a neo-liberal agenda. Evaluations of adult ethnic and socioeconomic inequalities over this time period are suggestive of differential effects of these reforms on groups in New Zealand (Ajwani et al., 2003; University of Otago & Ministry of Health, 2004). However this has not been looked at with reference to child mortality.

There has been a perception that child health in New Zealand has deteriorated over this time period, particularly with a resurgence of infectious diseases related to poverty and an increase in avoidable hospital admissions in children (Blaiklock et al., 2002; Dharmalingam et al., 2004). It is important to see whether poor child health has translated into mortality changes, and particularly whether any mortality changes have been differential by socioeconomic position. While child mortality is a relatively uncommon event, it remains extremely important to monitor, and any policy influences on mortality need to be assessed.

The discussion of inequalities in health and mortality is not a value free dialogue. This is particularly the case with child mortality, as children cannot be viewed as responsible for their own socioeconomic position. However from birth (and it could be argued before birth) parental socioeconomic position helps determine their risk of disease, death and future life opportunities. Hence this thesis takes the position that differences in child mortality based on parental socioeconomic position are inequities i.e. they are unfair.

Finally it is important to make the point that monitoring of inequalities is not an event that occurs in a vacuum. While monitoring of inequalities (such as this thesis) is a valuable activity, providing evidence that may justify policy changes, it is only a prerequisite to determine where to intervene to eliminate these inequities.

The New Zealand Census Mortality Study, anonymously and probabilistically linking census and death records, has allowed us to review socioeconomic trends in child mortality from 1981-1999 in detail. For the first time it is possible to examine socioeconomic differences in mortality at four points in time over 20 years of major change in New Zealand.

The main question that this thesis attempts to answer is:

- Have socioeconomic gradients in child mortality changed over time in New Zealand?

Subsidiary questions to be answered include:

- Has there been differential improvement in health status of socioeconomic groups?
- What causes of mortality generate socioeconomic gradients? Specifically are they driven by injury mortality or is there evidence of other causes of death contributing to gradients?
- Do gradients vary depending on the measure of socioeconomic position under study?
- Do gradients vary by demographic variables such as age, sex and ethnic group?

This thesis is structured around five chapters (this being the first). The second chapter introduces key concepts around social epidemiology, reviews current literature on the existence of socioeconomic gradients in child mortality and finally contextualises the New Zealand experience of policy change and child mortality. The third chapter looks at the methods used in this thesis and chapter three presents the results. Chapter five concludes the thesis with a discussion on the problems of the study and conclusions and implications of the findings from this study.

This thesis covers an enormous topic area, and will cover many of the areas quite broadly due space and time limitations.

Chapter 2: Literature Review

This chapter aims to introduce the concepts and background relevant to social inequalities, specifically those relating to child mortality. The first section looks at the historical development and current conceptualizations of social theory, and then delves into the interpretation of socioeconomic gradients with a section on social epidemiology. Lastly it deals with how to measure socioeconomic position, including some of the pitfalls and problems.

The second section looks at existing literature on the existence of socioeconomic inequalities in child mortality, including variation by age, sex and cause of death. It reviews the literature on time trends and some of the difficulties with time trends and international comparisons are highlighted.

The third section deals with the New Zealand situation specifically, looking at the policy change that affected children in the period of study and currently available data on inequalities in child mortality in New Zealand,

1 Socio-Economic Position

1.1 Social Theory

The social theorist views uneven distributions of health and illness as a reflection or embodiment of the inherent structure of a society (Susser, 1997). Social theory attempts to explain how these structures are generated, maintained and revised in a dynamic world. These theories initially arose in the context of a capitalist industrialist society. The main theorists of social class in the 19th century were Karl Marx and Max Weber, whose theories continue to dominate thinking in this area (Susser, 1997). Theory is essential to understand as it influences measurement and interpretation of social indicators.

Karl Marx defined classes (groups of people) in relation to their access to the productive resources in society (Lynch & Kaplan, 2000). Marx argued that conflict between different productive groups is inherent to the structure of capitalist societies, as grouped occupations are unequal in power and status (Susser, 1997). Classes emerge when a minority of the population is able to gain control of excess production for their own use and advantage (Lynch & Kaplan, 2000). This view sees domination and exploitation as inherent to class relations and these processes (inevitably) result in a society with a few having access to the resources that the majority produce. This theory is based solely on an economic model of production, although contemporary interpretations of Marx expand this to identify three forms of exploitation, based on ownership of assets, skills and control of organisational process (Lynch & Kaplan, 2000)

Max Weber is the other enduring theorist of social class. While not rebutting Marx's economic analysis of class he expanded the idea of class to encompass three different dimensions; economic/income dimension; status or prestige; and

power (Liberatos et al., 1988). The economic base is similar to Marx, implying access and control of resources. Status, however, recognizes that prestige in society may not be solely acquired through economic means. Certain groups may derive prestige from other avenues (Susser, 1997). Power is related to political context (Liberatos et al., 1988). All these dimensions are interrelated and reinforce each other. Social stratification may take place down one dimension or it may derive from a combination of more than one. Weber's view emphasized that individual's function voluntarily within the capitalist system and classes within this are groups that share similar life chances (Lynch & Kaplan, 2000).

The functionalist approach to class developed in the USA and is primarily a naturalist (or social selection) explanation of class (Lynch & Kaplan, 2000; Macintyre, 1997). The underlying theory of functionalism is that capitalist societies are complex and require stratification into sectors that are more or less valuable to the society (Lynch & Kaplan, 2000). Similar to Marx functionalists see stratification as being embedded in the way capitalist societies operate. However unlike Marx, functionalists view this stratification as acceptable. Implicit in this theory is the belief that those with ability will inexorably rise to positions of authority and therefore that interfering in this process is probably futile.

Socioeconomic position (SEP) is a term that describes an individual's place in the social hierarchy. In this thesis the use of socioeconomic position rather than socioeconomic status is deliberate. Socioeconomic status implies a predominant status domain, compared to position, which encompasses both status and resources (Krieger, 2001a). For the purpose of this thesis the definition of socioeconomic position is:

> "The social and economic factors that influence what position(s) individuals and groups hold within the structure of society" (Lynch & Kaplan, 2000).

As can be seen in this brief description of the theories of social class, the explanations are diverse and each engenders different data collection and intervention responses.

1.2 Social Epidemiology

While sociologists have been concerned with SEP as an endpoint of a sociological process, epidemiology is interested from a health perspective (Mannetje & Kromhout, 2003). Epidemiology is defined as the study of the distribution and determinants of health related states or events in specified populations and the application of this study to control of health problems (Last, 2001). While Last defines epidemiology quite broadly there has been increasing debate about the scope of epidemiology, particularly with references to the role of 'risk factor' epidemiology (Pearce, 1996; Susser, 1998). Social epidemiology differentiates itself from other types of epidemiology by concentrating on the social determinants of disease, rather than treating them only as a background to biomedical phenomena (Krieger, 2001a).

The relationship between adult health and mortality and socioeconomic status was noticed in ancient Greece, Rome and China (Krieger et al., 1997). The first formal measurement was in Britain in the time of the plague, when mortality of the upper classes who fled London was compared to the mortality of those who remained (Susser, 1997). However formal documentation of socioeconomic gradients in health or mortality requires a classification system of socioeconomic position (as well as a way of measuring the outcome).

Differences in life expectancy and mortality by occupation had been noted in England since 1840, however a formal classification system was not developed until much later. A social class classification system for measuring differences in mortality was initially proposed in the late 19th century in Britain, however the Registrar General's Social Classification of Occupations, which was a combination of occupational and industrial grading of employment, did not originate until 1913 (Macintyre, 1997).

Despite this historical interest, over the course of the 20th century in the UK interest in inequalities in mortality waned. The Black Report published in 1980, which reviewed data on inequalities in mortality from the 1930s and the 1970s,rekindled interest in the description and measurement of socioeconomic inequalities (Macintyre, 1997). The Black Report noted that inequalities in mortality by occupational class persisted in the UK despite changes in technology and health services between 1930 and 1970, and these inequalities were observed over the entire lifespan (Townsend et al., 1992).

The gradients in mortality by SEP have been preserved despite evolving causes of mortality. For example infectious diseases were a predominant cause of mortality 150 years ago, and socioeconomic gradients in mortality were noted during that period. Contemporary causes of mortality, such as ischaemic heart disease and cancer, have utterly different pathogenic mechanisms yet share the same pattern of gradients (Keating & Hertzman, 1999).

The first documented measurement of inequalities in child mortality was in 1913, when the Registrar General's Social Classification of Occupations was applied to infant mortality. A gradient of increasing mortality as paternal social class declined was demonstrated (Macintyre, 1997). Almost 70 years later the Black Report concluded that these differences persisted (Townsend et al., 1992).

Some historical evidence of social differentials in child mortality is available. For example Potts described the higher incidence of scrotal cancer in chimney sweeps (usually children of very poor families) in 18th century Britain (Waldron, 1983). This was the first malignant disease to be connected with an occupation, however social epidemiologists could view this cancer as an example of differential health outcomes for poorer children. The records of the Monte Delle Doti dowry fund in 13th and 14th century Florence have allowed analysis of mortality rates of girls, by the amount deposited into the fund by her father for the purpose of her future marriage. Those girls whose fathers could only afford to deposit small dowry funds had the highest mortality rates (Morrison et al., 1977). However these are contemporary analyses and interpretation of the historical data.

The question of why these differences in health and mortality exist is complex. The existence of gradients suggests that the factors that govern health are tied to the social structure and that differential placing in the social hierarchy determines your exposure and access to health promoting resources and exposure to health damaging experiences (Lynch & Kaplan, 2000). The Black Report summarised the various explanations for socio-economic gradients in mortality (see Table 1). However many of these ideas have been debated for at least the last century and continue to cause controversy (Macintyre, 1997; Townsend et al., 1992).

Table 1 Explanations for social gradient in mortality

Explanation	'Hard' Version	'Soft' Version
Artefact	No relationship between class and health-purely an artefact of measurement	Magnitude of observed gradient will depend on measurement of both class and health
Natural/Social Selection	Health determines class position therefore gradients are neutral and explained away	Health can contribute to class position and therefore help to explain observed gradients
Materialist/Structural	Material/Physical conditions of life associated with class structure are the complete explanation for class gradients in health	Physical and psychosocial features associated with the class structure influence health and contribute to observed behaviours
Cultural/Behavioural	Health damaging behaviours freely chosen by individuals in different social classes explain away social class gradients	Health damaging behaviours are differentially distributed across social classes and contribute to observed gradients

Source: Macintyre 1997

The hard and soft versions of explanation of socioeconomic gradients in mortality presented in the Black Report represent polarised ends of the spectrums of explanations. However they are an illustration of the observation that Nancy Kreiger made; that shared observations of inequality do not translate to common understanding of causes (Krieger, 2001b). The hard version tries to 'explain away' the gradients and the soft version accepts that gradients exist and looks to processes of social stratification to explain them. In reality both explanations have some validity, for example measurement error is a problem in social epidemiology and health behaviours are differentially distributed. Since the publication of the Black Report much research has been done both describing, as well as trying to elucidate the explanation of socioeconomic gradients in health. However many of the explanations given in the above table continue to be debated, although some of this debate may reflect ideology rather than fact.

Nancy Kreiger argued that currently within social epidemiology there are three streams of causal explanation of disease distribution (Krieger, 2001b). However it should be noted these are not mutually exclusive explanations in competition with each other. All theories have strengths and weaknesses, and the 'true'

causal explanations are likely to encompass many of the concepts within each theory.

The first explanation is psychosocial theory. The central hypothesis of this theory is that the psychosocial environment alters host susceptibility (Krieger, 2001b). Host susceptibility is altered by the physiologic response to stress, which has the potential to both help and harm an individual on the cellular level. This theory suggests that chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis can lead to 'allostatic load', which consists of a number of physiologically abnormal responses to stress. This in turn can lead to pathological processes in the cellular structure of the body (McEwen, 1998).

One of the reasons for socioeconomic gradients in health, this theory suggests, is that people in lower SEP are exposed to a greater number of stressful situations with less resources (both physical and psychological) and less control to cope with them. This theory underlies the 'premature ageing' explanation of black/white differences in the USA (Jones, 1999), and explanations of the differences in mortality in the Whitehall study when, despite the lack of a 'toxic' working environment, occupational class gradients in mortality continued to be seen (McEwen, 1998).

Stress mechanisms affecting health also include increased adverse health behaviours (e.g. smoking) seen in lower socioeconomic groups (Kawachi et al., 2002). It is argued that social capital and social cohesion explanations can be considered psychosocial explanations, as community level cohesion provides a social environment that modifies host responses (Krieger, 2001b). The psychosocial environment may also affect social participation (which is a requirement for a fulfilling, satisfying and healthy life, above simply meeting adequate material conditions of existence) (Marmot, 2002).

There are criticisms of the psychosocial theory. It cannot explain why the existence of gradients in health and/or mortality are (almost) universal, but the causes of death in which they are observed are heterogeneous between countries. It also fails to address the structural questions of why certain groups are exposed to these stressful experiences.

The second area is known as the political economy of health. This area of research concentrates on the distribution of the macro level determinants of health, as opposed to the direct biological processes described above. It is concerned with identifying the beneficiaries and those who are disadvantaged by a policy or practice (Krieger, 2001b).

Proponents of this theory focus on the ability of the hegemonic political ideology to capture orthodoxy, thus presenting itself as the 'only possible' economic and social option (Bambra et al., 2003; Navarro, 1998). By dominating political process this ideology alters health by altering the structural determinants of health. For example the current market oriented neo-liberal policies have increased income inequality, decreased wage and labour market stability and decreased social cohesion (Coburn, 2000; Navarro, 1998). These changes are thought to have acted to increase socioeconomic inequalities in health, although the country specific political environment probably ameliorates the effects of these policies (Navarro & Shi, 2001).

As well as literature on the effects of specific political movements there is also a growing body of work that has looked at the effects of massive economic upheaval on health and mortality. These studies suggest that, at least in the short term, we can expect deterioration in health status and, possibly, increasing mortality (Hertzman & Siddiqi, 2000; Men et al., 2003; Murray & Chen, 1993; Tangcharoensathien et al., 2000).

However again this theory is incomplete, it is hazy on how these broad societal changes are enacted at the level of the cell, where inequalities in health and mortality must eventually be expressed. The other issue is that even though there are different levels of inequality between countries, there does not seem to be a clear pattern related to the political ideology. For example Scandinavian countries have inequalities in health and mortality and some are as strong in relative terms as those in more 'unequal societies' such as the UK (Whitehead et al., 2000).

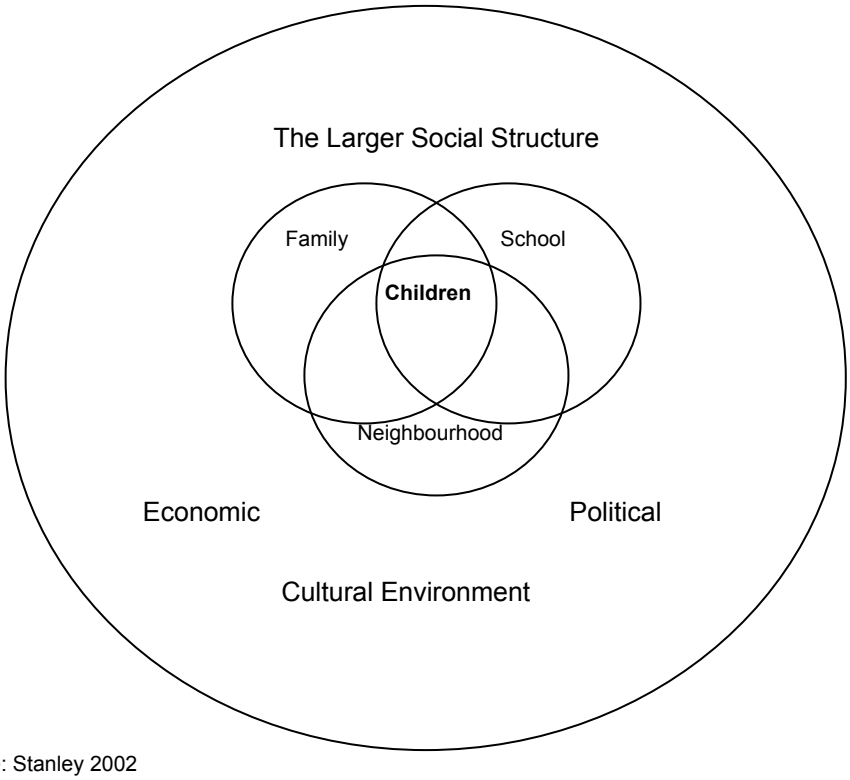
The third theory, proposed by Krieger is eco-social theory (although many of the ideas can be seen in prior publications, see for example (Brunner & Marmot, 1999)). This theory tries to overcome the limitations of the psychosocial theory and political economy of health by merging them (Krieger, 2001b). Eco-social theory attempts to explain who and what drives current and changing patterns of inequalities, by looking at all levels of 'causation'. Simply put broad political processes are reflected in institutions in civil society, which are lived by individuals and embodied in their biological processes (Krieger, 2001b). This theory calls for multiple levels of analysis and multiple methods of investigation, including new epidemiological tools as current methodology make it difficult to prove causation in social epidemiology (Kaufman & Cooper, 1999). This theory includes explanations relating to the role of health services and differential behaviours in generating and perpetuating health gradients (Krieger, 2001b). However it does not address health selection explanations (see section 1.3.1.3 for a discussion on health section).

Various diagrammatic models have been proposed, which operationalise aspects of aspects of these theories of explanation. For example see Dahlgren and Whitehead (Dahlgren & Whitehead, 1991), Brunner and Marmot (Brunner & Marmot, 1999), and Turrell (Turrell et al., 1999). These models differ in detail and to the extent that they look at the influence of the life course, cultural influences and psychosocial explanations. Local models incorporate needs

based on specific local conditions, such as colonial histories and multiethnic populations. Examples of these can be found in Stanley (Stanley, 2002) and Howden-Chapman and Tobias (Howden-Chapman & Tobias, 2000). Models specifically designed for planning where to intervene to reduce inequalities also exist (Ministry of Health, 2002b).

However the above are models that have been predominately designed for adults. The explanations for socioeconomic gradients in child mortality may (or may not) be slightly different from that of adults due to the differing influence of socioeconomic position on children, different mortality causes, the effect of school. Figure 1 illustrates the broad environmental determinants of child health (Stanley, 2002).

Figure 1 Diagrammatic model of the determinants of child health

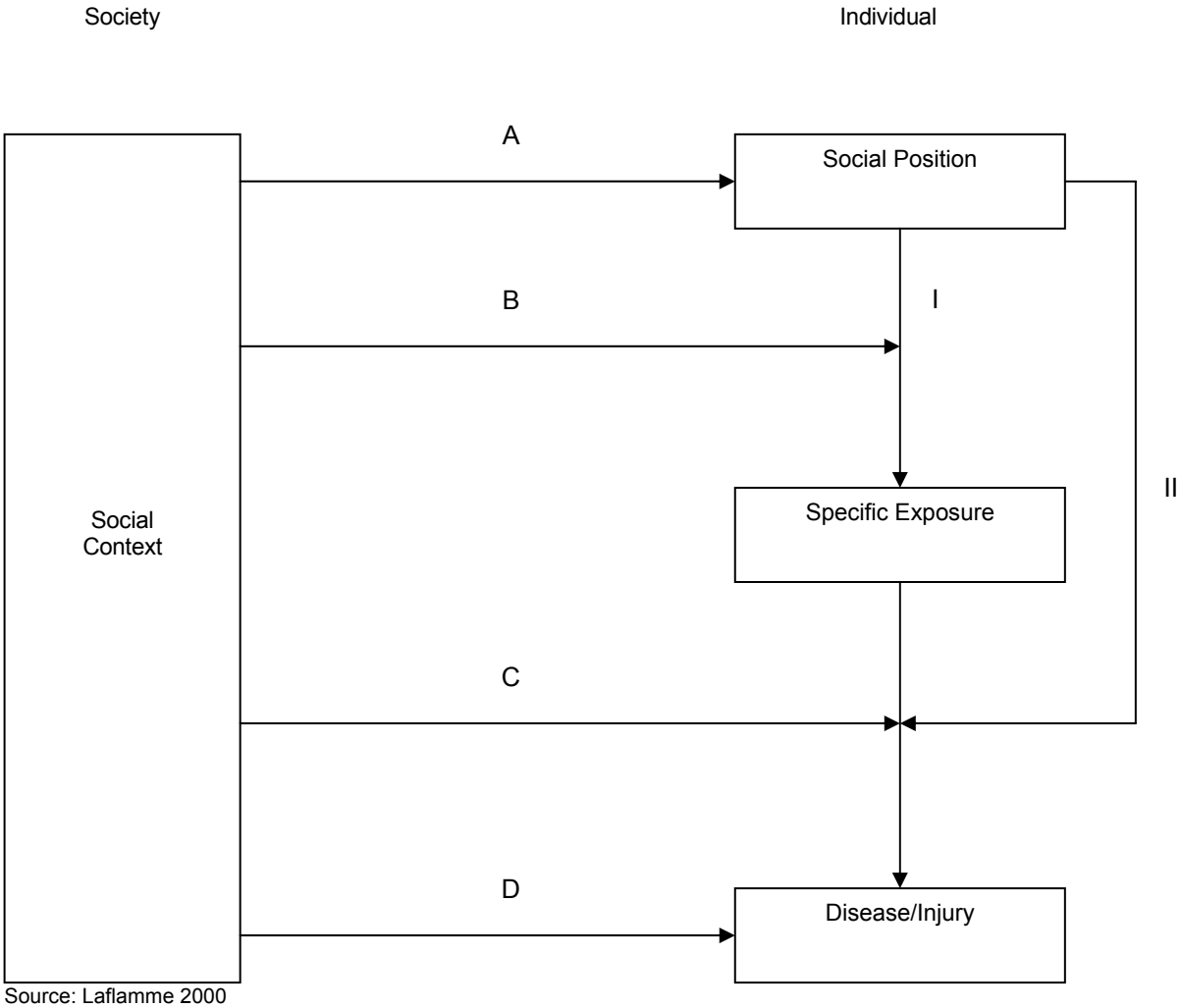


Source: Stanley 2002

Figure 2 shows the model proposed by Diderichson and Hallqvist for inequalities in childhood injury (Laflamme & Diderichsen, 2000). This model

encompasses the macro, micro and meso levels that influence childhood injury thus allowing a way of analysing where the socioeconomic gradients may arise in mortality and therefore where interventions may be appropriate i.e. an explanatory and intervention framework. In this model social context is regarded as encompassing interwoven elements in the environments that characterize a neighbourhood or society. Social context impacts on social stratification (A), on differential exposure to harmful factors (B), differential susceptibility (C) and directly on health (D). At the individual level it has been argued that two mechanisms are important, differential exposure (I) and differential susceptibility (II) (Laflamme & Diderichsen, 2000).

Figure 2 Diderichson and Hallqvist model of socioeconomic determinants of injury

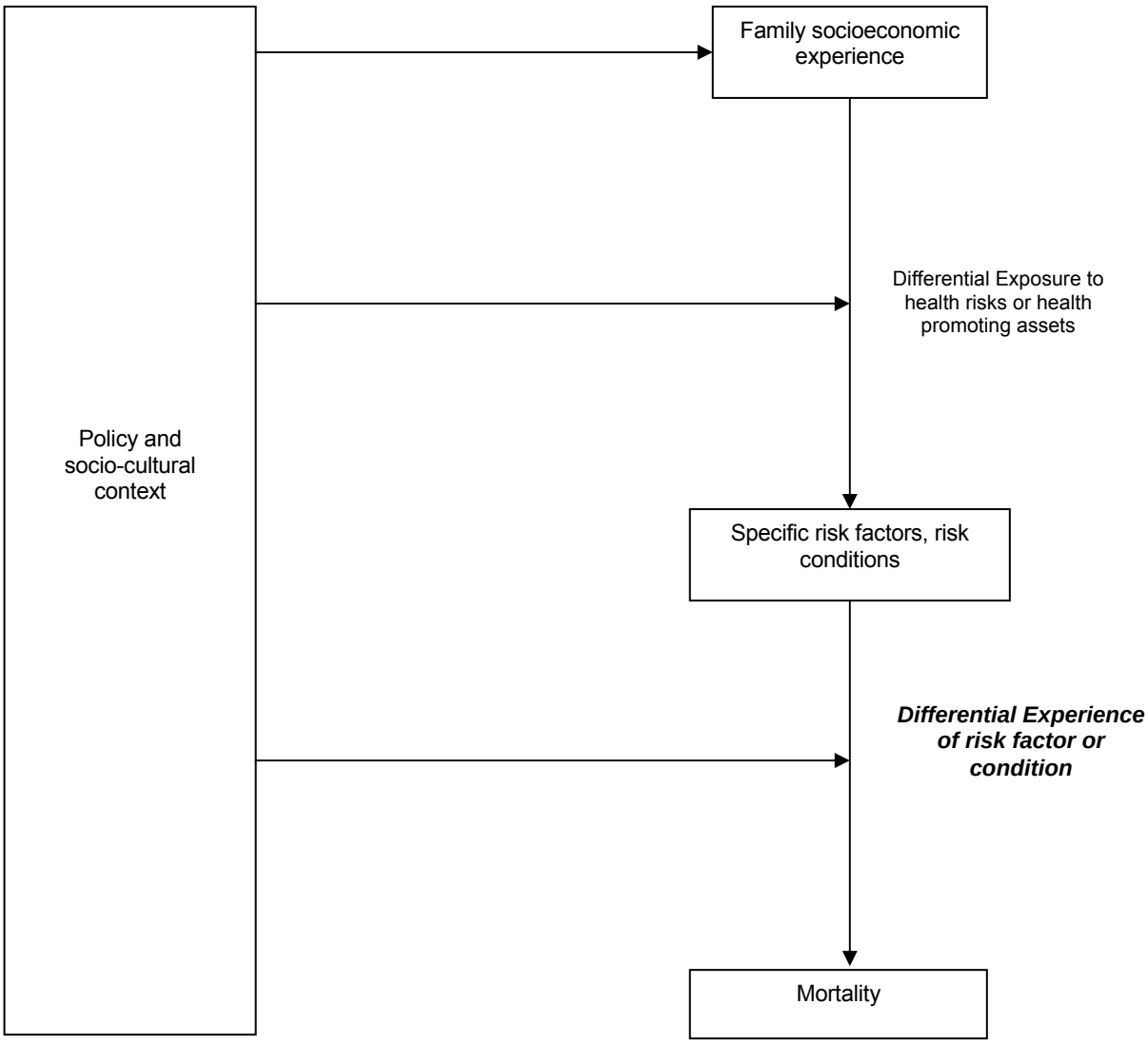


Source: Laflamme 2000

This model has been used to evaluate evidence and set research priorities in injury (Laflamme & Diderichsen, 2000), to explain social differentials in respiratory infections mortality in the developing world (Rehfuess, unpublished) and as a tool to develop policy interventions to assist mental health in single parents (Whitehead et al., 2000).

It has been adapted for this thesis to be more family focused and to encompass more causes of child mortality and temporal changes in policy and social context (see Figure 3). While some causes of child mortality do not show social gradients, this model provides a tool to analyse the reasons for this, allowing lessons to be applied to other conditions.

Figure 3 Modified Diderichson and Hallqvist model of socioeconomic determinants of injury



A few examples of how this model might work are provided. The wider context or structural determinants may directly influence family social experience, for example through the allocation of taxation and tax credits on families with children. This wider context could also mediate exposure to health risks through the placement of local schools. Selective school closure could mean that children were differentially exposed to traffic risks on their walk to and from school. Structural decisions could influence exposure to health promoting resources such as folic acid e.g. the current situation of no fortification means that many women of child bearing age do not have adequate intake of folic acid.

Equally individual SEP may influence folic acid consumption through receptivity to health promoting messages around folic acid or availability of food that is high in folic acid. Structural determinants may also influence how the risk or condition is experienced e.g. through the provision of universal free primary health care, which may mean that children with serious medical conditions are diagnosed earlier in the course of diseases. Individual SEP may influence this as well, for example through ability to access services. The dynamic nature of policy, social, demographic and economic changes that influence our lives are also captured by this model.

However as with any model there are limitations. For example there is no explicit mention of life course perspectives, including the effect of the in-utero environment, although it could be argued that the concept of differential exposure encompasses this.

There is a somewhat artificial blurring of divisions in this model, as interventions and explanations can be thought of in many frames. This is particularly the case when considering whether an exposure is governed by structural determination of SEP through societal distribution of life chances, taxation etc, or an individual's choice within their SEP. In reality it is probably not a dichotomy, but a combination of the two. The blurring does not mean that this is an ineffective way of looking at child mortality, it simply means (I think) that it is impossible to completely distil a complex issue such as the generation of inequalities into a few lines and arrows.

1.3 Measurement of Socio-Economic Position

This section will cover how socioeconomic position is measured, what is measured, the problems with these measurements, and the possible micro-level

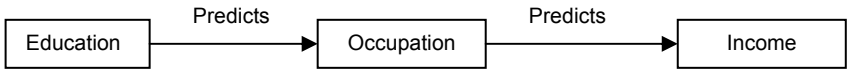
causal pathways between these exposures and child mortality. Much of the latter discussion is theoretical as there is little analysis of SEP as it specifically pertains to children. It should not be assumed that the eco-social pathways to inequality in mortality are the same for children and adults. Problems with comparing these measures over time and cross-nationally are addressed in section 1.1.

The measurement of SEP tries to capture, at the individual level, the process of societal stratification. The measurement of individual SEP is complex and continually evolving and no one measure can capture the complexity of the construct. The most common techniques to measure SEP are the triad of income, education and occupational class, however alternative methods include labour force status, ethnic/racial group, access to resources and area of residence. SEP can be measured at the level of the individual, household and community level depending on what is most appropriate (Krieger et al., 1997). In this study of children all socioeconomic variables are measured on the household level.

Measurement instruments should be considered to be evaluating different aspects of social theory e.g. Weber's economic dimension could be measured through income, and status through a prestige based occupational class measure. One of the difficulties in socioeconomic position measurement is that the theoretical underpinnings of class are constrained by the practicalities of data collection, leading to the most convenient measure being used rather than the actual measure of interest. Examples of this include using area based deprivation measures as a proxy for unknown individual data, or often in the USA, classification by race is viewed as a proxy for income or education (Krieger et al., 1997). However collection of data on SEP is not only influenced by convenience, but also by local political and cultural understandings of social stratification (Berkman & Macintyre, 1997).

One of the complexities of this area is that while measures of SEP are interlinked, each taps into a slightly different aspect of the powerful underlying construct of social stratification (Berkman & Macintyre, 1997). However as well as being interlinked, measures of SEP may be predictive of each other (see Figure 4), making it difficult to estimate the 'independent effect' of each variable, as a confounder may also be on the causal pathway.

Figure 4 Diagram showing how socioeconomic position measures could predict each other



It should also be noted that explanations of the causal pathways between individual measures of SEP and mortality are likely to be reductionist, as the pathways to inequalities are extremely complex. This quote is about occupational class, but similar themes apply to education and income:

> "In reality occupational class cannot be distilled to one or two causal paths as not only does it inform researchers about the working lives of individuals but offers insight into their social community, their financial and residential resources, their cultural experiences, their health related behaviours, and even the life-course opportunities open to them and their children" (Davis et al., 1997).

This thesis is about children whose socioeconomic position is determined by their parents or caregivers. In children with two parents, whose socioeconomic position should be utilised? Two approaches to this issue have been identified. The dominant approach considers that the highest level exerts the most power over the socioeconomic position of the household, while the cross class method assigns class based on a pre-assigned head of household (Krieger et al., 1997). Commonly father's occupational class is measured and/or mother's education but there seems to be little empirical evidence for this.

One study in Norway contrasted the independent effects of both maternal and paternal education on stillbirth and neonatal mortality. They found that mother's education seemed to be more closely associated with post neonatal mortality and, rather curiously, that father's education was more closely associated with stillbirth (OR 1.7 95%CI 1.25-2.31) (Arntzen et al., 1993). Interpretation of this study is hampered by the fact that the 20% of children with the highest death rates had no father specified on the birth certificate. It is possible that they may have had a different distribution of the factor under study than the other children thus raising the possibility of selection bias.

1.3.1 Income

Income is thought to be relevant to health in three ways: through Gross Domestic Product, personal income, and income inequality (Marmot, 2002). The relationship between increasing Gross Domestic Product and improving health (usually indicated through life expectancy) is well described (World Bank, 1993), and the relationship between income inequality and health continues to be debated (Judge & Paterson, 2002; Kawachi et al., 2002); it is individual and household income that are relevant to this thesis.

1.3.1.1 Individual Income and Wealth

Measurement of income captures the economic dimension of social class theory. While income seems a relatively transparent procedure of measuring SEP, the there are a number of well-known problems, mainly with methodology.

Income is usually measured as a snapshot in time, which is a practical but limited approach, as it does not capture the dynamic nature of income or the life course income experience of individuals. Longitudinal panel surveys have confirmed that persistent poverty for adults and children is more harmful to health then occasional episodes (Benzeval & Judge, 2001; Lynch & Kaplan, 2000). Average income over a time period may be more likely to capture work instability and subsequent health effects; however this is difficult to collect.

Income fails to capture the entire range of economic resources that an individual has access to. For example it does not capture bartering or the informal economy (e.g. cash jobs) (Berkman & Macintyre, 1997), neither can it account for differing regional costs of living which can alter the actual purchasing power of an individual (Liberatos et al., 1988).

Income also does not include assets/wealth, which are needed to accurately determine the economic position of an individual (particularly the elderly). Wealth is defined as the accumulated stock of assets or economic reserves, such as cars and houses. There is some evidence suggesting wealth exerts an independent effect on health (Duncan et al., 2002). However it may also be that wealth reflects life course income opportunities, for example there is evidence that different ethnic groups may have different levels of wealth even for the same income level (Lynch & Kaplan, 2000).

However regardless of methodological concerns, one of the main problems with income is respondent sensitivity to the topic, which frequently results in a high non-response rate. This leads to a potential selection bias problem in any analysis. To try and overcome this problem often a tick box is used that encompasses a range, however this then creates a discrete variable, with little idea of the actual distribution within the range (Liberatos et al., 1988).

1.3.1.2 Family Income

It is apparent that an individual's income may not be an accurate estimation of how much income they actually have at their disposal. This applies particularly in families where children are dependent on parental income, and one parent may only engage in part time work. It is also difficult to compare a family of three living on $30 000 to a family of six living on the same amount. To classify these people on the absolute income to which they have direct access would be misleading and comparisons would be meaningless. Hence a measure that adjusts for the number and type of people who depend on a certain income is needed, to allow meaningful comparisons (Statistics New Zealand, 1999b).

Equivalisation scales adjust for all these variables. They take into account the number of adults, and children in each household and adjust for their relative 'costs' and the economies of scale generated by the sharing of expenses. Factors that are considered important in equivalisation scales (besides income and household size) include gender, employment and relationship status (flat mates versus partners) (Easton & Ballentyne, 2001).

Equivalisation scales are contentious, with debate about the most valid scale (Easton & Ballentyne, 2001). The underlying assumption of equivalisation, that household income is distributed to the individuals within the house according to need, may also be contentious.

The possible causal paths through which income could affect child mortality are probably similar to those of adults. Material interpretations could be applied, i.e. increasing income allows access to health promoting physical resources such as car seats, fenced sections and 'child safe' appliances. Increasing income could also allow parents to decrease their children's exposure to 'toxic environments' such as living in areas with high traffic flow. Each step up the

income ladder may bring additional benefits, which may accumulate and be passed to children in terms of additional opportunities to learn, or access to better resources during crucial early development (Lynch & Kaplan, 2000; Marmot, 2002).

The neo-material pathway may also be important to the income-child mortality relationship. Neo-material interpretations are that increasing income could be associated with increased social participation, personal psychological resources, and health behaviours (Lynch & Kaplan, 2000). These could assist parents to provide a safe environment for child development. It is also possible that income could be a marker of education or occupation, which are the true causal determinants.

In adults (and therefore possibly children) improvements in health and mortality are subject to diminishing returns, that is as income increases there is a flattening of the effect of income (mortality cannot be reduced to zero) (Blakely et al., in press; Lynch & Kaplan, 2000). There is also some evidence of a contextual effect of income, where income of those around an individual affects their health irrespective of their own income (Kawachi et al., 2002). This may be particularly important for children (Krieger et al., 1997).

1.3.1.3 Health Selection

One of the key issues when considering the income/health gradient is reverse causation/health selection. This was one of the 'soft' explanations proposed in the Black Report for the explanation of health gradients (Townsend et al., 1992). This suggests that instead of income predicting health, health predicts income; a person in poor health is unable to work therefore will have less income. To be confident that the relationship between income and health is not

reverse causation, longitudinal studies with multiple measures of health and income over several points in time are required.

The literature suggests that, for adults, reverse causation may account for a small amount of the income/health gradient, however it does not completely 'explain away' the relationship (Benzeval & Judge, 2001; Chandola et al., 2003). For children it is even less likely that health selection is an issue, given that the majority of deaths in this age group are due to injury, which is an unanticipated event. It seems very unlikely that parental income would decline prior to an injury death, however for child cancer and congenital mortality there may be an effect. For example if one parent, who previously worked, stays home to care for a sick child then income may decline. However parental education is unlikely to be affected by health of the child, so the existence of gradients can be compared between these two measures.

1.3.2 Education

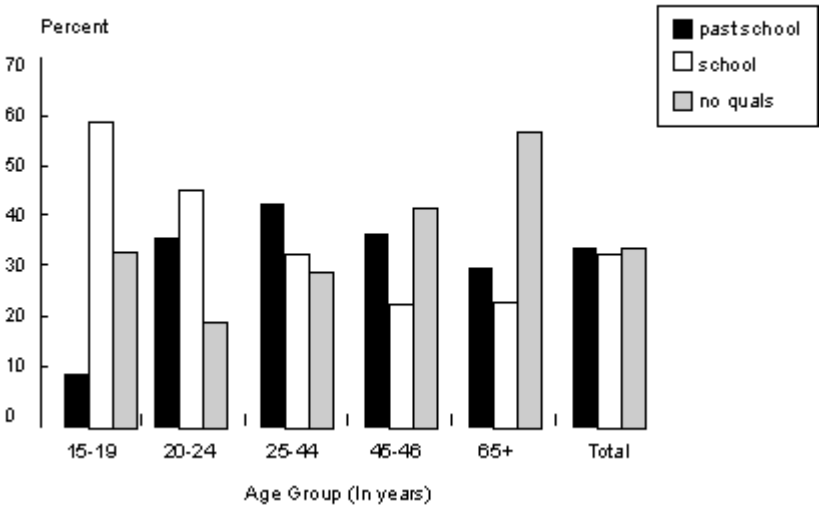
Education is probably the most widely used of the traditional SEP measures; it is easy to capture and applies to all adults regardless of their employment status. Education draws in the economic and prestige domains of Weber's typology of class (Berkman & Macintyre, 1997). It also has the advantage of usually being completed prior to illness (usually by the mid twenties), thus not being as susceptible to health selection. Education reflects the life course opportunities and paths offered in an individual's childhood and teenage years, as well as capturing the transition period from parental SEP to own attained SEP (Lynch & Kaplan, 2000).

There is some debate about the type of educational measure that should be used, years completed of schooling or qualifications achieved. Within most

countries there is a compulsory level of schooling, reflecting the concept of education as a merit good, which will skew the distribution of years of schooling. Thus it may be that the attainment of qualifications in those compulsory years is a better way to distinguish groups of individuals.

Education is a cohort specific measure. Changes to the social environment in the last one hundred years mean that each cohort has had a different experience of education, indicating that the social and economic value of education varies over time (Lynch & Kaplan, 2000). In New Zealand the cohort of now 70 year olds had fewer educational opportunities but lived in a time of great social mobility. More recently, in the last 30 years school retention and attainment of both school and post school qualifications has increased (see Figure 5). This means that comparisons of health gradients by education are more easily assessed within cohorts, rather than between them (Berkman & Macintyre, 1997). However regression based measures of inequality are a solution to this problem.

Figure 5 Highest Educational Qualification in population of New Zealand 15 years and over, 1996.



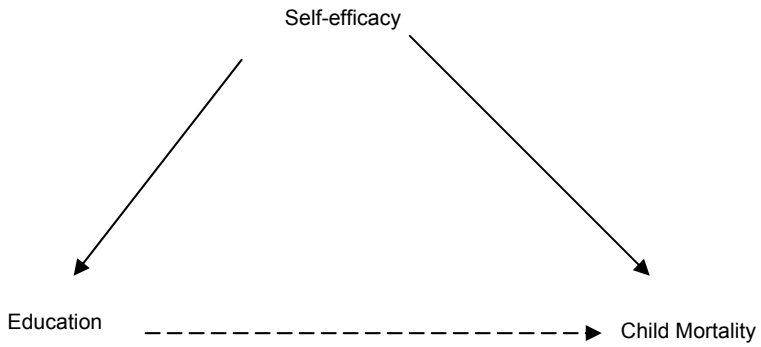
Source: Statistics New Zealand

It is important to note the benefits of education in terms of employment and income are not uniformly distributed in the population. There is evidence in the USA that women and minorities have lower economic returns for the same investment in education than white men (Lynch & Kaplan, 2000). This could represent a number of societal processes, from heterogeneity to discrimination. However it does mean that interpreting education as an indicator with the same consequences for all groups in a society is probably simplistic.

The paths by which education may influence child health are interesting. For adults one of the explanations the relationship between increasing education and decreasing mortality is that higher levels of education influence/improve receptivity to health education messages in adults and thus health behaviours (Blane, 2003). This is seen in child mortality in the developing world, where there is a recognised relationship between increasing maternal education and decreasing child mortality, thought to be mediated through increasing knowledge of health promoting skills, such as boiling water before a child can consume it (Macassa et al., 2003; Millard, 1994). This mechanism may have a role in explaining some of the gradient in child mortality in developed countries, particularly avoidable mortality, infectious diseases and neural tube defects. However, differences in receptivity to health promoting messages and subsequent behaviour are strongly shaped by the social environment (Lynch et al., 1997). The focus on individual knowledge and behaviour can ignore the structural constraints within which individuals make decisions.

Other causal pathways of educations are that education is a proxy for occupational class and/or income, which are the true causal paths, i.e. level of education predicts future occupational and economic opportunities (Blane, 2003; Schrijvers et al., 1999). Alternatively a background variable, which influences both education and child health, (e.g. parental self-efficacy, intelligence or 'willingness to invest') may actually be the causal variable (see Figure 6).

Figure 6 Illustration of background variable mediating education and child health



1.3.3 Occupational Class

Occupational class involves the classification of individuals into groups based on their employment. The assumption being that occupation is a reasonable basis to allocate people into socioeconomic hierarchy and that health effects flow from the division of life chances by occupation (Davis et al., 1997). However occupational information is used in a variety of other ways in research (other than as an indicator of social class) such as to infer occupational exposure, as a confounder or to examine hypotheses regarding which occupations are at greater risk of particular diseases (Mannetje & Kromhout, 2003).

Occupational class measurements have different meanings depending on the country and the scale used (Berkman & Macintyre, 1997). There are a myriad of measures used in different countries (Lynch & Kaplan, 2000). These can be loosely divided into those that are categorised into groups based on: income and/or educational level in the occupation, hierarchy within an organisation, prestige based on population opinion polls, or type of work e.g. manual/non manual. There are scales that look at individual variables and composite scales that take more than one aspect of these into account. Each of these scales has

a different theoretical underpinning. For example prestige-based measures encompass the Weberian status domain and those based on average income and education within a group tap into the class domain, which is more concerned with ownership and economic resources. The manual/non-manual distinctions may be more related to a Marxist view.

Micro level causal paths from occupation to health or death can be thought of as including direct and indirect explanations. The direct path is where occupation is a key determinant of exposure to workplace health hazards, such as asbestos. The differential risk of injury and mortality by occupation would suggest that this analysis is relevant (Feyer et al., 2001), despite occupational health legislation that seeks to control workplace hazards. This direct path may affect children by proxy as parental exposure to toxic substances such as carcinogens may be embedded in their development through prenatal exposure, although this type of exposure is extremely difficult to prove (Sharpe & Irvine, 2004). Presumably this type of exposure would only apply to non-injury mortality in childhood.

Indirect explanations of occupational class cluster around psychosocial theory. Employment in occupations with high demand and low control has been shown to have poorer health outcomes (Lynch & Kaplan, 2000). In relation to child mortality, these indirect effects of occupational class may expose parents to psychological stress, which may be related to child health/rearing practices.

Occupational class, like all measures of socioeconomic position, has a number of well-recognised shortcomings. It is only possible to classify those people in society with an occupation, but exclusion of the economically inactive may underestimate social class differences in health (Martikainen & Valkonen, 1999). There are populations who are difficult to classify within existing schema, including farmers, women who do not work, older persons and children (Berkman & Macintyre, 1997). Occupational based ranking is a culture bound

phenomenon and for different ethnic groups these classifications may operate differently. For example population based prestige measures based on pakeha values may be irrelevant for other ethnic groups such as Māori or Pacific people.

Similar to education, occupational class is also a dynamic concept. Group sizes change over time, there is relocation within the hierarchy and de novo professions arise that need to be accommodated within the structure, while others need to be removed as they are in no longer in existence.

1.3.4 Confounding and Effect Modification

Confounding is defined as a 'confusion of effects'. That is, the apparent effect between the exposure and the outcome is distorted because an extra factor is mistaken for or mixed with the actual exposure effect (Rothman & Greenland, 1998). This distortion may lead to an overestimation or an underestimation of the effect. The extra factors that distort the effects are called confounders, although these factors may only be a representation of the actual confounder. To satisfy the conditions for confounding a factor must meet three criteria (although even if a factor satisfies these criteria it may not be a confounder):

- The factor must be a risk factor for the outcome independent of the exposure,
- It must be associated with the exposure under study in the source population,
- It cannot be an intermediate step in the causal pathway between the exposure and the disease (Rothman & Greenland, 1998).

Effect modification means that there is variation in the size of the effect of an exposure if it is stratified into categories of a third variable. This heterogeneity of effect is different from confounding, as it is a property of the effect the study wishes to categorise (Rothman & Greenland, 1998). A factor can be both a confounder and an effect modifier.

In this study if we were assessing, for example, the independent effect of maternal education on child mortality, we would need to consider whether other factors would meet these definitions of confounding. Common demographic factors to consider are the age, sex and ethnic group of the child. Sex is unlikely to be a confounder as although it is a risk factor for the outcome, it is unlikely to be associated with the exposure. Maternal education is usually (but not always) finished prior to the birth of a child and sex is not on the causal pathway between maternal education and child mortality. However it may be an effect modifier in this relationship, as there may be heterogeneity of the effect of maternal education on child mortality by sex.

Ethnic group may act as a confounder as it is a risk factor for the outcome and may also be associated with the exposure if the distribution of education is different between ethnic groups in the population. Age could also act as a confounder as it is associated with the exposure (younger children die more than older children in New Zealand (Ministry of Health, 1998)) and it may also be associated with the exposure. That is younger children may be more likely to have parents who have not finished qualifications. However it may also act as an effect modifier if despite standardising for sex, there are still differing relationships by child age of the relationship between maternal education and child mortality.

Other factors to consider are whether other socioeconomic variables such as occupational class and income act as confounders of the relationship between maternal education and child mortality. They would both meet the first two

criteria of confounding as they are both associated with child mortality and are also associated with level of maternal education. However they may also both be on the causal pathway between maternal education and child mortality as maternal education level may predict occupational opportunities and also income (see Figure 4).

However this thesis is only describing gradients by socioeconomic variables, the purpose is not to assess each variable independently. Hence while these gradients will be confounded by the other factors (including socioeconomic factors), unless the level of confounding by these other factors has changed over time, this is unlikely to affect the trends. However effect modification by sex, age and ethnic group will be looked for in the results.

2 Inequalities in Child Mortality

While literature on the existence of adult socioeconomic gradients in mortality is extensive, there has been relatively less produced on children. However gradients in infant mortality have been observed for almost a century in the United Kingdom (Macintyre, 1997). This relative lack of contemporary literature may be due to methodological problems associated with data collection, and statistical uncertainty arising from small numbers of deaths in this age group, making it difficult to draw firm conclusions.

The research that has been conducted into childhood mortality inequalities has confirmed the existence of inequalities, but there is variation in both the level and the factor studied. There is also considerable variation in the methodology of studies, with differences in both the exposure and outcome variables collected.

The purpose of this section is to review contemporary literature on the existence of social inequalities in child mortality with particular interest in those that have looked at trends over time. Only literature from developed countries is considered, due to differing causes of mortality in the developing world and potentially very different interventions. However initially there is a short section highlighting some of the difficulties in comparing child mortality gradients across countries and assessing changes over time.

2.1 Comparing Inequality

This section aims to discuss some of the problems in comparing studies of socioeconomic gradients in health. These are in three broad subject areas of cohort changes, measurement differences between countries and methodological differences, however there is considerable overlap in them. Some of the problems are common to both international comparisons and trends over time.

2.1.1 Changes in Cohorts

Within a country changes between cohorts can make comparisons over time difficult. The change in the distribution of education and occupational class is associated with a change in the social meaning and life course opportunities open to individuals in the group. For example education distribution in New Zealand has changed (see Figure 5 in Section 1.3.2) making this variable not very useful to distinguish elderly people (and possibly younger people as education becomes more homogenous in this society). Valkonen provides an example of the changing distribution of occupational class over time (Valkonen, 1993).

2.1.2 Measurement Differences Between and Within Countries

The way in which SEP is measured is different between countries, making cross-national comparisons difficult. The direct comparison of two different measures is not appropriate given that each measure of SEP taps into a slightly different domain of the underlying construct. Evidence supporting the fact that

measures of SEP are not interchangeable comes from studies that look at multiple measures and see different strengths of association and different time trends with each measure (Blakely et al., 2003; University of Otago & Ministry of Health, 2004).

Valkonen identified a number of issues that arise in international comparisons, which include the coverage of the study, the level of detail of the socio-economic groupings and the time period covered (Valkonen, 1993).

Within each of the main measures of SEP there are well known problems. Within education there are different measures, such as qualification attained (Blakely et al., 2003), or years of schooling completed (Mare, 1982). These imply different levels of achievement, related to attendance (which may be determined by legislation) or mastery. If the comparison is years attended then the legislative environment needs to be considered as countries may have different minimum levels of education.

Income appears to be a more straightforward comparison, however careful attention needs to be paid to the way the income information was collected (eg pre tax, post tax, all income sources, wealth), refusal rates and how the incomes are grouped for comparison. Purchasing power of the income in different countries needs to be adjusted for, as do the effects of inflation.

Constructs such as poverty need to be interpreted with care in the international context as they may be based on different concepts and must be seen in the context of the surrounding society. For example the US Federal Poverty line is a construction of poverty as an absolute level, based on the income needed to buy a meal (Kawachi et al., 2002) whereas New Zealand does not have an official poverty line (Blaiklock et al., 2002). However some government

departments use a figure of less than 60% of median income e.g. Ministry of Social Development (Ministry of Social Development, 2001).

Occupational class is perhaps the most problematic measure for international comparisons (and interestingly the most common measure available in the literature on child mortality). The classification of occupational class may be completely dissimilar between countries. In the studies reviewed later in this section the Registrar Generals social class, the New Zealand Socio Economic Index (NZSEI) and manual/non manual classifications are all used (Blakely et al., 2003; Botting, 1997; Vagero & Ostberg, 1989). The number of occupational groups in each study ranges from 3 to 7. The theoretical basis of each of the measures is different with manual/non manual being based on the type of activities performed in each occupation (Vagero & Ostberg, 1989), while the NZSEI is based on a weighted statistical algorithm combining income and education and correcting for age (Davis et al., 1997).

Studies using occupational class deal with the economically inactive differently, either excluding them completely, or including them for some analyses. This is particularly concerning as exclusion of the economically inactive results in considerable underestimation of the relationship between socio-economic position and mortality in both children and adults (Judge & Benzeval, 1993; Martikainen & Valkonen, 1999).

Even if the same occupational classification was used for all countries there are differences in the number of people that will be eligible for classification in each country and the spread of persons within the different groupings (Quine, 1990). These differences may reflect the socio-economic structure of those societies, which is, in turn, tied to health and mortality outcomes. The size of the extremely deprived group may tell us something extremely important, as they often have the highest risks.

2.1.3 Methodology

Many of the issues above can be solved by standardisation of data collection between countries allow comparison of rate ratios and differences (although this may not address any country specific social meanings associated with the variable)(Mackenbach et al., 2003). However the methodology used to assess socioeconomic inequalities in health can also make comparisons difficult. One of the reasons for this is the multiple measures available and a prior lack of agreement on what should be presented. Mackenbach and Kunst identified 11 different methods of measuring socio-economic inequalities in health in their paper. However they also identified a systematic way of considering these methods, distinguishing them by whether they are measures of effect or impact and the degree of sophistication of the measurement techniques (see Table 2) (Mackenbach & Kunst, 1997).

Table 2 Methods of presenting data on socioeconomic inequalities in health.

	Relative	Absolute
Effect / Association	Rate ratio	Rate difference
↑ ↓	Relative index of inequality (RII)	Slope index of inequality (SII)
Impact	Population attributable risk (absolute version)	Population attributable risk (% or relative version)

One of the simplest issues to consider is the use of absolute and relative differences when reporting inequalities. Both absolute and relative differences are vital to fully consider inequality. The selective reporting of only one of these measures can be misleading, as greater inequality in a rare disease may be less important on a population level than less inequality in a common condition (Mackenbach & Kunst, 1997). This is particularly important when analysing time

trends, as changes in rates can lead to changes in both absolute and relative inequality.

The problem of comparing groups with dynamic numbers either between countries or over time was identified above. The main problem is that rate ratios and rate differences between high and low do not take into account the numbers in each group over time, which may be becoming more divergent in their distribution and life opportunities (Carr-Hill, 1988; Mackenbach et al., 2003; Quine, 1990; Valkonen, 1993). Regression based statistical methods identified in Table 2 that take into account the changing distribution of the socio-economic exposures across the population, changing group size over time and different distribution of groups between countries are able to be used to provide more robust comparisons (Mackenbach & Kunst, 1997).

2.2 Individual Level Socio-Economic Position Measures

Those studies that can link together a child's socioeconomic position and the death record of that same child are clearly in the strongest position to draw conclusions about the relationship between mortality and SEP gradient. Difficulties arise due to the large amount of resources needed to do cohort studies and the relative infrequency of mortality events in child populations, so data linkage studies are more numerous for investigating this area.

Table 3 summarises studies that have individual level SEP data. The majority of studies confirm the existence of socioeconomic gradients in all-cause childhood mortality. Studies are then discussed if there are additional points of interest.

Table 3 Summary of results of studies of socioeconomic gradients in all-cause child mortality using individual SEP measurements.

Authors	Country and Year	Study Type	Age	SE Variable- unless otherwise specified the comparisons are lowest/highest	Results (95% CI where available)
Blakely et al	New Zealand 1991-94	Linked census and death records	1-14	Education (highest in house) Occupational class (highest in house) Income (equivalised household) Car access Labour force status	RR 1.8 (1.3-2.3) RR 1.8 (1.4-2.4) RR 1.9(1.3-2.6) RR 2.2(1.6-3.0) RR 1.4(1.1-1.7)
Botting	UK 1991-93	Death records with census denominators- Unlinked	1-15	Registrar Generals Occupational Class (Paternal)	RR 2.33
	UK 1987-91	ONS Longitudinal cohort data	0-14		RR 3.29
Gissler et al	Finland 1987-1994	Linked Medical Birth Register and Death Records	0-6	Occupational Class (Maternal)	OR 1.32 (0.93-1.88)
Judge K, Benzeval M.	UK 1979-83	Death records and census denominators- Unlinked	1-4	'Unoccupied' compared with RGs Highest Occupational Class	RR 2.58(2.07-3.22)
			5-9		RR 2.56(1.98-3.30)
			10-14		RR 4.14(3.43-4.99)
Mare R	USA 1955-1975	Case-control study	0-19	Income (Family- unknown if equivalised)	OR 1.39 (Female 5-9) OR 1.53 (Male 5-9)

Authors	Country and Year	Study Type	Age	SE Variable- unless otherwise specified the comparisons are lowest/highest	Results (95% CI where available)		
				Education (maternal)	OR 1.80 (Females 59) OR1.50 (Male 5-9)		
Nelson M	USA 1985-1988	Death records linked to welfare payment records	28 days to 17 years	Recipients of Welfare payment or Medicaid compared to all other children in the state	RR 2.7		
Nerserian W, Petit M et al	USA 1976-1980	Death records linked to welfare payment records	8 days to 17 years	Recipients of Welfare payment compared to all other children in the state	RR 3.14 (2.68-3.67)		
Ostberg	Sweden 1981-86	Linked census and death records	1-19	Occupational class (Highest in house)	Boys 1.58 (1.4-1.8) Girls 1.32 (1.1-1.6)		
Pensola and Valkonen	Finland 1987-1995	Linked census and death records	5-34	Head of Household occupational class		5-9	10-14
					Females	RII 1.01	RII 1.36
					Males	RII 1.95	RII 0.89
Schuman	UK 1993-1995	Linked death and birth records	0-3	Occupational class at time of birth (Paternal)	RR 1.7 (1994 cohort of 1-2 year olds)		

Authors	Country and Year	Study Type	Age	SE Variable- unless otherwise specified the comparisons are lowest/highest	Results (95% CI where available)		
Singh and Yu	USA 1979-1985	National Longitudinal Mortality Cohort study	1-14	Income (Family- unknown if equivalised)	1-4	<10 000	RR 3 0 (1.39-8.05)
						$10 000 to $14 999	RR 3.4 (1.16-7.85)
					5-14	< $20 000	RR 1.62 (1.08-2.41)
West	UK 1959-63	Unlinked death records and census denominators	1-14	RG Occupational Class (Paternal)	No precise RR available but gradient observed 1-9 year olds, flattens for 10-14		
West	UK 1979-80 and 1982-83	Unlinked death records and census denominators	1-14	RG Occupational Class (Paternal)	No precise RR available but gradient observed 1-9 year olds, flattens for 10-14		
Vagero and Ostberg	Sweden 1961-79	Linked census and death records	1-19	Head of household occupational class (usually father)	Girls RR 1.38 (1.3-1.5) Boys RR 1.12 (1.0-1.2)		

Blakely et al used anonymously linked census and death records, weighted for linkage bias. Multiple measures of SEP were available through the census data and multiple outcomes were also reported, with a total of 435 deaths from 1991-1993. In the 1-14 age group an inverse relationship was noted for all measures of SEP and all-cause mortality (i.e. lower SEP showed an increase mortality risk (see Table 3 for absolute figures)). Interestingly the relative risk was strongest for car access (a resource based measure of SEP) and the weakest (although still statistically significant) for labour force status (Blakely et al., 2003).

Blakely et al also reported associations by mortality type for the 0-14 age group. Injury mortality was consistently graded by socioeconomic status, however gradients were also noted in cancer, congenital mortality and other mortality (although confidence intervals included 1 for all of these causes). This paper was particularly interested in exploring whether there was any additional relationship between child mortality and sole parenting, not explained through the deprivation associated with this family type. No such relationship was found.

Botting reported from two data sources. The first was unlinked death and census records between 1991-93. The age standardised mortality rate of RG occupational class group I was 18/100 000 children aged 1-15 and for Group 5, 42/100 000, giving a relative risk of 2.33 (no confidence intervals available) (Botting, 1997). The other occupational groups had standard mortality rates that were quite close (although slightly higher) to that of the social class group 1, but group 5 showed a threshold effect with a sudden rise in mortality rate. Unlinked data is subject to numerator/denominator bias, as the information on SEP of the children that died (i.e. the numerators) does not come from the same source as that of the denominator population (Valkonen, 1993).

Botting also reports on data from the ONS Longitudinal Study, which is a national cohort study involving 1% of the population of England and Wales. It showed mortality rates of 37-122/ 100 000 children of 0-14 with a RR of 3.29

(no confidence intervals available). Again the comparison is between Group 5 (lowest) and Group 1 (highest) of the RG Occupational Classification. The mortality rates of the other social class groups show more variation then the population figures, however they also seem to show a threshold effect at social class 5, with gradually increasing mortality rates in the intervening social classes.

Gissler et al, followed a retrospective birth cohort of 59 865 children born in 1987 in Finland. The data were collected using linked datasets (medical birth register, death records, population registers, childcare support, medication use database, hospitalisations and school registers). The exposure of social class was measured using a national occupational classification system: upper white collar, lower white collar, blue-collar workers and other (farmers, students, housewives and unknown). The adjusted odd ratio for mortality for ages 0-6 is presented in Table 3. However age specific mortality rates suggest that this gradient arises from differences in mortality by SEP in the under 1 age group. While adjusted odds ratios are not presented by age group, mortality rates in the 1-6 age group are 1.4/1000 live births in both the highest and lowest social class group. The study also examined health indicators and found a positive association between deprivation and poor health suggesting that socioeconomic gradients in health can occur even in a relatively egalitarian social structure (Gissler et al., 1998).

Of concern in the above study is that the exposure of social class was assessed when the children were ages 7, after the outcomes of interest had occurred. The authors state that measuring mother's social class at the time of birth may be more inaccurate as a woman is more likely to be in a temporary occupation at that time (Gissler et al., 1998). While this may be true, measurement at 7 years is unable to exclude health selection effects, particularly when looking at morbidity (i.e. maternal social class may have declined after onset of childhood illness).

Judge and Benzeval addressed the issue of those children that are excluded from social class analysis in the UK because parental employment was classified as 'unoccupied'. They noted that while only 6% of the children in the UK were in this class, it accounted for 14% of the total deaths. They hypothesised that the majority of these children live in sole mother families who are largely dependent on state benefits, with resulting deprivation, and thus may have had higher mortality risks than other children (Judge & Benzeval, 1993). Analysis of the age specific mortality rate of children in this 'unoccupied' group aged 1-15 showed a rate of 68.8/100 000 which was higher than 48.4/100 000 in social class 5. The relative risk of death, for 10-15 year old children, in this group when compared to the top occupational class group, was 4.14 (95% CI 3.43-4.99).

This study used unlinked data, thus being subject to numerator/denominator bias. The authors reviewed the techniques used to classify people into social classes and believed it was reasonable to assume that those who were classified as 'unoccupied' were the same in both census and death records (Judge & Benzeval, 1993). They also had to estimate the likelihood of this unoccupied group being single parents, as there was no direct measure. Other authors have challenged these assumptions (West, 1997), and while these methodological issues are unable to be completely addressed, this study highlights the inadequacy of any system based on economic activity for the complete monitoring of socio-economic gradients in child mortality.

Mare used cross sectional survey data collected from the 1975 Current Population Study questionnaires to retrospectively analyse child mortality experience in 45 000 households across the USA (Mare, 1982). Women (aged 75 and under) were interviewed for fertility histories and survival status of up to 5 children, however only births within the last 20 years were analysed for this study (i.e. born from 1965). While results show a relationship between

decreasing SEP (income level and maternal education) and increasing child mortality, design faults, which include the SEP exposure information being collected after the mortality event and a 25-50% undercounting of deaths, make this study extremely suspect.

Nelson studied the relationship between socioeconomic status and child mortality in North Carolina 1985-1988. He used a dichotomous classification of SEP, identifying those children who were either receiving welfare payments -Aid to Families with Dependent Children (AFDC), or were Medicaid participants who were eligible to receive AFDC as the exposed group. The non-exposed group was all other children in North Carolina aged 17 or younger obtained from population data. Deaths were matched to either of these two groups (Nelson, 1992).

The actual mortality rates were not presented in this study, however for all age groups combined children receiving AFDC were 2.7 times more likely to die than children who were not ($p<0.001$). The rate ratios varied by cause of death and age group, however extremely high rate ratios were seen for fire death in 1-4 year olds (RR 9.0), and pneumonia (RR 24.2) and homicide (RR 15.4) in the 10-14 age group. Risk by racial group was also compared and no differences were found between deprived black children and deprived white children suggesting any differences are due to SEP rather than ‘race’ (Nelson, 1992).

Due to the brevity of the paper there are some questions surrounding the methodology. The lack of confidence intervals does not allow us to look at point estimate precision, pooled results from 28 days to 17 years may not be age standardised (this is unclear) and there is no indication of the differences in absolute risk, which may be quite small despite high risk ratios. It is not specified as to whether the children had to be receiving AFDC at the time of death or whether ever receiving it was a marker of poverty. There is also no

indication of the proportion of children in North Carolina who were receiving this benefit or what the eligibility criteria were.

It is likely that the children in this study comprise a small, extremely deprived group of the population with very high risk of mortality, similar to the 'unoccupied' group in Judge and Benzevals' paper (Judge & Benzeval, 1993). Information obtainable from 1993 (5 years after the study had ended) supports this conclusion, showing that the upper income threshold for receiving this benefit was $6 528 for a family of three (which is 51% lower than the federal poverty line, which in turn is set at less than 60% of median income) (Pediatrics, 1994). While it is imperative to consider very deprived groups of children in society in any research or interventions, it is also important to recognise that the majority of deaths (in fact 86% in Nelson's study) will occur in the rest of the population who are at lower risk (Rose, 2001).

Similar to Nelson's work above, Nersesian studied a similar group of children in Maine in 1976-1980 (Nersesian et al., 1985). The exposure of low SEP was slightly different in that it covered Medicaid and Food Stamp recipients, as well as AFDC. Only children who were receiving these benefits at the time of their death were considered in the 'low income' group.

He found that children in this 'low income' group had much higher mortality rates than 'other' children e.g. 139.7/100 000 vs. 44.6/100 000 for all causes (aged 8 days to 17 years). The most significant drivers of inequalities were accidental deaths and disease related causes (which included perinatal conditions and congenital causes), which were both common and graded by income (RR 2.59 (95%CI 1.95-3.42) and 3.52 (95%CI 2.90-4.29) respectively). While some of the causes of death had very strong measures of effect e.g. fire deaths with a rate ratio of 5.01 (95%CI 2.78-9.04), because they were actually only a rare cause of death (52 deaths in total out of 1 038) they did not have a big impact on the absolute gap (Nersesian et al., 1985).

While grouped results were not age standardised in this study, age specific strata rates confirm the findings that low-income children had higher death rates in all age groups (Nersesian et al., 1985). This study focuses on socioeconomic differentials as a threshold rather than a gradient. This may be a function of data availability in the USA but similar to the previous study by Nelson it ignores the gradient.

Ostberg's paper used linked census and death registers in Sweden to study gradients in child (1-19 years) mortality during the period 1981-1986. The exposure of socioeconomic position was "social class" as based on parental occupation type (highest in house of either parent). In the 1-19 year group an inverse gradient was seen between mortality and level of social class, giving a rate ratio of 1.58 (95%CI 1.4-1.8) for boys and 1.32 (95% CI 1.1-1.6) for girls when comparing lowest to highest class. In finer age bands the most striking relationships were seen in the 5-9 age group with rate ratios of 1.89 (95% CI 1.4-2.6) and 2.15 (95% CI 1.4-3.4) for boys and girls respectively (Ostberg, 1992). There was evidence of monotonic increased in mortality risk with decreasing SEP in this study (as opposed to the thresholds seen in other studies).

Socioeconomic gradients were also seen by causes of death, with significant gradients ($p<0.05$) being seen in accident mortality, congenital malformation mortality for both boys and girls and cancer for boys. Ostberg also compared this cohort with a previous cohort from 1960; this will be discussed in the time trend literature section (Ostberg, 1992).

Pensola and Valkonen linked census and death records allowing them to review the deaths of 5-34 year olds who died between 1987 and 1995 in Finland (Pensola & Valkonen, 2000). They used the same measure of SEP as Gissler

et al (Gissler et al., 1998). Unfortunately point estimates are not presented in this paper, but graphs show no evidence of a socioeconomic gradient in the relative risks for all-cause mortality in females aged 5-14 and males aged 10-14. However a gradient was seen in males aged 5-9 and in both males and females in the older age groups (15-34). Gradients can be seen for specific mortality causes (e.g. accidents and violence) but most confidence intervals appear to include 1. The relative index of inequality (RII) is presented for each age strata and sex and these confirm the findings of the rate ratios (see Table 3 for RII values).

There was some systematic bias in this study, with the children of farmers, entrepreneurs and other (who comprised 17-18% of the person time available in the 5-14 age group) being excluded from the analysis, as they were "impossible to fit into the socioeconomic hierarchy"(Pensola & Valkonen, 2000). There is some evidence suggesting that children of farmers have a higher mortality risk than other children (Ostberg & Vagero, 1991), raising the possibility of selection bias in these results.

These Finnish studies are interesting as they contradict the trend seen in other studies that inequalities do exist in child mortality. This suggests that mortality gradients by socioeconomic position are not inevitable and may be modified by local environment.

Schuman presents data from the ongoing project of linking child deaths to birth records in the UK, which started in 1993. At this stage only data from children age 1-3 are available, however as the cohort ages more will be presented. Current findings indicate a gradient, using the RG classification, in the <1 age group (RR 1.56), the 1-2 age group (RR 2.17), and the 2-3 age group (RR 1.29) (Schuman, 1998). These figures may not be entirely accurate as data are being accrued and some of these numbers could be influenced by any yearly

anomalies. Data for the unoccupied are not presented, neither are confidence intervals.

Vagero and Ostberg studied 1.5 million children in Sweden from 1961-1979 enumerated in the 1960 census (Vagero & Ostberg, 1989). Subsequent deaths were linked back to census records allowing analysis of socioeconomic gradients in this huge cohort. Socioeconomic position was classified according to father's occupation: self employed, manual, non-manual. Gradients in mortality were seen when comparing children of manual workers to non-manual workers in the combined age group (1-19) in both boys and girls, with rate ratios of 1.38 (95% CI 1.3-1.5) and 1.12 (1.0-1.2). Finer age bands also demonstrated gradients. Children of self-employed fathers had mortality risks about mid-way between the other groups.

Cause specific mortality was also studied, with children of manual workers having significantly higher rates of death from both accidental and non-accidental mortality (Vagero & Ostberg, 1989). This is an excellent study with only 1% record linkage failure, a large number of outcomes and substantial person time.

West was interested in age specific socio-economic gradients in mortality, hypothesing that they may vary by age. He reanalysed data from the Decennial Supplements[1] of 1960 and 1980, reviewing child mortality for 5 years surrounding them. A marked socioeconomic gradient was seen in 1-4 year olds when comparing class 5 to class 1, but this gradient became shallower in 5-9 and 10-15 year olds before becoming steeper again (West, 1988, 1997). Unfortunately no point estimates or confidence intervals are available. This unlinked data do not include the 'unoccupied' class as highlighted by Judge and

[1] These are produced from the decennial census in the UK

Benzeval (Judge & Benzeval, 1993), although it is difficult to know the size of this 'unoccupied' group in the earlier period.

2.3 Area Level Measures of Socio Economic Position

There are a number of other studies that look at child mortality using area level measures of socioeconomic position. The main reasons for using an area-based measure are convenience and the assessment of a contextual effect (i.e. neighbourhood effects above individual level effects) (Salmond & Crampton, 2000). The majority of studies fall into the first category and use an area level measure of SEP, which aggregates individual level data into an index, as a proxy for individual information that is not available (Greenland, 2001).

As with all measures of SEP area measures can reflect different aspects of social theory, e.g. area measures can consist of measures of economic resources (i.e. average income), or social conditions related to deprivation (or a combination of these). There is considerable variation in the formation of area level measures of SEP. They can be complex constructs with multiple variable inputs (e.g. NZ Deprivation Index (Salmond et al., 1998)), or more simple measures consisting of one variable such as phone access (see section 2.4.3 for an example of this).

The main issues with area-based measures of SEP are measurement error or misclassification. Within an area measure of SEP, there is heterogeneity as not all poor people live in poor areas and vice versa (Salmond & Crampton, 2002), however there is some evidence (and indeed a theoretical expectation) that smaller area units are less susceptible to this misclassification (Crayford et al., 1995). Thus the use of area measures as surrogates for individual SEP should be interpreted with caution.

Wise studied child mortality in Boston between 1972 and 1979. Given the limited data provided on the death certificate (basic demographic details only), Wise linked the child who died to their previous area of residence (one of 146 census blocks in Boston). Socioeconomic position was estimated from the median family income in that census block and income groupings were divided into tertiles. In the 1–19 age group, the lowest income group had an increased risk of dying of all causes when compared to the highest income group, with an odds ratio of 1.47 (95% CI 1.25-1.94). In the 'race' analysis of this data black children had higher mortality rates in all income groups (except the highest) when compared to white children (numbers not presented) (Wise et al., 1985).

This study illustrates the above point; doubt can arise over whether children who died actually had low income or were misclassified into this group by the use of their area of residence. Additional issues in this study are the lack of precision around the income, for example there is equivalisation and there doesn't appear to be any CPI adjustment.

2.4 Time Trend Studies

There are only a few studies of childhood mortality inequalities that have monitored the level of inequalities over time. Different methodology has been employed for almost all of them, making comparisons difficult. However given the paucity of data in this area and the goal of this thesis being to review changes over time all available studies have been included.

Time trend studies that use area level measures as a proxy for individual SEP have the additional problem that trends may not be interpretable because the degree of misclassification of SEP may change over time. This will occur for

example, if progressive socioeconomic residential segregation occurs over the time, meaning that heterogeneity of individual SEP in an area declines as 'poor' neighbourhoods have more poor people and 'rich' neighbourhoods have more rich people. Even if mortality differences by parental SEP remain the same the progressive decrease in misclassification by area will make it appear as if socioeconomic inequalities have increased over time. It is also important to consider that area measures may have external boundary changes over time, also making comparisons difficult.

2.4.1 Theory

The theory behind the determinants of change in inequalities is not well developed. This is probably because social epidemiology theory, in general, is not robust as was seen in section 1.1. Despite there being a number of studies describing trends frequently the reasons for the changes are not scrutinized.

One theory proposed is the idea that decline in mortality is differential by SEP, resulting in increased relative inequalities over time (Roberts & Power, 1996). One process that could explain this is the inverse equity hypothesis. This hypothesis suggests that public health messages are slower to penetrate poorer groups in society compared to the wealthier sections. This means that any intervention will result in an increase in relative inequalities initially, followed by a decline as the message penetrates through different socioeconomic groups (Victora et al., 2000). This theory is backed by the behavioural theory diffusion of innovations (Victora et al., 2003).

Other theories around trend include the structural hypothesis, that changes in the broader structure of society (e.g. economic recessions, increases in poverty and social inequality) will enact changes in health and mortality inequalities (this

is closely related to the political economy of health discussion in section 1.1) (Bremberg, 2003; DiLiberti, 2000). Presumably changes in structure of society would then filter through the (unknown) pathways to inequality in mortality. This is difficult to prove given the multitude of other influences such as institutions, which are thought to modify the influence of structural changes (Bremberg, 2003)

2.4.2 Time Trend Studies- The Repeated Snapshot Approach

The repeated snapshot approach involves simply comparing a population at two (or more) points in time and monitoring the inequalities.

Botting, in her report on child mortality inequalities that was discussed previously, presented data from the previous decennial supplement as a comparison to the current mortality rates. These data from the 1980 and 1990 UK unlinked censuses and mortality statistics show a slight increase in relative inequalities, despite falling overall mortality. The rate ratio between social classes 5 and 1 increased from 2.04 to 2.33, for all-cause mortality in 1-15 year olds for that time period (no confidence intervals are available) (Botting, 1997). There is variation of the changes in rate ratios by age and sex. These findings suggest that absolute differences between the social class 1 and 5 have been preserved as mortality has declined.

Botting also reports linked data from the ONS Longitudinal Study, which show an increase in relative inequalities from 2.82 in 1982-86 to 3.29 in 1987-91 for 0-14 year old children (Botting, 1997). This was comparing the same classes as the above census data and again no confidence intervals are available. The large number of deaths in the <1 age group, who were included in this study, may drive the overall changes seen, as they tend to be more strongly patterned

by SEP than older age groups. This study supports the concept of differential decline in mortality by SEP as no decline in mortality rates were seen in social class 5, although all other groups had a reduction.

Turrell and Mathers conducted a study that compared inequalities over two time periods in Australia, 1985-87 and 1995-97. They analysed data for all ages, however the results that are discussed here pertain to children aged 0-14 only. Turrell and Mathers measured SEP used an area based deprivation measure, graded and grouped into quintiles. The outcome of interest was inequality, which was measured in three different ways: rate ratios, Gini coefficients measuring mortality inequality across the quintiles of deprivation (if all quintiles of deprivation had the same mortality risk the Gini coefficient would be 0), and population attributable risk (%) (i.e. mortality that would be avoided if all quintiles had the same mortality rates as the least deprived quintile).

Turrell and Mathers showed that while age standardised mortality rates declined in all deprivation quintiles between 1985 and 1995, there was variation in what happened to inequality. In all-cause mortality 0-14 for males relative inequality increased over time as did the Gini coefficient of population inequality, and the population attributable risk. For females of the same age, all measures of relative inequality decreased, as did the Gini coefficient and population attributable risk. However when looking at specific causes of death there were some differences and some similarities among the sexes for example relative inequality of SIDS increased while that of perinatal conditions declined (Turrell & Mathers, 2001).

This study highlights the complexities of assessing inequalities (as well as the difficulty of interpreting area based trends). Multiple measurements must be used, as presenting only one could give a deceptive result. However it is difficult to interpret all the measures and to determine what they denote for interventions. It is also important to realise that while the change in inequalities

may appear small, on a population level they may translate to large absolute change.

The best study in this area is undoubtedly by Ostberg. This study compared inequality in child mortality between two linked cohort periods of 1961-66 and 1981-86 in Sweden (Ostberg, 1992). The methodology of the main analysis (1981-86) has been commented on previously (see section 2.2), however to ensure comparability between the two time periods time there were minor changes to the later cohort for this time trend analysis.

The most striking feature in this study was the overall decline in childhood mortality during that time period, for example the mortality rates for sons of manual workers dropped from 68.1/100 000 to 33.7/100 000 over the time period under study. While social class differences were present in both time periods, when comparing the children of manual workers to all others, whether they had changed is not entirely clear. There was a small widening of class differentials in girls (RR 1.09 (95% CI 1.0-1.2) to 1.25 (95% CI 1.1-1.4)) driven by an increase in the excess death rate among the 5-9 year old daughters of manual workers (RR 1.06 (95% CI 0.9-1.3) - 1.70 (95% CI 1.2-2.3)). For boys aged 1-19 there was a preservation of the relative gradients as overall mortality declined i.e. absolute differences between the groups also declined. However the age specific trends were different, with an increase in relative inequality in 5-9 year old boys (RR 1.26 (95% CI 1.1-1.5) - 1.64 (95% CI 1.3-2.1)). Conversely there may be a diminution of the gap in 1-4 and 10-14 year olds boys (Ostberg, 1992). It is difficult to know if these are true changes or represent statistical instability.

There are a few minor differences in the sampling frames of this study due to differences in the collection of census data, however they are unlikely to have affected the results. Children excluded from the analysis because no parent was in the labour force had the highest mortality, however unless this group

striking difference in mortality risk and there was differential selection bias over the time periods, it is unlikely to have dramatically affected the results.

Roberts and Power looked specifically at time trends in injury mortality using unlinked data comparing 1980 and 1990. Over that period the relative risk of injury mortality for boys and girls aged 0-14 increased from 2.1-2.6 comparing RG class 1-3NM and class 3M-5 (Roberts & Power, 1996). However these again do not account for the changing social class group size and thus should be interpreted with caution. A similar analysis of injury mortality in Scotland between 1981 and 1995 appeared to show a slight reduction in absolute inequalities (the rate difference declined from 7.1/100 000 in 1981 to 5.1/100 000 in 1995) and a non-significant increase in relative inequalities (from 1.7 in 1981 to 1.9 in 1995) (Morrison et al., 1999a). However this was using an area based measure of SEP, thus being susceptible to misclassification

2.4.3 Time Trend Studies- The Continuous Approach

There are only two studies that have looked at continuous time trend data and both are ecological.

DiLiberti aimed to evaluate the hypothesis that changes in social stratification between 1960s and 1990s in the USA increased the burden of child mortality attributable to social stratification. He operationalised the complex prestige based concept of social stratification using a resource measure, the proportion of households in a county with access to a telephone over the 30-year period (DiLiberti, 2000). Counties were grouped into quintiles depending on the proportion of individuals with a telephone.

Results of the population attributable risk (PAR) and relative risks showed a decline in the deaths attributable to poverty from 1960s to 1980s and then an increase from the mid 1980s until the study ended in 1992, however it is clear that this is due to differential mortality decline among the deprivation quintiles(DiLiberti, 2000). The least deprived (reference) quintile continuing to enjoy substantial declines in mortality while all other quintiles had convergence and stabilization of their mortality rates, thus making the PAR increase.

This study had poor internal validity possible selection bias (3 states were excluded from the analysis), residual confounding from ethnic group (there was no standardisation over time of the different ethnic groups) and possible differential misclassification of exposure over time due to increasing neighborhood segregation.

Interestingly to prove his original hypothesis the author cross-correlated the proportion of children living in poverty with the proportion of deaths attributable to social stratification. There was a high correlation (0.96 $p<0.001$) if a 9-year lag was built into the model (DiLiberti, 2000). The theoretical basis of the discussion about the time lag between poverty and child mortality is dubious. The authors hypothesise that increasing poverty reduces the rate of accumulation of health promotive assets, eventually slowing the decline in mortality rates (DiLiberti, 2000). However it is difficult to imagine that this decline in health promotive assets occurs for every quintile of the population except the top quintile.

Bremberg studied a period of recession in Sweden in the early 1990s and tried to evaluate the effects of this recession on child health and mortality inequalities. The recession period (1990-93) was contrasted with preceding and subsequent years (Bremberg, 2003).

The exposure in this study is an area deprivation measure and the outcome population inequality, measured by relative index of inequality (RII). The results of this study show an overall decline in mortality, from 56.3-33.3/100 000 between 1988 and 1995 and no change (i.e. a non-significant decline) in the RII of mortality by area deprivation over the same time period. Morbidity RIIs showed no clear patterns of change. Bremberg interprets the lack of change in inequalities as reflecting on the relative strength of social institutions such as education and social insurance (Bremberg, 2003).

There are a number of methodological issues that make the internal validity of this study concerning as well. The exposure measure was constructed using data from the mid point of the study, when recession effects would be experienced and there was a change in the geographical composition on the areas being measured mid way through the study, meaning that comparisons over time are different. These problems mean that there may be misclassification of deaths into quintiles that had changed either in deprivation or area over time.

Possibly the key problem with this study is that while the effects of the recession on income were maximal in 1996, as income transfers had protected individuals until then, the mortality data ends in 1995. This limits conclusions about possible delayed effects of the recession mediated by the drop in income.

2.5 Age Differences

While there is evidence that inequalities are a persistent feature across most of life, the level seems to vary at different periods in the life course. Infant mortality has been thought to be strongly patterned by SEP (Blakely et al., 2003; Schuman, 1998), while there is debate about whether during adolescence there

is a relative flattening of inequalities (Blane et al., 1994; Macintyre & West, 1991; West, 1988, 1997).

West has been particularly interested in the period of 'youth' defined as 10-19 year olds. Evidence has been presented from the decennial supplements in two time periods (1960 and 1980 census) to illustrate the flattening of social class gradients in these ages. West hypothesised that any small gradients that did exist were due to injury mortality, rather than medical mortality (West, 1988). He suggested that the explanation for these findings were that youth specific conditions at this stage in the life course cut across social class differentials to equalise mortality. This effect would weaken in older ages as adult occupational influences begin to dominate groups (West, 1997).

However there has been considerable debate about this concept, with disagreement about the interpretation of the results and speculation that this may be an artefact of the low death rates in this age group and the difficulty classifying the SEP of an adolescent due to the transition from parental SEP to own attained SEP (Blane et al., 1994).

In studies reviewed here that confirmed the existence of a socioeconomic gradient of mortality during childhood, and where age stratified results were available there was a range of rate ratios seen (see Table 4).

Table 4 Age specific gradients for all-cause mortality

Authors	Country	Variable	Year Studied	Rate Ratio (95% CI)#		
				1-4	5-9	10-14
Blakely et al	New Zealand	Occupational Class- NZSEI	1991-94	2.1 (1.3-3.3)	1.6 (1.0-2.6)	1.9 (1.5-2.5)
Botting	UK	Occupational Class- RG	1991-93	2.63 (N/A)	2.14 (N/A)	1.71 (N/A)
Judge and Benzeval	UK	Occupational Class- Modified RG	1978-80, 82-83	2.58 (1.75-2.49)	2.56 (1.98-3.30)	4.14 (3.43-4.99)
Nelson	USA	Welfare recipients	1985-88	2.9 (N/A)	2.5 (N/A)	2.3 (N/A)
Neserian	USA	Welfare recipients	1976-1980	3.22 (2.70-3.84)	3.15 (2.50-3.98)	
Ostberg*	Sweden	Occupational Class- Swedish	1981-86	1.37 (0.8-2.3)	1.89 (1.4-2.6)	1.62 (1.2-2.2)
Singh and Yu	USA	Income	1979-85	3 (1.39-8.05) (<$10 000/pa)	1.62 (1.08-2.14) (<$20 000/pa)	
Vagero and Ostberg	Sweden	Occupational Class- Swedish	1961-79	1.43(1.1-1.8)	1.42 (1.2-1.7)	1.32 (1.1-1.5)

Rate ratios are highest/lowest of variable under study
* Boys only

While there are difficulties comparing these studies directly (see section 1.1 in this chapter for a full discussion of this), there seems to be evidence of variation in the level of inequalities by age, although many of the confidence intervals overlap. Some of the studies support West's theory, but others do not. What this does tell us is that the level of inequality is not immutable.

2.6 Gender Differences in Gradients

Consistent differences are seen in the risk of mortality by sex, with boys having higher mortality rates than girls (regardless of SEP) in the majority of studies that reported by sex (Blakely et al., 2003; Botting, 1997; Ostberg, 1992; Vagero & Ostberg, 1989). Interestingly the only study that did not show differences in child mortality rates by sex was the Finnish study (which showed very little

inequality by occupational class), although in the older ages studied (15-35) males had higher mortality (Pensola & Valkonen, 2000).

There are a number of explanatory frameworks to look at gender differences in health. One framework uses the following levels of analysis; biological factors, acquired risks relating to different exposures and/or behaviours, reporting differences and access to healthcare (Emslie et al., 1999). Most of the excess male mortality in childhood seems to be due to mortality causes that relate to injury, either intentional or unintentional (at least in New Zealand) (Blakely et al., 2003). However there is some evidence of a gender bias in utero with female foetuses less likely to miscarry than male (Sen, 1998). It is difficult to disentangle the effects of biology and sociology in gender differences. However differences in mortality by sex are likely to be due to a combination (to a greater or lesser degree) of biology and sociologically circumscribed gender roles, which act to either expose boys to more risk or protect girls from risk (or both).

2.7 Cause of Death Contributing to Gradients

The explanation for gradients observed in all-cause mortality, must be consistent with the gradients seen in the underlying causes of mortality in children. The most common causes of death in the developed countries in the 1-14 year old age group internationally are unintentional injury, cancer and congenital causes. The same pattern in seen in New Zealand (Ministry of Health, 1998). Evidence of gradients in specific causes will be discussed here. New Zealand literature will be discussed later.

2.7.1 Unintentional Injury

Unintentional injury mortality is made up of a number of different groupings, which include road traffic accidents, pedestrian mortality, cyclist deaths, fire deaths, drowning and falls. The literature reviewed above supports the hypothesis that unintentional injury mortality is patterned by socioeconomic variables (Blakely et al., 2003; Nelson, 1992; Nersesian et al., 1985; Ostberg, 1992; Vagero & Ostberg, 1989). Other studies looking specifically at injury mortality in children support these findings (Hjern & Bremberg, 2002; Laflamme & Diderichsen, 2000; Roberts et al., 1998; Roberts & Power, 1996).

There is some suggestion that different causes of injury mortality have different socioeconomic gradients in mortality associated with them, e.g. in 1992 British children in social class 5 were 14 times more likely to die in a fire than children in social class 1 and 4 times more likely to die in a motor vehicle crash (Roberts & Power, 1996).

2.7.2 Cancer

Cancer mortality showed socioeconomic gradients in a number of studies (Blakely et al., 2003; Nelson, 1992; Ostberg, 1992), however the pathways to this are more complicated. The main causes of child cancer mortality, leukaemia and brain tumours, are different etiologically, however none of studies looked at cause specific mortality, probably due to small numbers. The two processes to consider that will create differential mortality by SEP are whether there is differential incidence and/or case fatality of cancer by SEP.

Looking briefly at acute lymphocytic leukaemia (ALL) there is inconsistent evidence around the role of parental SEP and ALL incidence (Dockerty et al., 2001; Murray et al., 2002; Schillinger et al., 1999; Swensen et al., 1997). There is little evidence around differential survival by SEP, however one large UK study showed no relationship between cancer survival and parental social position (Schillinger et al., 1999).

Data on the incidence and case fatality of other common childhood cancers such as CNS tumours is even less common. It is suggested that there is an association between decreasing parental socio-economic position and incidence of neuroblastoma (Davis et al., 1987; Menegaux et al., 2004), but not of other solid tumours (McNally et al., 2003).

2.7.3 Congenital Anomaly

Congenital anomaly was also highlighted as a possible cause of socioeconomic inequalities in the studies reviewed (Blakely et al., 2003; Nelson, 1992; Nersesian et al., 1985). Again the issue of differential incidence/case fatality is unresolved. Increasing deprivation has been associated with increasing incidence for some anomalies such as neural tube defects (Wasserman et al., 1998), and specific types of cardiac malformations (Vrijheid et al., 2000). The aetiology of this is not entirely clear, but differential environmental exposures may be an influence, this includes folic acid (de Walle et al., 1998) and possibly living near hazardous waste landfill sites (Dolk et al., 1998; Vrijheid et al., 2002). Whether the increased incidence explains the mortality gradients or whether there is differential survival is not clear.

3 New Zealand: Children in Times of Change

This section will look at the societal changes that have occurred over the period of study, focusing specifically on children and then also at any New Zealand evidence of mortality gradients. Unfortunately it is not possible to adequately cover socioeconomic gradients and time trends in the morbidity of New Zealand children in this thesis. Although time trend data are limited, there is some evidence that the health of New Zealand children may have deteriorated between 1981 and 1999, with an increase in avoidable hospital admissions, including infectious diseases (Dharmalingam et al., 2004; D'Souza & Wood, 2003; Mills et al., 2002). Concern was raised during period of structural reforms that children, and their health, would be unduly affected by any policy reforms (Boston & Dalziel, 1992; Fancourt, 1997; Pearce, 1994). Subsequent reports by government departments and NGOs continue to highlight this concern (Blaiklock et al., 2002), however a comprehensive evaluation of changes over time of the health of New Zealand children has not been undertaken.

3.1 Structural changes in New Zealand 1981-1999

It is not within the scope of this thesis to provide a detailed evaluation of the policy changes that impacted on children from 1981-99. However it is clearly necessary to place any changes in mortality inequalities in the social context in which they occurred. Table 5 provides an overview of the social and economic policy changes that took place in New Zealand in the 1980s and 1990s. These reforms took place under successive Labour, National, and coalition governments. Coalition governments were formed after the advent of the

proportional representation electoral system in 1996. The main themes of change were similar to those that took place in most OECD countries in the same time period (Navarro, 1998). They included a move from a protected economy with a mainly universal welfare system, to a free market economy with a residualist welfare state, followed by the repeal of many of the welfare reforms but continued neo-liberal economic policies (Boston et al., 1999; Boston & Dalziel, 1992).

These policies, in combination with other demographic and social changes, resulted in a transformed environment for children (see Table 6). While not all of these changes impacted negatively, for example there are now more Māori children involved in Māori language based schooling, there are significant concerns about other changes such as the increase of the proportion of children in low-income households. The reforms differentially affected groups of people, favouring those already better off, pakeha and families with two parents (Blaiklock et al., 2002).

Table 5 Major economic and social policy changes in New Zealand 1981-1999.

Area	1981	1986	1991	1996
Electoral System and Governance	FPP- National government 1981-84	FPP- Labour government 1984-1990	FPP National government 1990-1996	Proportional Representation System (Mixed Member Proportion) National and NZ First 1996-1999
Economic Policy and Employment policy	Keynesian/ state interventionalist economy	After 1984-progressive deregulation of the economy • Floating of NZ dollar • Increase in indirect taxation eg GST • Deregulation of industry • Flattening of income tax rates with reduction of highest tax rates • Selling of state assets and corporatising government departments	• Recession 1991 • Employment Contracts Act 1991 Eliminated compulsory unionism Decentralised wage bargaining Individual contracts not collective agreements	
Education	• Free, universal education • Compulsory until age 15 • Student allowances for those in tertiary education • Increasing trend of mainstreaming children with disabilities into conventional state schools	Education Act 1989: "Tomorrow's Schools" • Separation of education policy and provision • Control transferred to school boards • National standards • Consumer choice concept • Tertiary education fees set at $1300 annually	• Abolition of "zoning" to increase consumer choice • Quasi market • Increase in tertiary fees to a percentage of cost • Tertiary student loans scheme instigated • Tertiary student allowances means tested School leaving age increased to 16 in 1993	

Area	1981	1986	1991	1996
Health Services	• 29 Hospital Boards-locally run • Funded by Department of Health • Free secondary and tertiary care • Primary care on a fee-for service basis • Pharmaceutical co-payments started in 1885 • From 1983 move to Area Health Boards with elected and appointed members, increased accountability and national targets • By 1989 14 Area Health Boards established over entire country • However there were concerns with o Increasing costs o Increasing waiting lists		1993 Health Reforms • Split of purchaser/provider roles into 4 Regional Health Authorities (purchasers) and 23 Crown Health Enterprises (providers-hospitals) • CHEs were "for profit" organizations, with competition in service provision. • Devolution of service provision to NGOs such as iwi • Part charges established in hospitals • Co-payments for primary care continue to rise • Targeted assistance for primary care for low income/high need groups through community services cards and high user cards • PHARMAC established to contain ballooning government pharmaceutical costs	• Move back to 1 purchaser instead of 4 • Hospitals no longer for profit • Move away from competitive contracting for services • 1997- removal of part charges in hospitals • Free primary care and pharmaceuticals for under 6s
Housing	Government provision of housing through: • Subsidised rent and loans (income related) • Accommodation benefits • Direct provision of housing via Housing Corporation • Later in decade more targeted approach to those with "serious housing needs"		• Move out of supply of housing to supplementing income via accommodation supplements • Housing Corp to be managed on a commercial basis • Sale of public housing stock and mortgages • Market rental prices	• Continuing sale of public housing stock • Increase in value of accommodation supplement
Welfare	• Mix of residualist and universal system but overall most social expenditure not means tested in early 1980s • Universal child benefit	• Increase in targeted benefits e.g. means testing on student allowances, surcharge on national super	• Move to residualist or needs based model with multiple targeted benefits and supplements • Benefits cut by up to 25% in 1991 (including benefits to single parents) • Family benefits abolished • Income support targeted, but overall amount decreased	

Source: (Blaiklock et al., 2002; Boston et al., 1999; Boston & Dalziel, 1992; Devlin et al., 2001)

Table 6 Changes in the social determinants of child health in New Zealand during the 1980s and 1990s.

Social Determinant	Changes
Household Income (absolute)	Between 1982 and 1996 people in the highest income households have become better of, while those in middle or low-income households have either become slightly worse off or have remained the same (Statistics New Zealand, 1999b). Households with children are more likely to be in the lowest two quintiles of income and had equivalent incomes that were 87% of mean income in 1988 and 86% in 1998 (Mowbray, 2001). Sole parent households have the lowest income compared to other households with children (Statistics New Zealand, 1999b). 67% of single parent households are dependent on means tested benefits as their main source of income (Ministry of Social Development, 2002), and are therefore the highest proportion of households in poverty (Ministry of Social Development, 2001)
Poverty	In 1987/88 16% of children lived in houses with an equivalised income less than 60% of median, net of housing costs. This increased to 35% in 1991/92 and later declined to 29% in 1997/98 (Ministry of Social Development, 2001)
Income Inequality	Rise in household equivalised disposable income Gini coefficient from 0.259 in 1982 to 0.322 in 1996 reflecting rising income inequality between households (Statistics New Zealand, 1999b)
Family Composition	Sole parent households have increased from 62 514 in 1981 to 114 957 in 1996 census (Statistics New Zealand, 1999b). From 1986-1996 the proportion of children living in sole parent households increased from 15.7% to 23.6% (189 900 children) (Statistics New Zealand, 1999a).
Employment	In 1986 13.7% of children had no parent(s) in the paid workforce. This had increased to 23.4% by 1996 (Statistics New Zealand, 1999a).
Housing	The proportion of children living in homes that are owned by their parents fell between 1986 and 1996, from 52% to 44% in single parent households and 78% to 75% in two parent households (Blaiklock et al., 2002). Crowding seems to have increased, particularly for Pacific children in Auckland (Child Poverty Action Group, 2003).
Living Standards	No time trend data available but in 2000 average living standard scores were lower for families with children than the rest of NZ and sole parent households had much lower living standards than two parent families with 28% compared to 7% being in the two lowest living standard categories. 50% of families in the most restricted living standards category reported being unable to afford to send their children on school trips and 38% report being unable to afford school supplies (Ministry of Social Development, 2002).
Education	In 1980 there were 55 992 children enrolled in early childhood education, by 1990 this had increased to 228 440 and in 1996 there were 303 259 children enrolled in early childhood education (Ministry of Education, 2004). School retention rates have increased over time (for both Māori and non-Māori children) (Blaiklock et al., 2002). There has been a decrease in the number of children leaving school without qualifications (Blaiklock et al., 2002).
Cultural issues	In 1973 only 18% of Māori spoke te reo Māori, however by 2003 14.8% 0-4 year olds and 22.7% 5-14 year old Māori children spoke te reo Māori. In addition between 1992-1996 the proportion of Māori children receiving at least 12% of their education in Māori medium increased from 12.7 % to 16.8% (Ministry of Social Development, 2003).

3.2 New Zealand Child Mortality

This section aims to use routinely collected and published data to examine trends in child mortality in New Zealand. Data specifically pertaining to socioeconomic gradients are sparse (until recently), which may reflect the dominant paradigm of epidemiological research. While there are studies that are concerned with individual causes of death, there are few that encompass time or all causes.

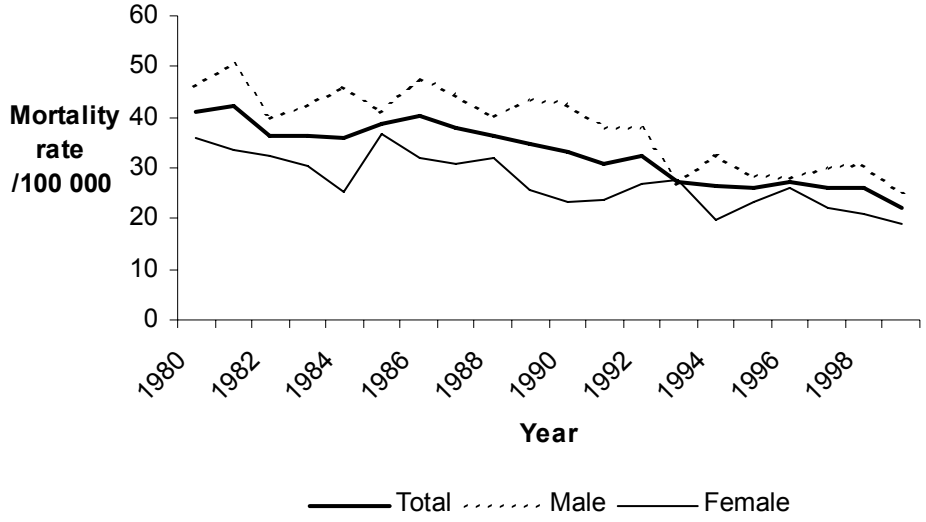
3.2.1 Child Mortality Trends

Routinely collected data on the number of child deaths in New Zealand is available. Figure 7 illustrates trends in child mortality between 1980 and 1999, which is the period that the NZCMS cohorts cover. This demonstrates the improvements that have been made in child mortality in New Zealand over this time period. Despite this there has been considerable concern that New Zealand has waned in the OECD rankings of child mortality rates. In the 1940s New Zealand was heralded as having one of the lowest infant mortality rates in the world (Titmuss, 2001). However New Zealand has slipped from 6th in 1960 in 0-4 year old mortality to 15th in 1995 due to a slower rate of decline in mortality compared to other countries (Ministry of Health, 1998). League tables of cause specific death reveal that New Zealand performs poorly in injury mortality and maltreatment deaths (UNICEF, 2001, 2003).

It is worth noting also that most child mortality is avoidable. Calculations from New Zealand estimate that 65% of mortality in the 1-14 age group is avoidable. This equated to 139 out of 216 deaths in the 1996/97 period. The majority of the avoidable mortality in this age group is due to accidents and most of the deaths

that could have been avoided occur in the most deprived deciles of the population (Tobias & Jackson, 2001).

Figure 7 All-cause mortality per 100 000 by sex aged 1-14 years 1980-1999



Source: Statistics New Zealand and NZHIS

3.2.2 Socioeconomic inequalities in New Zealand child mortality

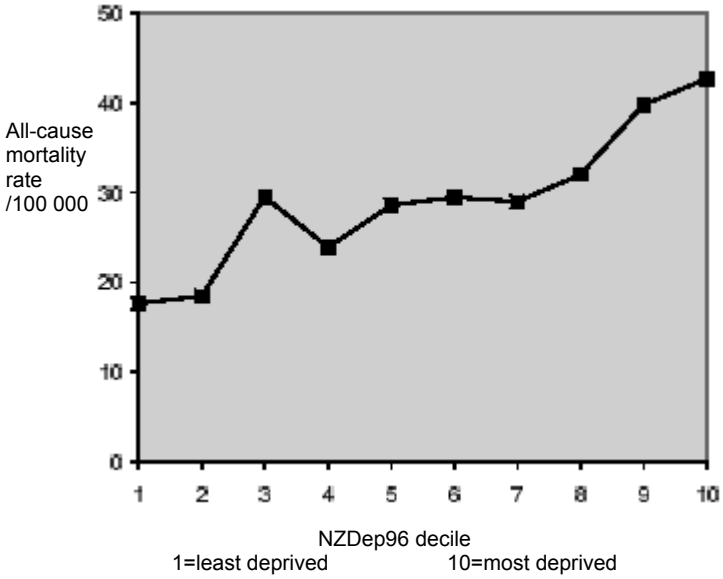
3.2.2.1 All-cause mortality

There has been some data available on inequalities in child mortality in New Zealand. As discussed previously, Blakely et al looked at socioeconomic inequalities in child mortality between 1991 and 1994 and found a relationship between increasing deprivation (measured by multiple measures of socioeconomic position) and increasing risk of mortality (Blakely et al., 2003). An increased risk of death was seen in children ages 0-14, with an equivalised income of less than $10 000, for all-cause and injury mortality. An increased risk ratio was also seen in cancer, congenital mortality and other mortality in the 0-

14 age group and SIDS in the under 1s. However in these latter causes of death all had confidence intervals that included 1.

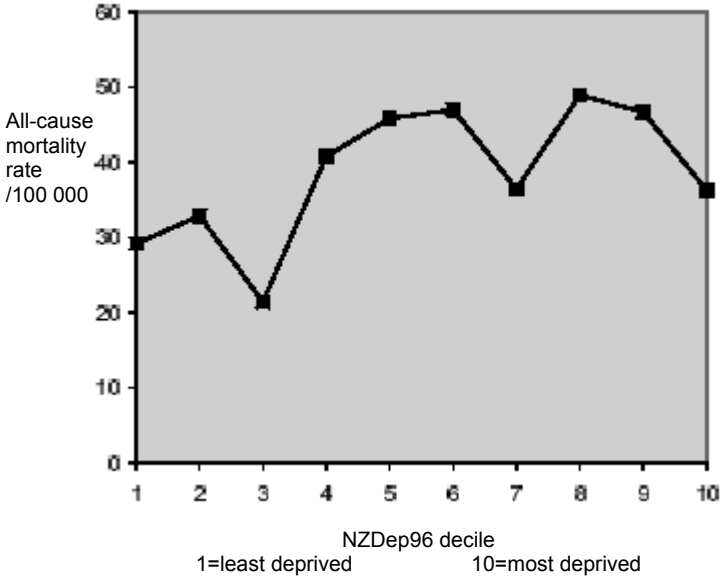
Ecologic studies of mortality between children aged 1-14 in 1996-97 using area deprivation as the socioeconomic exposure show similar trends to the above findings. There is evidence that increasing deprivation is associated with increased mortality in 1-14 year old children, however this varies by gender and ethnic group (see Figure 8 and Figure 9) (Salmond & Crampton, 2000).

Figure 8 Male all-cause mortality/100 000 by area deprivation ages 1-14 years 1996-97.



Source: Ministry of Health 1999

Figure 9 Female all-cause mortality/100 000 by area deprivation ages 1-14 years 1996-97.



Source: Ministry of Health 1999

3.2.2.2 Unintentional Injury

Unintentional injury is made up of a number of different mortality causes. Between 1990 and 1998 the main causes of mortality due to unintentional injury in children aged 0-14 (in order of frequency) were occupant in motor vehicle fatality, suffocation (this occurs mainly in the under 1 age group), drowning, pedestrian death and fire deaths (Kypri et al., 2000). New Zealand has relatively high unintentional injury death rates for children in comparison with other countries (UNICEF, 2001). However when these overall rates are broken down into specific causes it is evident that while New Zealand has much higher rates per 100 000 children for pedestrian and occupant road deaths and drowning compared to Australia, USA and UK, while rates of fire deaths are similar to, or less than, these countries (Langley, 2001). Data on socioeconomic gradients within each injury mortality type are scare.

An unpublished thesis that used the NZCMS data from 1991-1994 estimated the independent effect of income on child injury mortality. Thirty percent of injury

mortality was attributable to income and comparing the lowest to highest income group gave an odds ratio of 1.83 (95%CI 1.02-3.28) for injury mortality. However modelling a scenario of eradicating child poverty only reduced injury mortality by 3-7%, highlighting the importance of other factors in injury mortality (D'Souza, 2004).

Motor vehicle fatalities are the commonest cause of death among children (IPRU, 2003a), and mortality rates of children aged 0-9 who die in motor vehicle accidents have not declined since 1980 (Ministry of Health, 2002a). Local research suggests that there may be a socioeconomic gradient associated with who is likely to be involved in vehicle incidents, with adult drivers of vehicles involved in crashes (but not fatalities) being more likely to have no educational qualifications and low occupational class (Whitlock et al., 2003). International research indicates that children who are less than15 who are driving a car and are injured are more likely to be of low occupational class (Hasselberg et al., 2001). However the majority of children between 1-14 would be the occupants of motor vehicles that crashed rather than the drivers and there is little, if any, research on socioeconomic gradients in motor vehicle occupant deaths and the risk factors associated with this.

Drowning remains a common cause of death in New Zealand children, particularly in the 1-4 age group which had a rate of 5.1/100 000 person years between 1986 and 1995 (IPRU, 2003b; Kypri et al., 2000). There have not been substantial declines in child mortality from drowning since 1990 (IPRU, 2003c; Ministry of Health, 2002a). There is little, if any, evidence about the existence of socioeconomic gradients in this particular cause of mortality. However given that 50% of the deaths from drowning occur in private pools (Kypri et al., 2000), and that the environmental intervention to prevent drowning (compulsory pool fencing regulations) is inconsistently adhered to (Morrison et al., 1999b), this may be worthy of investigation.

Child pedestrian death in New Zealand has been declining over time (Ministry of Health, 2002a), although there is some evidence that the rate of decline may be mediated by traffic volume (Roberts et al., 1992). However it remains a common cause of child mortality, particularly among the 0-9 age group (IPRU, 2003b). The body of work on child pedestrian injury in New Zealand is supportive of the hypothesis that child pedestrian injury is graded by socioeconomic status, with children in lower occupational classes having a high chance of injury (Roberts et al., 1995b). There were too few deaths in this case control study to determine if this also applied in death, however it is likely that differential incidence would lead to differential mortality.

Fire deaths over all ages in New Zealand are graded by socioeconomic status measured on an area level. The risk of people in the most deprived decile dying is 5.6 (95% CI 1.9-13) times higher than those in the least deprived decile (Duncanson et al., 2002). Other research has identified that children aged 0-4 are the age that are at much higher risk than older children of dying from thermal injury (Waller & Marshall, 1993).

3.2.2.3 Cancer

Analysis of the socioeconomic trends in cancer incidence and case fatality among children internationally is contradictory as discussed in section 2.7.2. The New Zealand literature is similar. Child cancer fatalities in New Zealand consist of two main types: leukaemia and brain tumours (personal communication New Zealand Health Information Service).

A case-control study of newly diagnosed leukaemia cases in New Zealand 1991-93 showed evidence of higher incidence of leukaemia among those children whose fathers had no occupation (RR 2.0, 95%CI 1.1-4.0) (Dockerty et

al., 1999). However a contemporary record linkage study of incident childhood leukaemia and lymphoma and birth records suggests no association between occupational class and cancer incidence (personal communication John Dockerty). The only other New Zealand data looks at ethnic differences of childhood leukaemia, suggesting there are differences in both incidence, and case fatality, by ethnic group within New Zealand (Dockerty et al., 1996; Ridgway et al., 1991). Data on the incidence and case fatality by socioeconomic position of central nervous system tumours are not available in New Zealand.

3.2.2.4 Congenital

Congenital anomaly is a common cause of mortality in the 1-14 age group in New Zealand, although the rates decline with age (Ministry of Health, 1998). The most common causes of mortality in this group are Neural Tube Defect (NTD) and congenital heart disease (mainly transposition of the Great Arteries and Tetralogy of Fallot) (personal communication Ministry of Health). Similar to cancer the issue of differential incidence/case fatality is unresolved. However difficulty with estimating the true incidence of congenital malformation makes this unlikely to ever be resolved. Spontaneous miscarriage and elective termination mean that children born with congenital malformations reflect prevalence rather than incidence.

There is no evidence of an association between prevalence of NTD and the Registrar General's occupational class in New Zealand, which is consistent with other low prevalence countries (Borman & Cryer, 1993). However a birth cohort started in 1991 did show evidence of an association between decreasing parental occupational class (Elley Irving) and increasing birth defects in children (p trend 0.02). This was not seen by maternal education and was a small cohort that included all birth defects, most of which are not fatal (Tuohy et al., 1993).

3.2.2.5 Other

"Other" is a heterogeneous group of causes of fatalities. Most of these have not been explicitly researched in New Zealand with traditional markers of socioeconomic position. Some causes that have shown an association between measures of socioeconomic position include suicide (Beautrais, 2000, 2001), meningococcal disease (Baker et al., 2000) and asthma mortality (Jackson, 1988).

Summary

This section has shown that there is some evidence of socioeconomic gradients in injury and all-cause mortality in the early 1990s by individual SEP and for all-cause mortality between 1996 and 1997 by area deprivation. This were cross sectional data and trend data examining whether these inequalities are changing over time are do not currently exist. There is also little literature examining the existence of socioeconomic gradients in specific causes (other than injury) and by age and ethnic group.

Chapter 3: Methods

This first section of this chapter looks at the data used in this study, the data source, cohort definition and variables creation. The second section looks at the methods used to perform analysis on this data.

1 Data

As can be seen from the literature review, to be able to examine the socioeconomic gradients in child mortality an extremely large study is needed, due to the (fortunately) rare event of child mortality. In New Zealand this needs to be a total population sample, as anything less may be subject to type 1 and 2 errors. A large record linkage study is the best way to get enough statistical power to assess the outcome of mortality.

1.1 The New Zealand Census Mortality Study

The data used in this thesis is a subset of the New Zealand Census Mortality Study (NZCMS). The NZCMS is a retrospective record linkage study designed to analyse trends in ethnic and socioeconomic disparities in mortality in New Zealand over the decades of the 1980s and 1990s. The process of creation of this study is described in detail elsewhere (Hill et al., 2002), however a short description pertinent to this thesis will be included here.

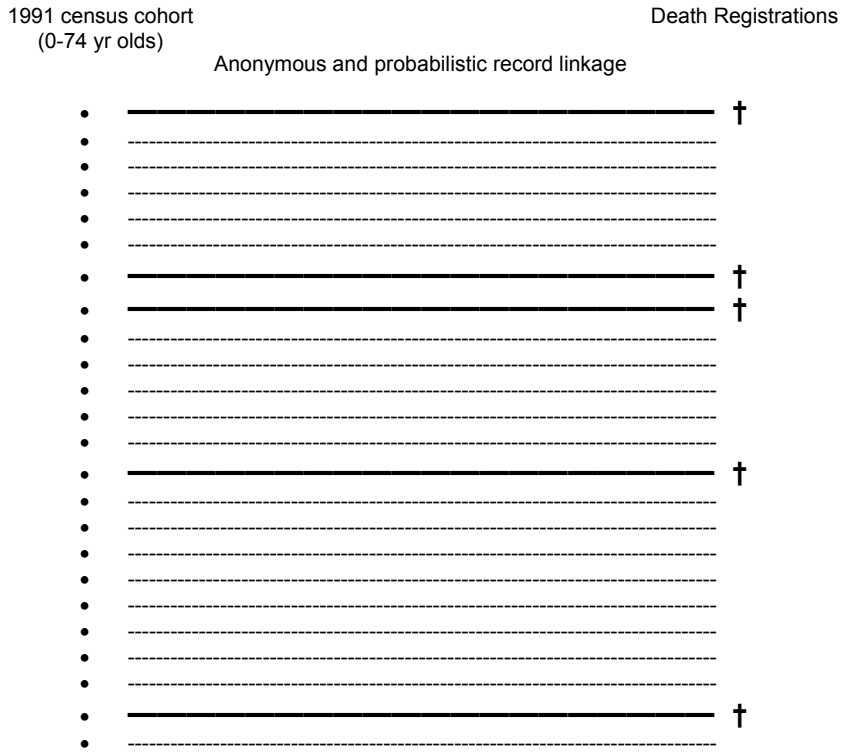
The NZCMS was granted ethics approval from the Wellington Ethics Committee in 1998 (reference 98/7 - Socioeconomic factors and mortality in New Zealand: A probabilistic record linkage study).

1.1.1 NZCMS Cohorts

The NZCMS is four retrospective cohorts, created by linking an individual's death record to his or her census record from the preceding census. All New

Zealanders who completed a census form on census night in 1981, 1986, 1991 and 1996 were in the cohorts. All deaths that occurred in New Zealanders aged 0-74, who were alive on the preceding census night, were eligible to be matched. Thus the follow up time of each cohort was the time between census night and death or the 'end' of that cohort, which was 3 years after the census. Those who did not have a death certificate matched to their census record were considered to be alive at the end of follow up (see Figure 10 for an example of how each cohort was formed).

Figure 10 NZCMS cohort formation



The actual linkage process was done anonymously using the computer software Automatch®. All deaths records of those aged 0-74 on census night were submitted for linkage with all census records of the preceding census. The linkage took place within geographic units, which were assigned from addresses on the death record and census form. The units were either a meshblock (containing about 100 people) or census area unit (about 2000

people). Matching was performed using probabilistic methods (Blakely & Salmond, 2002). Variables that were used for matching were date of birth (day, month and year as separate variables), country of birth, sex and ethnic group. Weighting was assigned to different matching variables, dependent on the probability of the variable creating a true link. Approximately 75% deaths were linked using this process (Hill et al., 2002). It was estimated that of the links created over 96% were true links (Blakely & Salmond, 2002).

Linkage rates for the 0-14 age group were slightly poorer than overall linkage rates (see Table 7). As well as differences in linkage rate between cohorts, there were also differences within strata in each cohort. For example linkage rates ranged from 52% for Pacific females of 0-14 in 1996 to 80% of Pacific males 0-14 in 1981 (Fawcett et al., 2002).

Table 7 Percentage of death records linked in different age groups by cohort

Census	Death Records Linked Ages 0-74(%)	Death Records Linked Ages 0-14 (%)
1981	71%	66%
1986	74%	71%
1991	77%	69%
1996	78%	69%

Source Fawcett et al 2002

A weighting methodology to adjust for any bias that may arise from incomplete linkage is presented in section 1.1.3.

1.1.2 Child Mortality Thesis Cohorts

The children who were eligible to be in the cohorts in this thesis were between ages 0 to14 years on census night (i.e. a maximum age of 14 years and 11 months). However the outcome of interest in this thesis was mortality in those aged 1-14 years. Since the actual analysis was performed at age at follow-up i.e. age at death or age when cohort ceased 3 year after the census, an open-ended cohort needed to be created. This allowed children under 1 on census

night to contribute person-time to the analysis as they aged and therefore moved into the cohort. Children in the older age range also contributed person time before they 'moved out' of the age period of interest.

Additional restrictions were needed to ensure adequate data were available for each child. The children had to be in their usual residence, which was also a private dwelling (e.g. boarding schools were excluded). Absentee records were excluded from the analysis and preparation of variables due to incomplete data. Absentee records were children who were not physically present at their homes on the night of census, but some information had been derived from the household census form. At least one adult over the age of 16, who was also in their usual residence and private dwelling (i.e. not a child minder) was required to be present in the house on census night in order to obtain household level variable information. See Table 8 for the numbers of children in each cohort after these restrictions. The possible effects of these restrictions are discussed in Chapter 4.

Table 8 Restrictions to create cohorts for this thesis.

	Cohort			
	1981	1986	1991	1996
All Children 0-14 years on Census Night	886 425	815 328	806 463	854 376
Children in their usual residence, which was a private dwelling	812 420	757 434	744 714	781 149
Children with a resident adult over age 16 -**Actual Cohort**	811 578	756 468	743 976	779 355

1.1.3 Linkage Weighting

Differences in linkage can be thought of as type of misclassification bias (Rothman & Greenland, 1998). The incomplete linkage process means that there will be a certain proportion of people who have had their outcome misclassified, i.e. they are still classified as alive in the cohorts. If rates of non-

linkage were the same across strata (e.g. age, sex, ethnic group and socioeconomic position) this misclassification would be considered non-differential. This would mean that while standard rates would be underestimated, rate ratios would still be accurate as they are a ratio of two similarly underestimated rates. However, if the misclassification was differential by any factor then some standard rates (but not others) may be underestimated and rate ratios would be inaccurate.

The misclassification was differential, by age, rurality, ethnicity and small area deprivation (Fawcett et al., 2002). The actual reasons for the differential misclassification are not entirely clear. Errors in data handling and some residential mobility are the most likely causes.

To eliminate the effect of the linkage bias, weights were created so that absolute mortality rates, rate ratios and rate differences by demographic and socioeconomic factors should be little affected by any bias. Details of the method used are described elsewhere (Fawcett et al., 2002), but briefly: weights were assigned to individuals in specific strata to make up the difference between the total *linked* deaths and the total *known* deaths in those strata. The strata were sex, 5-year age bands, ethnicity, cause of death and area deprivation. For example if 20 out of 30 children in one strata were linked then each linked record would have a weight of 1.5 assigned to it (30/20).

The weights performed well for adults but had not been fully investigated for children. There was the potential for them to become unstable for fine strata of children (such as cause of death, 5 year age bands and ethnic group) because of the small numbers in each stratum. This instability would have shown up as variation between the ‘known’ number of deaths in each category and the weighted numbers.

This problem was indeed the case when it was investigated. Details of the process of creating a new weighting variable specifically for children (and this thesis) and for the causes of death that are common in children can be seen Appendix 1.

1.2 Data Used in this Study

1.2.1 Exposure Measurement

While a large number of socioeconomic variables were already available on the cohorts, specific variables had to be created for children. The New Zealand census requires each individual to complete a form (or in the case of children have it completed for them) and a separate form to be completed for each household. This allows us to identify and group the individuals in households for the creation of socioeconomic variables for children. The creation of each specific variable is described in the following sections. Once variables were formed this information was then attached to every child's census record. Children were retained in the dataset even if we were unable to create a variable for them. They were given a 'missing' value for that variable and separate analysis was performed, where useful, for these children.

In the creation of variables household information from children under the age of one was included as they contributed person time to the analysis. Restricting out those that died before age one did not make a difference to the distribution of variables.

1.2.1.1 Income

Household equivalised income was used as the income exposure measurement. The equivalisation process ensures comparability between different size households. If income was not recorded on a census form or if there was an adult missing from the household on census night, then a household equivalised income was unable to be calculated and was coded as missing. While the mortality rates, rate ratios and rate differences can be looked at for the group of children with no household income the regression-based indices could not utilise this data.

It took several steps to calculate the equivalised real household income for each child. First, the total personal income of each adult in the household was required. This was collected in bin-categories for each of the four censuses. For the 1986,1991 and 1996 censuses, income was collected as annual gross (before personal tax) income from all sources, including benefit support, for the financial year ended 31st March in the year of the census. In 1981 income from benefits (tax free in 1981) was collected separately to income from wages and salaries (taxed) and combined to produce total personal annual income.

Given that individual incomes were in categories of income, we first assigned each individual the median household income for the same income band from the corresponding New Zealand Household Economic Survey for the latter three censuses and mid-point income (and Pareto estimate for top category) for the 1981 census. Next the personal incomes were summed to give a total household income for as many households as possible. The sum or total household income was then equivalised for household economies of scale using the New Zealand-specific Jensen Index (see Table 9)(Jensen, 1988). The equivalised household income in the 1981, 1986 and 1991 census cohort data

was further adjusted for spending parity, to 1996 dollars, using the consumer price index.

Table 9: The revised Jensen Index

Number of adults	Number of children						
	0	1	2	3	4	5	6
1	0.65	0.91	1.14	1.34	1.52	1.69	1.85
2	1.00	1.21	1.41	1.58	1.75	1.91	2.06
3	1.29	1.47	1.65	1.81	1.96	2.11	2.25
4	1.54	1.71	1.87	2.02	2.16	2.30	2.44

Once this process was complete this household equivalised, CPI adjusted income was attached back to each child in the house. The children were then ranked from lowest to highest income and divided into tertiles for all census cohorts combined. The approach of ranking the individual children, rather than households,+ was preferred, as the equivalisation process already shifts households with more children down in the income ranking. Had we then ranked households this would have resulted in a disproportionately large number of children in the lower tertile. For the majority of analyses in this thesis, these three categories of income were used: low (< $20 600), medium ($20 600 to $33 000) and high (≥$33 000)[2]. These are lower than adult household incomes due to the equivalisation process and because families with children may have less household income due to fewer adults working.

There was some movement in the median and mean incomes in each group between each cohort (see Table 10). Most of the alteration was in the upper income group with an increase in the mean and median income over the time period under study, which is consistent with the increase in income inequality (Statistics New Zealand, 1999b).

[2] Note these are rounded to the nearest $100 for confidentiality reasons

Table 10 Mean and median income across all cohorts

	1981		1986		1991		1996	
	Median Income	Mean Income	Median Income	Mean Income	Median Income	Mean Income	Median Income	Mean Income
Low Income	$14 300	$12 992	$14 900	$14 343	$14 100	$13 576	$13 800	$12 994
Medium Income	$26 500	$26 756	$26 200	$26 447	$26 700	$26 674	$26 800	$27 065
High Income	$43 600	$51 131	$42 400	$47 199	$44 800	$50 925	$47 300	$56 689

The median income is rounded for confidentiality reasons but the mean income is not, as it is a calculated statistic rather than an actual income.

Quintile categories of income were used to calculate the slope and relative indices of income (this method is described in detail in section 1.1).

1.2.1.2 Occupational Class

A similar process to that for income was undertaken to assign occupational class to all children. The occupational class of all adults (over 16 years) in the house was assigned and then the highest was attached to each child in the house.

The assignment of occupational class first requires a valid occupation or job type. In the NZCMS cohorts, occupation has been coded according to at least one of three New Zealand Standard Classifications of Occupation: NZSCO68 (i.e. the 1968 version; all four cohorts), NZSCO90 (1991 and 1996 cohorts) and NZSCO95 (1996 cohort). Occupational class classifications are available for each of these occupational classifications. To ensure maximum comparability of the class classification across the four cohorts, we used the NZSCO68 linked Elley-Irving Classification (Elley & Irving, 1976).

The Elley-Irving is one of two occupational classification schemes in use in New Zealand. It was initially created in 1972, based on the 1966 census and then updated for the 1971 and 1981 census periods (Elley & Irving, 1976) (Johnston,

1983). Each of the 451 male occupations enumerated in the census was classified according to the New Zealand version of the ISCO68 and ranked into 6 groups, based on median income and education within each occupation (Elley & Irving, 1976).

For this thesis the Elley-Irving scale was divided into a dichotomous variable to allow comparison of mortality rates, rate ratios and rate differences. Groups 1-3 were the reference group and group 4-6 the 'low occupational class' group. For the regression-based indices a 4 level classification was created, comprising groups 1-2, 3, 4, and 5-6.

The Elley-Irving scale codes farmers into different levels depending on the type of work they perform. There is some suggestion that children who live on farms may be exposed to different patterns of risk than non-farming children (Ostberg, 1992). It was decided to create a 'flag' for children who had an adult in the household identified as a farm worker, to investigate any possible differences in risk. This flag was used to compare the mortality rates, rate ratios and rate differences in children who had a farmer parent with those who did not. However it is important to note that the flag does not necessarily mean the children live on a farm.

1.2.1.3 Education

Two different education variables were created for each child: highest educational qualification in the house and maternal education.

To determine the highest educational qualification in the household the highest qualification was taken from any adult over the age of 16, and once again attached to each child in the household. The highest educational qualification

was the highest qualification gained since leaving school or, where the respondent had no post-school qualifications, the highest school qualification. Two problems needed to be addressed in the categorisation of the education variable. First, the census instruments for collecting and categorising the educational qualifications were different for each census. Second, maintaining a hierarchy of qualifications from high to low was problematic because of the hierarchical position of some qualifications. In particular determining whether a post-school non-university degree qualification was higher ranked than a higher school qualification was not always possible

In order to ensure that the categorisation of education across the four cohorts was as comparable as possible an inter-censal classification of education, developed by Statistics New Zealand, was used. This classification was further grouped into five and three level groupings to obtain categories of sufficient size for robust analyses (see Table 11). The decision about grouping of qualifications into five and three groups was based on the ideal of maintaining a hierarchy of qualifications from low (none) to high (university degrees) while at the same time maintaining consistency across the four cohorts.

Table 11 Five and three level groupings of highest educational qualification

Five Level Education Grouping		Three Level Education Grouping	
Description	Label	Description	Label
No qualifications	No Qualifications	No qualifications	Low
5th Form School Qualification	School-Low	Any School Qualification	Medium
6th/7th For School Qualification	School-High		
Trade and other post-School	Post-School – Low	Any Post-School Qualification	High
University degree, nursing or teaching diploma or NZCS or Technician's Certificate	Post-School – High		

The three level educational qualification variable was used for comparison of mortality rates, rate ratios and rate differences. For the regression based indices the five level classification was used.

However there was some concern with using a 'highest in the household' classification. These concerns arose mainly because it is not possible to consistently identify exact family relationships in each household. This meant that a non-parent who may have had the highest qualification in the house would be identified in the programming and this level would be erroneously attached to any children in the house. These concerns arise from the assumption that parental education is the most influential on a child.

There were two situations where it was apparent this was a problem. The first was when an older sibling in the family had higher educational qualifications than their parent(s). This scenario is entirely possible given the pattern of increasing educational qualifications in each generational cohort (see Figure 5 in Section 1.3.2). The second situation applied to the 16.7% of children who live in houses with other individuals or families in New Zealand, who are mainly in sole parent families, and of Māori or Pacific ethnicity (Statistics New Zealand, 1999a). There may have been a number of non-parent adults who could have been identified as having the highest qualification level, who may not have been related to the child.

There was also concern that the changing demographics of households in New Zealand may alter trends by education. There has been a large increase in single parent households over the time period under study. Between 1986 and 1996 there was an increase in the proportion of children living in single parent households from 15.7% to 23.6% (Statistics New Zealand, 1999a). These households are mainly headed by women with no educational qualifications. This meant that in the first two cohorts there were more households with 2 parents to 'find' a higher qualification within each house. So it was possible that there may be some bias towards finding a higher qualification in the first cohorts than in the later ones.

In order to address these concerns the second variable was created: maternal education. This was a probabilistic variable as there was no consistent way of ascertaining the mother in each house in every cohort. This 'probable mother' variable was coded using a SAS programme to find the female in the house who was 15-45 years older than the youngest and oldest children in the house respectively. This variable was checked against the 1986 census, which had family coding. The probabilistic method as a 'test' compared to the actual 1986 family coding variable had a sensitivity of 92.8%, specificity of 71.1% and positive predictive value of 96.3% (see Table 12).

Table 12 Checking the maternal education variable using 1986 census data.

Probable Mothers	True Mothers	
	Yes	No
Yes	378669	14607
No	29517	36090

Only education trends by maternal education are presented in the body of the thesis. The results for education by highest level in the household are presented for comparison in Appendix 3, and were similar.

1.2.2 Outcome Measurement

Cause of death was taken from mortality files provided by New Zealand Health Information Service (NZHIS), who collate national mortality data. Cause of death is usually assigned from the death certificate, which is completed by a medical practitioner. However information is also gathered from multiple sources, including autopsy reports, cancer case registrations, coroner's reports, police reports, hospital case summaries, Land Transport Safety Authority and Water Safety Council reports (Child and Youth Mortality Review Committee, 2004; NZHIS, 2003). The cause of death is assigned to the underlying basis of

death as recommended by the World Health Organisation's International Classification of Diseases (ICD) (NZHIS, 2003).

Once the mortality files were linked to census records the ICD codes were grouped into broad categories by Statistics New Zealand for privacy reasons. This is particularly important for children, as death is a relatively uncommon occurrence that might lead to disclosure of individual identity. The cause specific mortality analysed in this thesis are those causes that are most pertinent to children (as shown in Table 13).

Table 13 Causes of death and ICD codes

Cause of Death	ICD Code
Unintentional Injury	800-949
Cancer	140-209
Congenital Mortality	740-759
Other	Remaining ICD codes

The group labelled 'Other' is a heterogeneous group, consisting mainly of communicable diseases, asthma and respiratory infections and violent deaths. The number of deaths in each of these groups was too small to allow cause specific analysis. Other causes of death within this group include diabetes, non-congenital heart disease, ischaemic heart disease, cerebrovascular disease, SIDS, suicide and deaths caused by perinatal events.

2 Analysis

All analysis was conducted using deaths weighted for any linkage bias. The analysis was conducted on absolute numbers of (weighted) deaths and person-time in the datalab at Statistics New Zealand. However these numbers (of weighted deaths and person-time) are then rounded for confidentiality reasons by Statistics New Zealand for extraction of tabular data, hence numbers may be slightly different on some tables. The important point is that all rates reported in this thesis are calculated on exact data in the datalab.

2.1 Crude Rates

Crude mortality rates per 100 000 were calculated using the following method for incidence density after Rothman and Greenland (Rothman & Greenland, 1998):

$$\frac{\text{New occurrences over a period of time}}{\text{Time spent by study population at risk over this time period}} \times 100\,000$$

Or in this thesis:

$$\frac{\text{Number of deaths}}{\text{Person-Time at Risk}} \times 100\,000$$

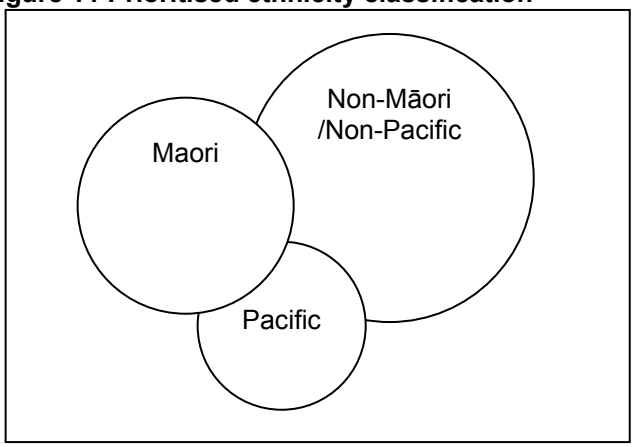
Person-time was calculated for each individual in the cohort. If they had died during follow up the person-time they contributed was the difference between their age (in months) at census and age (in months) at death. For those who did

not die, or were not linked, the total person-time of follow up was 36 months each. Person-time was calculated in the following age bands: 0-1, 1-4, 5-9 and 10-14. Individuals, who moved from one age band into another, contributed the number of months spent in each band to that particular age group. These age groups allowed children to 'age' into the bottom end of the cohort, but excluded children who died aged under one year. For example in this cohort a child who was 8 months at the time of the census and died at age 23 months would have contributed 11.5 months to the person time group of 1-4 year olds (age at census and age at death had 0.5 months added onto them as the age variable was an integer available in months).

2.2 Standard Mortality Rates, Rates Ratios and Rate Differences

Direct standardisation to the 1991 census population, by sex, ethnic group and 5-year age bands, using the method described in Rothman and Greenland (Rothman & Greenland, 1998). The age bands were adjusted slightly for this children's study to 0-1, 1-4, 5-9 and 10-14 age groups. The ethnicity classification used was the prioritised ethnic classification (see Figure 11). This means that respondents who identified as Māori on their census form (even if they identified other ethnic groups as well) are classified as Māori. Individuals who have NOT identified as Māori but have identified a Pacific ethnic group are classified as Pacific ethnicity. The residual ethnic group of Non-Māori/Non-Pacific comprises most of the population and is mainly New Zealand European (commonly known as pakeha).

Figure 11 Prioritised ethnicity classification



Most results are presented as sex combined results, to increase the power of the results. Sex specific results have not been sex standardised, just as ethnicity and age specific results have not been ethnic group or age standardised respectively.

Standard rate differences were calculated by subtracting the reference group standard mortality rate from the standard mortality rate of the group of interest. Confidence intervals were calculated using the method of Rothman and Greenland (Rothman & Greenland, 1998). This measure indicated the actual magnitude of the difference between the groups, whereas rate ratios compare the relative size of the disparity.

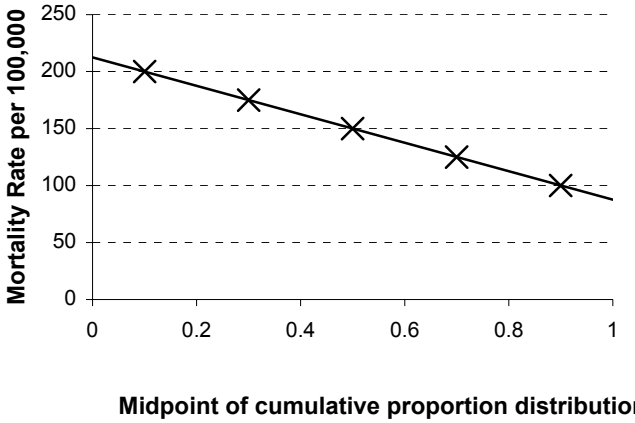
Rate ratios were calculated by dividing the standard mortality rate of one group by the standard mortality rate of the reference group. Thus a rate ratio of greater than one indicates a higher mortality risk in that group compared to the reference group and less than one, lower mortality than the reference group. The reference group was always the highest group (e.g. highest occupational class).

2.3 Relative Index of Inequality and Slope Index of Inequality

To overcome the limitations of standard rate ratios and standard rate differences (see discussion in section 1.1), population measures of inequality were used. The regression-based measures of inequality were calculated according to the methods of Kunst and Mackenbach (Mackenbach & Kunst, 1997). The relative index of inequality (RII) and slope index of inequality (SII) are more sophisticated *relative* and *absolute* measures, respectively. However their calculation requires the exclusion of 'missing' data, thus giving information on the impact of relative and absolute inequality of those children with available data.

Figure 12, below demonstrates how the RII and SII are calculated for income. Income quintiles are ranked from the lowest socioeconomic group to the highest. Each quintile comprises 20% of the population. Thus, the poorest quintile is plotted at 0.1 on the cumulative proportionate distribution of the population (x axis), with a mortality rate of 200 per 100,000. The next richest income quintile will have an x-axis value of 0.3 (0.2 for the previous quintile, plus half of the current quintile), and a y-axis value of 175 per 100,000, and so on.

Figure 12: Hypothetical example of mortality risk by income to demonstrate the calculation of the RII and SII



Having plotted these x-y points, the regression-based slope and intercept can be calculated. In this simple example, the slope is -125 and the intercept is 212.5. The RII is then 212.5/[212.5 –125] = 2.43. That is, the poorest person has an expected mortality risk that is 2.43 times that expected of the richest person, somewhat more than the relative risk of 2.0 derived from simply comparing the (midpoints of the) lowest and highest income quintiles. The SII is simply 125 per 100,000. That is, the poorest person has an expected mortality rate is 125 per 100,000 greater than that expected for the richest person.

In this thesis, RIIs and SIIs are calculated using weighted linear regression of the age and ethnicity standardised mortality rates for quintile groupings of income, and for the five education groups and five occupational class groups. The weights were the person-time in each socioeconomic exposure group (i.e. income quintile or education levels).

2.4 Homogeneity

To explore effect measure modification, effect measures were stratified by a third variable of interest i.e. sex, age, ethnic group and cause of death. The SRRs, SRDs, RIIs and SIIs were then compared across strata. A statistical test

was also used in the comparison, the Wald test of homogeneity. This was calculated by the method described in Rothman and Greenland (Rothman & Greenland, 1998).

Chapter 4: Results

This chapter is divided into four parts. The first section looks at the demographics of the cohorts of children, and at the demographics of the children who died. The next three sections look at socioeconomic trends in mortality by the different socioeconomic exposures: income, maternal education and a hybrid variable of occupational class combined with labour force status. Each of the latter three sections in the chapter commences with a summary of the key findings, and then is divided into sections that look initially at overall mortality (all-cause, over the entire age range), then cause specific mortality, and subsequently at the socioeconomic variable stratified by age, sex and ethnic group. However where there are few differences in the patterns in the occupational class and education sections, these tables have been put into an appendix for reference.

Note that while the analysis was conducted on actual numbers, in order to meet Statistics New Zealand confidentiality protocol any numbers that involve actual people (e.g. deaths, person time) are then rounded to base 3 with a minimum cell size of 6 when presented in tables. Hence the summing of numbers involving multiple cells may not always agree.

1 Cohorts

Table 14 shows the demographics of the children in each cohort on census night. Please note this is slightly different from the final mortality analyses as some will have 'aged out' and some children will have come up from the 0-1 age group into the cohorts. Of interest is the slight decline in the total number of children, which reflects overall falling birth rates, although the effects of the increase in birth rate in the early 1990s can be seen. However the most considerable change seen is in the ethnic mix, with an increase in the number of Māori and Pacific children over this time period. This is due both to higher birth rates and changing definitions of ethnicity (from ancestral descent in 1981 to self identification in 1996).

Table 14 Sex, age and ethnicity of children in study cohorts on census night

			Cohort			
Age	Sex	Ethnicity	1981	1986	1991	1996
0	Males	Māori	5,337	5,346	6,180	7,221
		Pacific	1,488	1,503	2,244	2,280
		Non Māori Non Pacific	17,658	17,766	19,527	17,280
	Females	Māori	5,040	5,259	6,057	6,636
		Pacific	1,374	1,326	2,250	2,187
		Non Māori Non Pacific	16,662	17,139	18,828	16,278
			47,559	**48,339**	**55,086**	**51,882**
1-4	Males	Māori	20,547	20,292	22,845	27,963
		Pacific	5,961	5,814	8,073	9,264
		Non Māori Non Pacific	72,783	71,079	75,888	74,283
	Females	Māori	20,079	19,296	22,317	26,136
		Pacific	5,715	5,511	7,413	8,502
		Non Māori Non Pacific	69,891	67,464	72,516	70,422
			194,976	**189,456**	**209,052**	**216,570**
5-9	Males	Māori	27,207	24,915	24,768	33,057
		Pacific	7,089	7,317	8,244	10,479
		Non Māori Non Pacific	110,223	93,864	91,620	95,349
	Females	Māori	26,394	24,009	23,784	31,824
		Pacific	6,714	6,972	7,731	9,783
		Non Māori Non Pacific	105,528	89,964	87,204	91,011
			283,155	**247,041**	**243,351**	**271,503**
10-14	Males	Māori	25,119	24,396	22,857	26,964
		Pacific	5,949	6,903	7,854	8,409
		Non Māori Non Pacific	113,904	106,398	89,190	86,997
	Females	Māori	24,900	23,943	22,719	26,202
		Pacific	5,784	6,504	7,806	8,034
		Non Māori Non Pacific	110,238	103,494	86,052	82,788
			285,894	**271,638**	**236,478**	**239,394**
Total			***811,584***	***756,474***	***743,967***	***779,349***

Numbers are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Table 15 presents demographic information on children who died. It also shows the numbers of deaths linked by the anonymous record matching process versus the number of weighted deaths. As can be seen, the (weighted) number of deaths declined by 29% over the 18-year period. Boys had a higher number of deaths than girls, but the absolute number of deaths declined by 32% compared to a 24% decline for girls. Injury was the most common cause of mortality among all age groups, followed by other causes and then cancer. Congenital mortality was more common among the 1-4 age group.

Table 15 Linked and weighted deaths by sex, age and cause in all cohorts

			Cohort							
			1981		1986		1991		1996	
Sex	Age at Death	Cause of Death	Linked Deaths	Weighted Deaths	Linked Deaths	Weighted Deaths	Linked Deaths	Weighted Deaths	Linked Deaths	Weighted Deaths
Males	1-4	Injury	54	84	45	78	36	51	33	48
		Congenital	12	18	12	18	9	12	18	21
		Cancer	18	24	12	18	9	12	12	18
		Other	33	45	30	42	33	51	18	24
	5-9	Injury	45	72	30	45	27	36	30	45
		Congenital	6	6	6	6	6	9	6	9
		Cancer	18	27	18	24	15	18	18	21
		Other	21	30	15	24	9	12	18	21
	10-14	Injury	48	75	45	60	33	48	36	60
		Congenital	6	6	9	12	6	6	6	6
		Cancer	15	27	21	27	9	15	9	12
		Other	30	42	36	51	24	42	18	24
			294	**453**	**276**	**399**	**213**	**309**	**222**	**315**
Females	1-4	Injury	30	45	27	45	24	39	21	33
		Congenital	12	18	15	21	12	18	9	15
		Cancer	9	18	15	15	9	12	9	12
		Other	27	39	15	21	9	18	21	30
	5-9	Injury	33	45	21	30	21	33	15	27
		Congenital	6	6	6	6	6	6	6	6
		Cancer	9	12	9	12	12	15	15	15
		Other	12	21	15	21	21	33	9	12
	10-14	Injury	24	39	21	33	12	18	18	27
		Congenital	6	12	6	9	6	6	6	6
		Cancer	12	15	6	9	6	9	9	15
		Other	21	27	18	24	18	24	21	27
			195	**295**	**180**	**252**	**162**	**228**	**159**	**222**
Total			***492***	***744***	***456***	***648***	***375***	***537***	***378***	***537***

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

2 Trends in Mortality by Income

- Mortality rates fell in all income groups between 1981 and 1999.
- A gradient existed for all-cause mortality with mortality rates increasing monotonically from highest, to middle to lowest income groups in the 1991 and 1996 cohorts. However the earlier cohorts suggested a threshold, with the low-income group having higher mortality compared to the middle and high-income groups, which had similar mortality rates.
- The key finding of this chapter is that, between 1981 and 1999, absolute mortality differences (in all-cause mortality by income) remained stable, but increasing relative inequalities were seen.
- Injury mortality is the main determinant of the gradient in all-cause mortality. However there is some evidence of gradients in congenital and other mortality, but none for cancer mortality.
- Male children had higher mortality than female children, but declines in overall mortality rates have affected inequality within the sexes differently.
- There was heterogeneity of inequality by age, with evidence of 1-4 and 5-9 year olds having stronger income-mortality gradients then 10-14 year old children.
- Māori children had higher mortality than non-Māori/non-Pacific children, less decline in mortality has been seen in Māori children.

2.1 Restriction of Cohort

Table 16 shows the person-time and number of deaths within each grouping of income. 16-18% of the person time that was potentially available in each cohort was unable to be utilised, as no income information was available. This amount of information missing from analyses raises the possibility of selection bias. This possibility is discussed in Chapter 4.

Table 16 Person time and number of deaths in each income tertile by age and cohort.

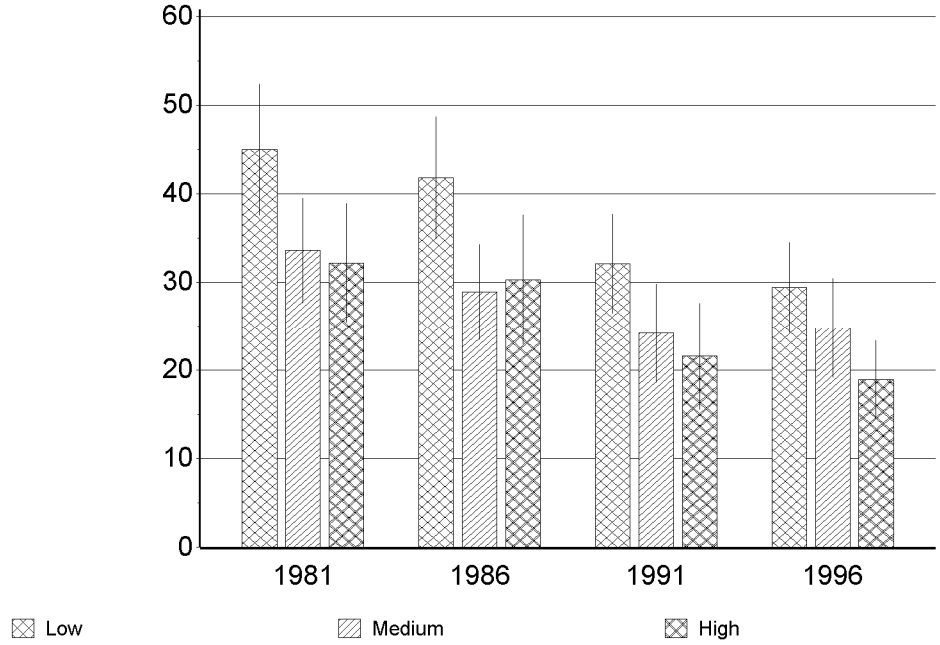
		Cohort											
		1981			1986			1991			1996		
Income	Age	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %
Both Sexes													
Low	1-4	102	124,738	6%	99	135,627	7%	93	172,215	9%	75	162,661	8%
	5-9	75	203,486	9%	60	212,526	11%	63	242,248	12%	57	265,461	13%
	10-14	51	197,182	9%	60	193,595	10%	42	206,466	10%	63	213,551	10%
		228	**525,406**	**24%**	**219**	**541,747**	**27%**	**198**	**620,929**	**31%**	**195**	**641,673**	**31%**
Medium	1-4	84	160,717	7%	60	148,708	7%	36	131,173	7%	45	132,609	6%
	5-9	45	256,699	12%	42	235,615	12%	36	179,979	9%	30	206,565	10%
	10-14	69	254,254	12%	66	246,859	12%	42	167,817	8%	45	175,856	8%
		198	**671,669**	**31%**	**168**	**631,181**	**32%**	**114**	**478,969**	**24%**	**120**	**515,030**	**25%**
High	1-4	48	106,694	5%	39	89,835	5%	30	124,854	6%	36	144,561	7%
	5-9	51	210,323	10%	27	147,439	7%	33	188,501	9%	30	228,259	11%
	10-14	84	293,584	14%	57	217,718	11%	45	223,255	11%	42	235,037	11%
		183	**610,601**	**28%**	**123**	**454,992**	**23%**	**108**	**536,610**	**27%**	**108**	**607,858**	**29%**
Missing	1-4	51	88,972	4%	66	101,414	5%	48	107,026	5%	48	90,012	4%
	5-9	48	132,104	6%	36	134,807	7%	33	134,463	7%	33	132,232	6%
	10-14	33	133,486	6%	45	126,559	6%	36	112,138	6%	30	113,171	5%
		132	**354,562**	**16%**	**147**	**362,780**	**18%**	**117**	**353,627**	**18%**	**111**	**335,415**	**16%**
		744	***2,162,238***	***100%***	***648***	***1,990,701***	***100%***	***537***	***1,990,134***	***100%***	***537***	***2,099,976***	***100%***

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

2.2 All-Cause Mortality

Figure 13 demonstrates that during the time period under study, there was a decline in the overall mortality for 1-14 year olds. This decline was seen for all income groups, although the rate of decline varies by income group.

Figure 13 All-cause mortality by income both sexes ages 1-14 years.

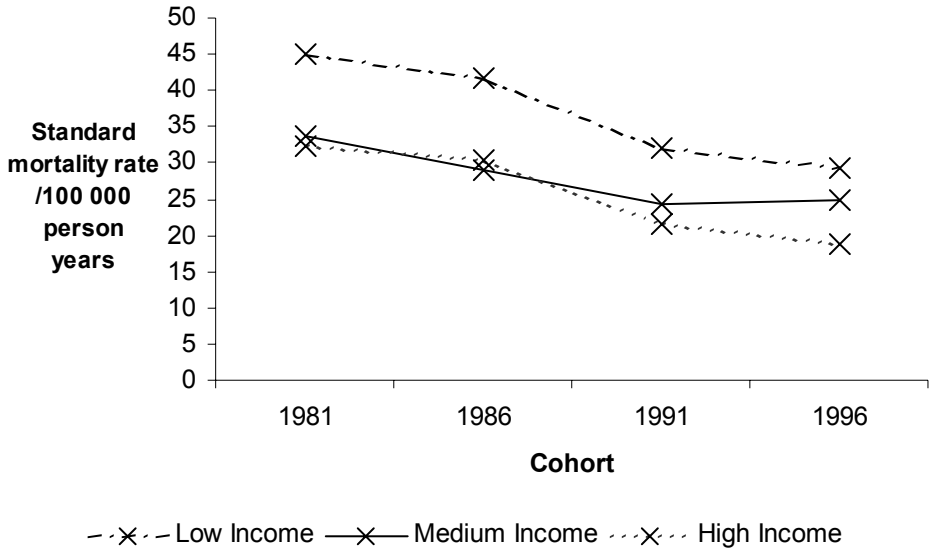


Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years

Mortality from all causes in the low-income group declined by 35%, from 45/100 000 to 29/100 000 over the entire study period (p-trend 0.03). The greatest drop for the low-income group was between the 1986 and 1991 cohorts. The middle income and high-income groups showed declines in mortality rates, from 34/100 000 to 25/100 000 (p-trend 0.08) and 32/100 000 to 19/100 000 (p-trend 0.03) respectively. This equated to a 26% decline in the middle-income group and 41% in the high-income group over the time period.

There appeared to be a threshold effect of income in the 1980s, with middle and high-income groups having almost identical standard mortality rates over this period. This would suggest that only low-income children had an elevated mortality risk, however in the 1990s more evidence of a monotonic gradient emerged (see Figure 14). This was due to a flattening of the decline in mortality rates in the middle-income group between 1991 and 1996.

Figure 14 Standard mortality rates by income both sexes ages 1-14 years.



Age, sex and ethnicity standardised

Evidence of differential mortality between low and high-income groups was seen in the standard rate ratio (SRR), which indicated an elevated mortality risk of 40-60% for low-income children compared to high-income children in all cohorts (see Table 17). Only the 1986 SRR had a confidence interval that includes 1, suggesting that these findings are unlikely to be due to chance.

Table 17 All-cause mortality rates per 100 000 person years, SRR and SRD, by income both sexes ages 1-14 years.

Cohort	Equivalised Income	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1981	Low	225	45	(38 - 52)	1.4	(1.1 - 1.8)	13	(3 - 23)
	Medium	201	34	(28 - 40)	1.0	(0.8 - 1.4)	1	(-8 - 10)
	High	186	32	(26 - 39)	1.0		0	
1986	Low	213	42	(35 - 49)	1.4	(1.0 - 1.8)	12	(1 - 22)
	Medium	171	29	(24 - 34)	1.0	(0.7 - 1.3)	-1	(-11 - 8)
	High	123	30	(23 - 38)	1.0		0	
1991	Low	201	32	(27 - 38)	1.5	(1.1 - 2.1)	11	(2 - 19)
	Medium	114	24	(19 - 30)	1.1	(0.8 - 1.6)	3	(-6 - 11)
	High	108	22	(16 - 28)	1.0		0	
1996	Low	195	29	(24 - 35)	1.6	(1.2 - 2.1)	11	(4 - 17)
	Medium	123	25	(19 - 30)	1.3	(0.9 - 1.8)	6	(-1 - 13)
	High	108	19	(15 - 23)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Standard rate differences (SRD) between the low and high-income groups remained stable at 12/100 000 (95%CI 3-23/100 000) in 1981 and 11/100 000

in 1996 (95%CI 4-17/100 000). On the background of falling mortality rates, a preservation of absolute inequalities must be accompanied by a rise in relative inequalities. This was indeed seen with the increase in the standard rate ratio between low and high-income groups over time from 1.4 (95%CI 1.1-1.8) to 1.6 (95%CI 1.2-2.1). However, despite the logic supporting the increase in relative inequality, the test for trend was not statistically significant (p-trend 0.09).

Absolute and relative differences between the high and middle-income groups reflected the emerging gradient previously discussed. The rate differences are initially minimal, 1/100 000 (95%CI –8-10/100 000) in 1981, but increase by 1996 to 6/100 000 (95%CI -1-13/100 000). There was a related increase in the SRR from 1.0 (95%CI 0.8-1.4) in 1981 to 1.3 (95%CI 0.9-1.8) in 1996. However confidence intervals are wide for both these measures.

The results of the relative index of inequality (RII) and slope index of inequality (SII) in Table 18 confirm the patterns seen in the SRR and SRD. The SII was stable over the time period under study at 15/100 000 (95%CI 1-29/100 000) in 1981 and 14/100 000 (95%CI 8-20/100 000) in 1996, and an increase was seen in the RII from 1.5 (95%CI 1.0 –2.2) to 1.8 (95%CI 1.1 –2.9). Trend analysis of the RII showed that this increase was of borderline significance (p=0.06).

Table 18 All-cause mortality RII and SII by income both sexes ages 1-14 years.

Cohort	RII	95%CI	SII	95%CI
1981	1.5	(1.0 - 2.2)	15	(1 - 29)
1986	1.6	(1.0 - 2.4)	15	(0 - 29)
1991	1.8	(1.1 - 3.0)	15	(3 - 28)
1996	1.8	(1.1 - 2.9)	14	(8 - 20)
P (Trend)	*0.06*		*0.12*	

Age, sex and ethnicity standardised, SII per 100 000 person years

2.3 Variation in Mortality by Cause of Death

As discussed in the literature review there is considerable variation internationally in gradients by cause of death. New Zealand data also shows variations and this thesis updates previous findings from work published from the NZCMS children's cohort of 1991 to 1994 (Blakely et al., 2003).

2.3.1 Injury

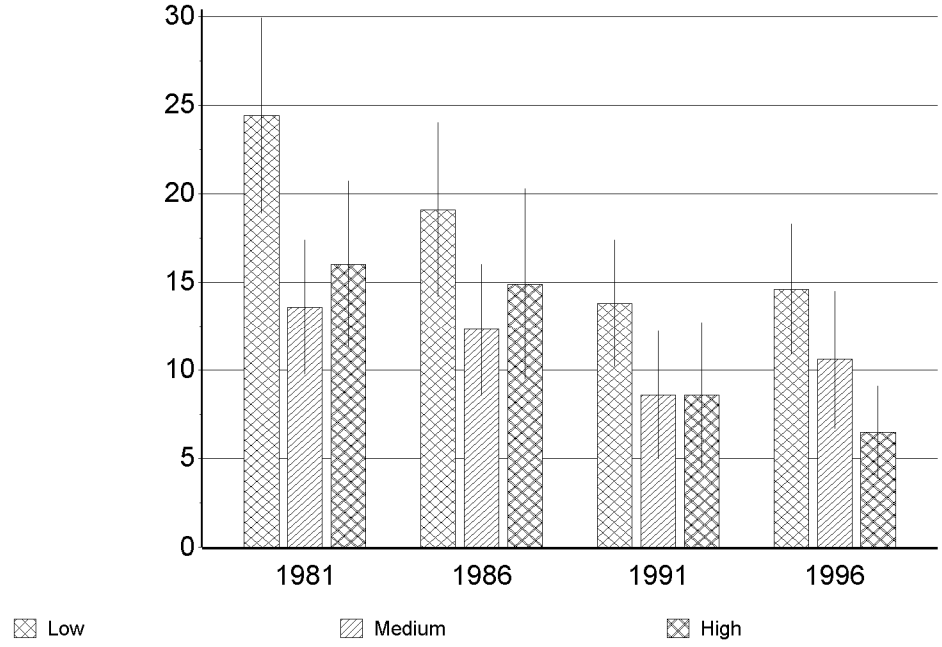
Figure 15 and Table 19 show the mortality rates and gradients for injury mortality in New Zealand 1981-1999. While the data show that injury mortality declined for all income groups between 1981 and 1999, these declines preferentially favoured the high-income group whose mortality rates fell by 52% compared to 21% and 37% for middle and low-income groups. In the low and middle income groups most of the decline occurred in the first three cohorts and the rates stabilised in the last period. The high-income groups had greatest decline between 1986 and 1991.

Table 19 Injury mortality rates per 100 000 person years SRR and SRD, by income both sexes ages 1-14 years.

Cohort	Equivalised Income	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1981	Low	123	24	(19 - 30)	1.5	(1.1 - 2.2)	8	(1 - 16)
	Medium	81	14	(10 - 17)	0.9	(0.6 - 1.3)	-2	(-8 - 4)
	High	99	16	(11 - 21)	1.0		0	
1986	Low	96	19	(14 - 24)	1.3	(0.8 - 2.0)	4	(-3 - 11)
	Medium	69	12	(9 - 16)	0.8	(0.5 - 1.3)	-3	(-9 - 4)
	High	60	15	(10 - 20)	1.0		0	
1991	Low	87	14	(10 - 17)	1.6	(0.9 - 2.8)	5	(-0 - 11)
	Medium	39	9	(5 - 12)	1.0	(0.5 - 1.9)	0	(-6 - 6)
	High	42	9	(5 - 13)	1.0		0	
1996	Low	99	15	(11 - 18)	2.3	(1.4 - 3.6)	8	(4 - 13)
	Medium	51	11	(7 - 15)	1.6	(0.9 - 2.8)	4	(-1 - 9)
	High	36	7	(4 - 9)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Figure 15 Injury mortality by income, both sexes and ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years

The mortality gradient by income showed an interesting pattern. Children in low-income families (compared to those in high income families) consistently had higher mortality risk, seen in the higher SRR of the low-income group. This excess risk ranged from 30% to 130% compared to the high-income group. The middle-income group actually had a slightly lower standard mortality rate in 1981 and 1986 compared to the high-income group. However by 1991 middle and high-income groups have the same standard mortality rates and in 1996 there is a stepwise gradient, between all income groups (see Table 19). These gradients were mainly driven by income-injury mortality gradients in the 1-4 age group where mortality rates are higher. However a gradient was also seen in the 5-9 age group with flattening in the 10-14 group (data not shown).

Trends in relative and absolute inequalities are most easy interpreted from the population measures of inequality in Table 20. The SII initially declined from 12/100 000 (95%CI 0-23/100 000) in 1981 to 6/100 000 (95%CI 2-10/100 000) in 1991 and subsequently increased in 1996 to 11/100 000 (95%CI 6-16/100 000). The RII increased from 2.0 (95%CI 1.1-3.7) to 3.0 (95%CI 1.2-7.6)

although it declined between 1981 and 1986 (see Table 20). These RII and SII have wide confidence intervals and some of the findings may be due to chance however overall they indicate that that there is an increase in relative inequality, similar to the results seen in all-cause mortality. This is not unexpected given that injury mortality is the main cause of death in children.

Table 20 Injury mortality RII and SII by income both sexes ages 1-14 years.

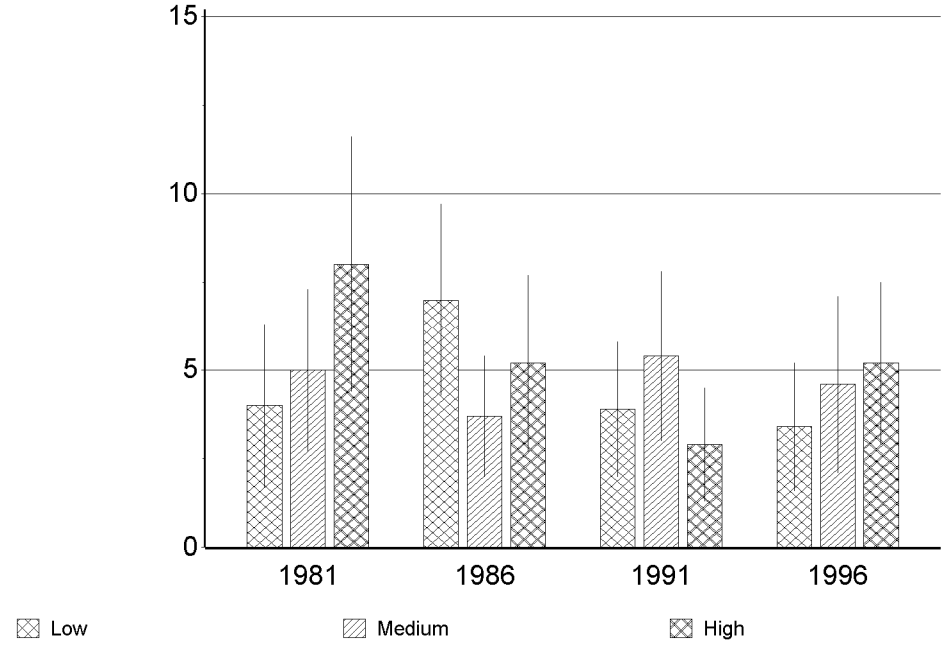
Cohort	RII	95%CI	SII	95%CI
1981	2.0	(1.1 - 3.7)	12	(0 - 23)
1986	1.6	(0.8 - 3.1)	7	(-9 - 23)
1991	1.9	(0.8 - 4.3)	6	(2 - 10)
1996	3.0	(1.2 - 7.6)	11	(6 - 16)
P (Trend)	*0.45*		*0.71*	

Age, sex and ethnicity standardised, SII per 100 000 person years

2.3.2 Cancer

Overall cancer mortality was low; with standard rates ranging from 2.3-8.6/100 000 (see Figure 16). The small numbers in each income group within each cohort make it difficult to draw conclusions about the existence of mortality gradients, and interpretation of the trends of them would have been even more hazardous. Hence pooled results for all cohorts are presented in Table 21 to examine whether inequalities existed over all cohorts.

Figure 16 Cancer mortality by income both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years

Table 21 All years cancer mortality rates per 100 000 person years SRR and SRD, by income both sexes ages 1-14 years.

Equivalised Income	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Low	105	4.6	(3.5 - 5.6)	0.84	(0.60 - 1.17)	-0.9	(-2.6 - 0.8)
Medium	108	4.7	(3.6 - 5.8)	0.87	(0.62 - 1.22)	-0.7	(-2.4 - 1.0)
High	108	5.4	(4.1 - 6.8)	1.00		0.0	

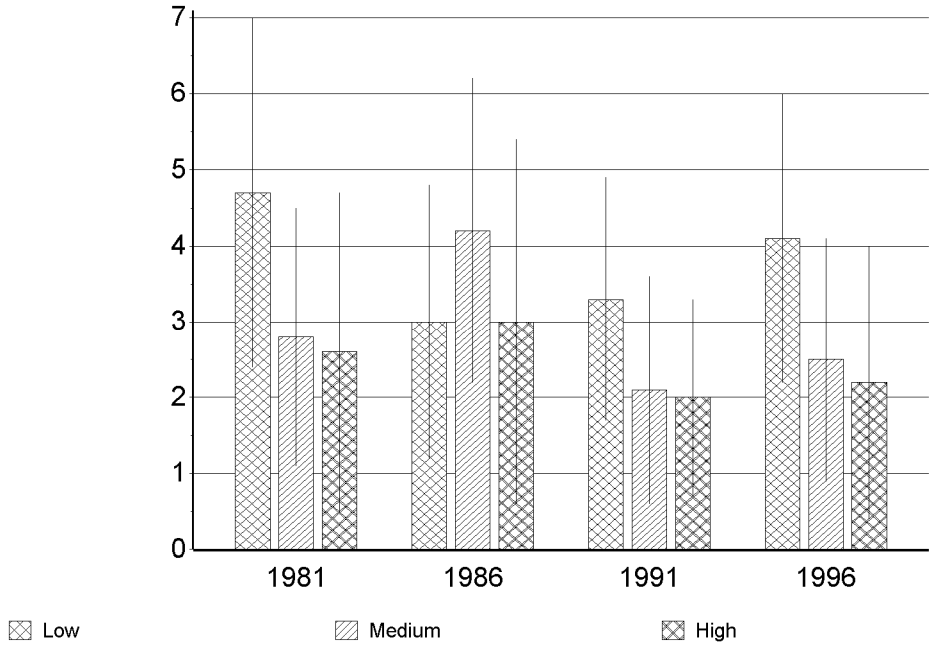
Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Table 21 showed evidence of either no income-cancer mortality gradient for children, or a very slight gradient of increasing mortality with increasing income. The SRR for the low-income group was 0.84 (95%CI 0.60-1.17) and for middle-income children was 0.87 (95%CI 0.62-1.22). However the confidence intervals all include 1, so it is difficult to conclude that there is any difference in cancer mortality by income.

2.3.3 Congenital

Congenital mortality rates were also low; between 2-5/100 000 over all cohorts (see Figure 17). The small numbers and wide confidence intervals again make trends difficult to interpret, however there is some suggestion of a gradient in mortality by income in congenital mortality. Pooled results seen in Table 22 confirmed this, with a SRR for low-income children of 1.52 (95%CI 0.95-2.43) and for medium income children 1.17 (95%CI 0.72-1.91). However confidence intervals cross one meaning that this gradient is not statistically significant.

Figure 17 Congenital mortality by income both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years

Table 22 All years congenital mortality rates per 100 000 person years, SRR and SRD, by income both sexes ages 1-14 years.

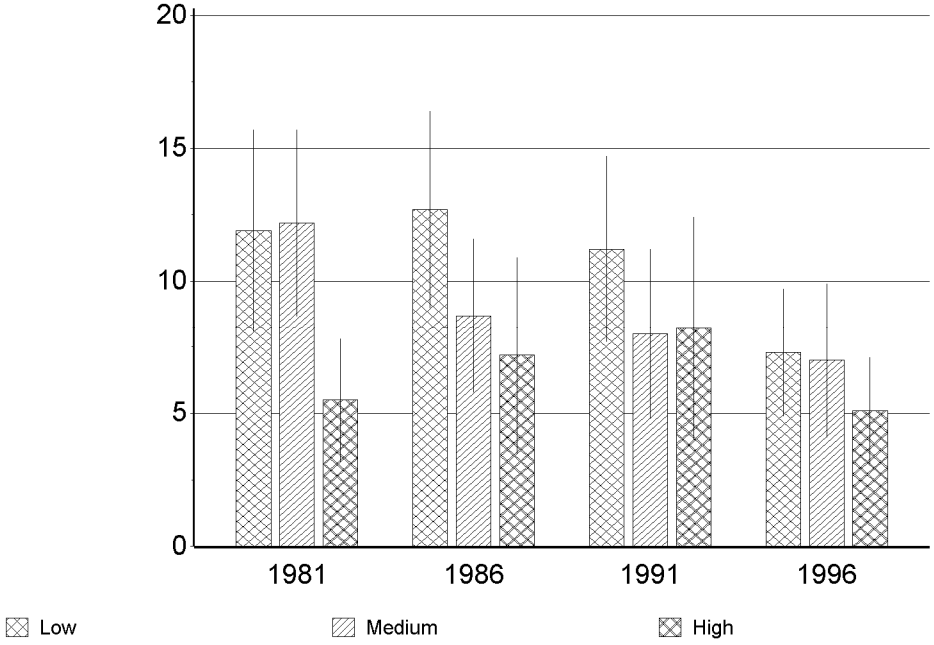
Equivalised Income	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Low	84	3.7	(2.8 - 4.7)	1.52	(0.95 - 2.43)	1.3	(-0.1 - 2.6)
Medium	60	2.9	(2.0 - 3.7)	1.17	(0.72 - 1.91)	0.4	(-0.9 - 1.7)
High	45	2.5	(1.5 - 3.4)	1.00		0.0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

2.3.4 Other Causes

Figure 18 shows that mortality from all other causes has reduced over time in both the low and medium income groups. The standard rate of the high-income group increased from 1981 to 1991 and then declined again. However given the declining mortality rates in the other income groups, the apparent increase in mortality in the high income group may represent random variation rather than a true trend.

Figure 18 Other mortality by income both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years.

Figure 18 also shows that low-income children had higher mortality rates than high-income children in all cohorts. The medium income group was initially at the same level as low income and then declined to the same level as the high-income group in 1991 and subsequently remained static in 1996. These figures have wide confidence intervals; however the pooled estimate in Table 23 confirms the existence of income gradients. The low-income group had a SRR

of 1.69 (95%CI 1.28-2.23) compared to the high-income group, and the medium income group had a SRR of 1.44 (95%CI 1.08-1.93).

Table 23 All years other mortality rates per 100 000 person years, SRR and SRD, by income both sexes ages 1-14 years.

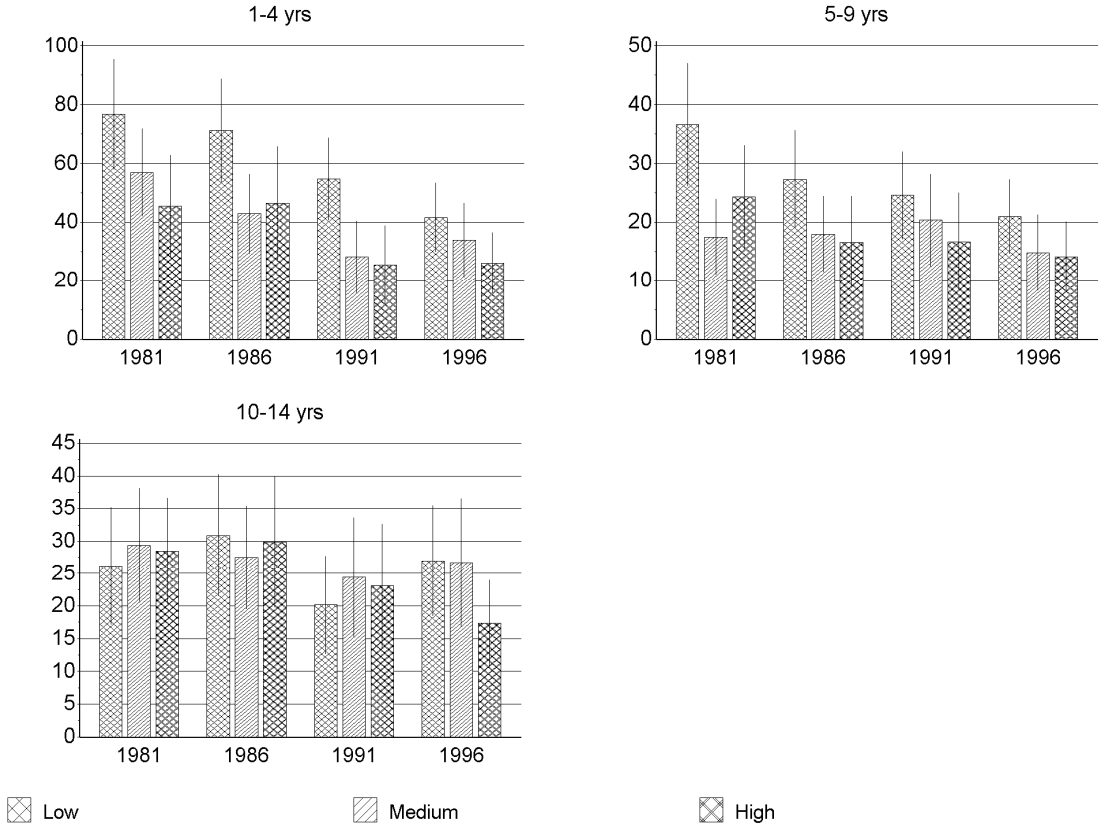
Equivalised Income	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Low	240	10.6	(9.0 - 12.3)	1.69	(1.28 - 2.23)	4.3	(2.1 - 6.5)
Medium	195	9.1	(7.5 - 10.6)	1.44	(1.08 - 1.93)	2.8	(0.6 - 4.9)
High	132	6.3	(4.8 - 7.7)	1.00		0.0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

2.4 Variation in All-cause Mortality by Age

As noted during the literature review, age specific mortality rates show considerable variation in both the level and existence of socioeconomic gradients in mortality. Figure 19 illustrates the age differences in this study. Mortality rates were highest in the 1-4 year age group, with lower rates in the 5-9 and 10-14 year olds.

Figure 19 Age specific all-cause mortality by income both sexes.



Sex and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years..

There were declines in standard mortality rates for most income groups within each age range (see Table 24). Mortality in the 1-4 age group declined 45% for the low-income group from 77/100 000 (95%CI 58-95/100 000) in 1981 to 42/100 000 (95%CI 30-53/100 000) in 1996. Mortality fell 40% for the middle-income group from 57/100 000 (95%CI 42-72/100 000) in 1981 to 34/100 000 (95%CI 21-47/100 000). In the high income group mortality declined 43% from 46/100 000 (95%CI 28-63/100 000) in 1981 to 26/100 000 (95%CI 16-37/100 000) in 1996.

In the 5-9 age group similar declines in mortality were seen, with a decline of 43% for the low-income group from 37/100 000 (95%CI 26-47/100 000) in 1981 to 21/100 000 (95%CI 15-27/100 000) in 1996. In the high income group mortality declined 41% from 24/100 000 (95%CI 16-33/100 000) in 1981 to

14/100 000 (95%CI 8-20/100 000) in 1996. However the rate of decline was slightly lower in the middle-income group, at only 20%, from 18/100 000 (95%CI 11-24/100 000) in 1981 to 15/100 000 (95%CI 8-21/100 000) in 1996.

The 10-14 age group was slightly different, with the middle and low-income group mortality rates changing little and the high-income group mortality rates declining. The low-income group showed a 4% increase in mortality from 26/100 000 (95%CI 17-35/100 000) in 1981 to 27/100 000 (95%CI 19-36/100 000) in 1996. The middle-income group showed a 6% decline in mortality from 29/100 000 (95%CI 21-38/100 000) to 27/100 000 (95%CI 17-37/100 000). However, mortality in children in high-income families decreased by 41% from 29/100 000 (95%CI 20-37/100 000) in 1981 to 17/100 000 (95%CI 11-24/100 000) in 1996.

Table 24 Age specific all-cause mortality rates per 100 000 person years, SRR and SRD, by income both sexes.

Cohort	Equivalised Income	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1-4 yrs								
1981	Low	102	77	(58 - 95)	1.7	(1.1 - 2.6)	31	(6 - 57)
	Medium	84	57	(42 - 72)	1.3	(0.8 - 2.0)	12	(-11 - 34)
	High	48	46	(28 - 63)	1.0		0	
1986	Low	99	71	(54 - 89)	1.5	(0.9 - 2.5)	25	(-1 - 51)
	Medium	60	43	(30 - 56)	0.9	(0.5 - 1.5)	-4	(-27 - 20)
	High	39	47	(27 - 66)	1.0		0	
1991	Low	93	55	(41 - 69)	2.2	(1.2 - 3.9)	29	(10 - 49)
	Medium	36	28	(16 - 41)	1.1	(0.6 - 2.2)	3	(-16 - 21)
	High	30	26	(12 - 39)	1.0		0	
1996	Low	75	42	(30 - 53)	1.6	(1.0 - 2.6)	16	(0 - 31)
	Medium	45	34	(21 - 47)	1.3	(0.8 - 2.2)	8	(-8 - 24)
	High	36	26	(16 - 37)	1.0		0	
5-9 yrs								
1981	Low	75	37	(26 - 47)	1.5	(1.0 - 2.4)	12	(-1 - 26)
	Medium	45	18	(11 - 24)	0.7	(0.4 - 1.2)	-7	(-18 - 4)
	High	51	24	(16 - 33)	1.0		0	
1986	Low	60	27	(19 - 36)	1.6	(0.9 - 2.9)	11	(-1 - 22)
	Medium	42	18	(12 - 24)	1.1	(0.6 - 1.9)	1	(-9 - 11)
	High	27	17	(9 - 24)	1.0		0	
1991	Low	63	25	(17 - 32)	1.5	(0.8 - 2.6)	8	(-3 - 19)
	Medium	36	20	(13 - 28)	1.2	(0.7 - 2.3)	4	(-8 - 15)
	High	33	17	(8 - 25)	1.0		0	
1996	Low	57	21	(15 - 27)	1.5	(0.9 - 2.5)	7	(-2 - 16)
	Medium	30	15	(8 - 21)	1.1	(0.6 - 1.9)	1	(-8 - 10)
	High	30	14	(8 - 20)	1.0		0	
10-14 yrs								
1981	Low	51	26	(17 - 35)	0.9	(0.6 - 1.4)	-2	(-14 - 10)
	Medium	69	29	(21 - 38)	1.0	(0.7 - 1.6)	1	(-11 - 13)
	High	84	29	(20 - 37)	1.0		0	
1986	Low	60	31	(22 - 40)	1.0	(0.7 - 1.6)	1	(-13 - 15)
	Medium	66	28	(20 - 35)	0.9	(0.6 - 1.4)	-2	(-15 - 11)
	High	57	30	(20 - 40)	1.0		0	
1991	Low	42	20	(13 - 28)	0.9	(0.5 - 1.5)	-3	(-15 - 9)
	Medium	42	24	(15 - 34)	1.1	(0.6 - 1.8)	1	(-12 - 15)
	High	45	23	(14 - 33)	1.0		0	
1996	Low	63	27	(19 - 36)	1.6	(0.9 - 2.5)	10	(-1 - 20)
	Medium	45	27	(17 - 37)	1.5	(0.9 - 2.6)	9	(-3 - 21)
	High	42	17	(11 - 24)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

The existence of mortality gradients between the income groups is different by age. Table 24 shows evidence of both monotonic gradients and threshold effects in the 1-4 and 5-9 age groups, depending on the cohort period. However for the 10-14 age group there was no evidence of an income-mortality gradient, except in 1996, when SRRs of 1.5 (95%CI 0.9-2.6) and 1.6 (95%CI 0.9-2.5) and SRDs of 9/100 000 (95%CI -3-21/100 000) and 10/100 000 (95%CI –1-30/100 000) were seen in middle and low-income groups respectively when compared to the high income group. Whether this is a chance finding or represents the emergence of socioeconomic gradient in mortality in this age group remains to be seen.

There was also variation in the strength of the income-mortality gradients between the age groups with a pattern of greater relative and absolute inequalities among the 1-4 age group compared to the 5-9 age group. This is evident in both Table 24 and Table 25, where in all but the 1996 cohort the magnitude of the relative effect measures was greatest in the 1-4 years olds, followed by the 5-9 age group and lowest in the 10-14 year olds. This heterogeneity is supported by the results of the Wald test statistic in Table 26.

The trends in relative and absolute inequality differ by age group. The 1-4 age group had an overall decline in both absolute and relative inequalities. However this was not a linear decline and an increase in relative inequality was seen in 1991 in the SRR (up from 1.53 (95%CI 0.9-2.5) to 2.2 (95% CI 1.2-3.9)) and RII (up from 2.3 (95%CI 1.1-4.9) in 1986 to 4.0 (95% CI 1.2-13.1)). This rise was not seen in the other age groups and tests for heterogeneity indicate this is significantly different from the pooled SRR (p=0.02) and RII (p=0.04) (see Table 26).

In the older age groups the pattern was different. In the 5-9 age group a small decline in population absolute inequality was seen, the SII declined from 12/100 000 (95% CI –19-43/100 000) in 1981 to 8/100 000 (95% CI 0-16/100 000) in 1996. The RII stayed relatively stable over the time period. For 10-14 year old children the RII and SII reflect the more egalitarian pattern of their mortality in the first 3 cohorts, however in 1996 the RII increased to 2.2 (95% CI 0.9-5.3) and the SII to 17/100 000 (95% CI 6-29/100 000).

These findings suggest that there is some support for the West hypothesis of decreasing inequalities in early adolescence (West, 1988). However these findings may be due to chance

Table 25 Age specific RII and SII for all-cause mortality by income-both sexes.

Cohort	RII	95%CI	SII	95%CI
1-4 yrs				
1981	2.0	(1.0 - 3.8)	40	(17 - 63)
1986	2.3	(1.1 - 4.9)	42	(3 - 80)
1991	4.0	(1.2 - 13.1)	46	(11 - 82)
1996	1.7	(0.8 - 3.6)	18	(-1 - 36)
P (Trend)	*0.88*		*0.19*	
5-9 yrs				
1981	1.6	(0.8 - 3.4)	12	(-19 - 43)
1986	1.7	(0.8 - 3.8)	11	(-1 - 24)
1991	1.1	(0.5 - 2.5)	3	(-4 - 10)
1996	1.6	(0.7 - 3.6)	8	(0 - 16)
P (Trend)	*0.69*		*0.87*	
10-14 yrs				
1981	0.9	(0.5 - 1.7)	-3	(-18 - 13)
1986	0.9	(0.5 - 1.8)	-2	(-6 - 2)
1991	1.1	(0.5 - 2.5)	3	(-4 - 10)
1996	2.2	(0.9 - 5.3)	17	(6 - 29)
P (Trend)	*0.13*		*0.08*	

Sex and ethnicity standardised, SII per 100 000 person years

Table 26 Wald test for age variations of effect measures by income.

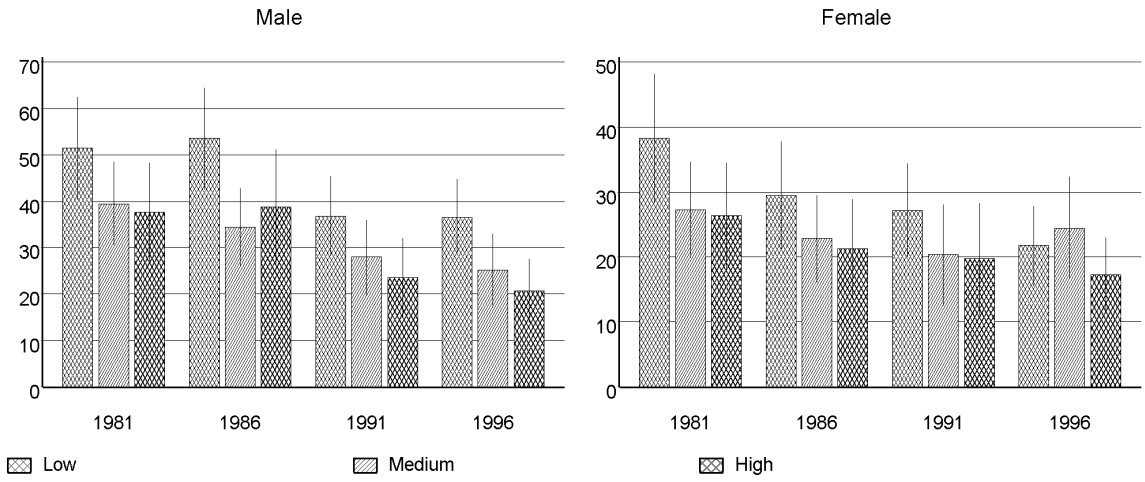
Cohort	P value for Homogeneity of SRR	P value for Homogeneity of SRD	P value for Homogeneity of RII	P value for Homogeneity of SII
1981	0.04	<0.01	0.08	<0.01
1986	0.14	0.07	0.06	<0.01
1991	0.02	<0.01	0.04	<0.01
1996	0.84	0.29	0.57	0.10

P values significant when null hypothesis of homogeneity rejected

2.5 Variation in All-cause Mortality by Sex

The majority of the results are presented for both sexes, as it is unlikely that sex is a confounder in the relationship between income and mortality, as discussed in the methods section 1.3.4 (on confounding and effect modification). However it is possible that sex is an effect modifier, so separate male and female results are shown here to consider this.

Figure 20 Sex specific all-cause mortality by income ages 1-14 years.



Age and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years.

Figure 20 illustrates the main differences between male and female children. These are that male children had higher overall mortality rates compared to female children in all cohort periods. There was a decline in mortality rates for all income groups for both male and female children. However the rates of decline differed by sex, with male mortality declining more than female. For male children in low-income groups there was a 54% decline in mortality from 52/100 000 (95%CI 41-63/100 000) to 37/100 000 (95%CI 29-45/100 000), and in the high-income groups there was a 45% fall, from 38/100 000 (95%CI 27-48/100 000) to 21/100 000 (95%CI 14-27/100 000). The middle-income group had a 40% decline from 40/100 000 (95%CI 31-49/100 000) in 1981 to 25/100 000 (95%CI 17-33/100 000) in 1996. Female children in low-income groups experienced a 42% decline in mortality from 38/100 000 (95%CI 28-48/100 000) to 22/100 000 (95%CI 16-28/100 000), and in the high-income groups there was a 34% fall from 26/100 000 (95%CI 18-35/100 000) to 17/100 000 (95%CI 12-23/100 000). The middle-income group stayed relatively stable at 27/100 000 (95%CI 20-35/100 000) in 1981 and 25/100 000 (95%CI 17-32/100 000) in 1999.

Both sexes showed evidence of an income-mortality gradient between low and high-income groups, although SRR confidence intervals for low-income groups

include one for all groups except males in 1996 (see Table 27). The medium income group seemed to show more of a threshold effect in females and in the earlier cohorts for males.

Table 27 Sex specific all-cause mortality rates per 100 000 person years, SRR and SRD, by income ages 1-14 years.

Cohort	Equivalised Income	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Male								
1981	Low	135	52	(41 - 63)	1.4	(1.0 - 1.9)	14	(-2 - 29)
	Medium	123	40	(31 - 49)	1.1	(0.7 - 1.5)	2	(-12 - 16)
	High	108	38	(27 - 48)	1.0		0	
1986	Low	141	54	(43 - 65)	1.4	(0.9 - 2.0)	15	(-2 - 31)
	Medium	105	35	(26 - 43)	0.9	(0.6 - 1.3)	-4	(-19 - 11)
	High	72	39	(27 - 51)	1.0		0	
1991	Low	114	37	(28 - 46)	1.6	(1.0 - 2.4)	13	(1 - 25)
	Medium	69	28	(20 - 36)	1.2	(0.7 - 1.9)	4	(-7 - 16)
	High	66	24	(15 - 32)	1.0		0	
1996	Low	123	37	(29 - 45)	1.8	(1.2 - 2.6)	16	(6 - 27)
	Medium	66	25	(17 - 33)	1.2	(0.8 - 1.9)	5	(-6 - 15)
	High	57	21	(14 - 27)	1.0		0	
Female								
1981	Low	93	38	(28 - 48)	1.5	(1.0 - 2.2)	12	(-1 - 25)
	Medium	81	27	(20 - 35)	1.0	(0.7 - 1.6)	1	(-10 - 12)
	High	78	26	(18 - 35)	1.0		0	
1986	Low	75	30	(21 - 38)	1.4	(0.9 - 2.2)	8	(-3 - 20)
	Medium	63	23	(16 - 30)	1.1	(0.7 - 1.7)	2	(-9 - 12)
	High	48	21	(14 - 29)	1.0		0	
1991	Low	84	27	(20 - 34)	1.4	(0.8 - 2.3)	8	(-4 - 19)
	Medium	48	20	(13 - 28)	1.0	(0.6 - 1.8)	1	(-11 - 12)
	High	39	20	(11 - 28)	1.0		0	
1996	Low	72	22	(16 - 28)	1.3	(0.8 - 1.9)	5	(-4 - 13)
	Medium	57	25	(17 - 32)	1.4	(0.9 - 2.3)	7	(-3 - 17)
	High	45	17	(12 - 23)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

The interesting difference between males and females was that as mortality rates came down in females, relative inequality (as measured by either the RII or SRR) remained stable and absolute inequality (as measured by the SII or SRD) declined (see Table 28). In contrast a pattern of increasing relative inequality was seen for male children, as the absolute differences were retained even as overall mortality fell. However differences in the SRRs and RIIs by sex during any period are not statistically significant (see Table 29).

Table 28 Sex specific RII and SII for all-cause mortality by income 1-14.

Cohort	RII	95%CI	SII	95%CI
Male				
1981	1.5	(0.9 - 2.5)	17	(2 - 32)
1986	1.6	(0.9 - 2.7)	19	(-3 - 40)
1991	2.2	(1.1 - 4.4)	23	(4 - 42)
1996	2.3	(1.2 - 4.6)	22	(8 - 37)
P (Trend)	*0.05*		*0.09*	
Female				
1981	1.5	(0.8 - 2.8)	13	(-8 - 33)
1986	1.6	(0.8 - 3.1)	11	(-1 - 23)
1991	1.4	(0.7 - 3.0)	8	(1 - 15)
1996	1.3	(0.7 - 2.5)	5	(-10 - 19)
P (Trend)	*0.13*		*<0.01*	

Age and ethnicity standardised, SII per 100 000 person years

Table 29 Wald test for sex variations by income.

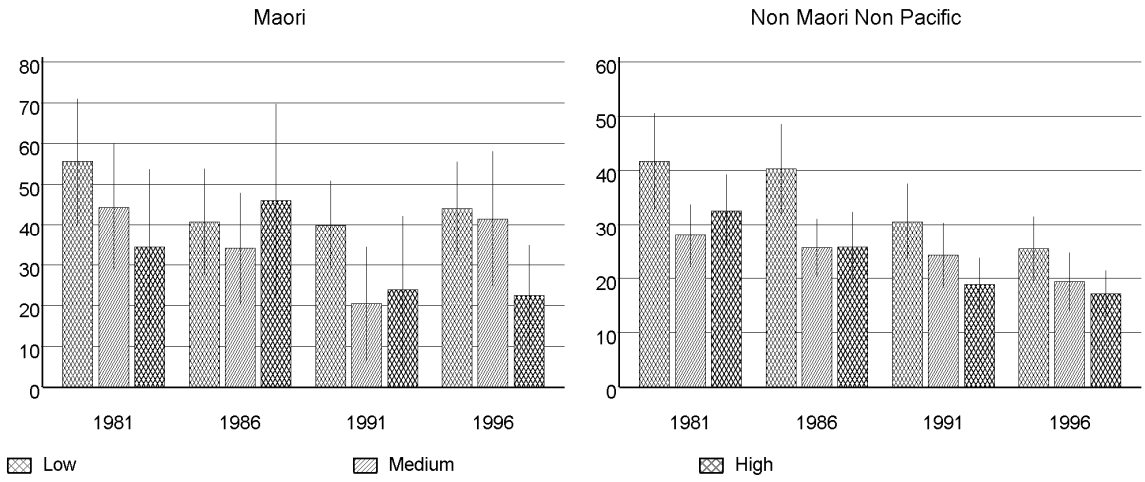
Cohort	P value for Homogeneity of SRR	P value for Homogeneity of SRD	P value for Homogeneity of RII	P value for Homogeneity of SII
1981	0.81	0.85	0.97	0.74
1986	0.98	0.50	0.98	0.46
1991	0.70	0.48	0.40	0.04
1996	0.25	0.08	0.20	0.10

P values significant when null hypothesis of homogeneity rejected

2.6 Variation in All-cause Mortality by Ethnic Group

Ethnic differences in mortality have been more extensively covered in other NZCMS reports but are included here for comparison (Ajwani et al., 2003). Pacific children are not included here as their death rates were too low, particularly in the earlier cohorts, to allow statistical precision. Figure 21 shows the main differences between the ethnic groups. Non-Māori/non-Pacific children had lower mortality rates than Māori children for each income group within each cohort, although 95% confidence limits of each group overlap.

Figure 21 Ethnic specific all-cause mortality by income both sexes ages 1-14 years.



Age and sex standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years.

All income groups for non-Māori/non-Pacific children had a decline in mortality. For low-income children there was a 38% decline in mortality from 42/100 000 (95%CI 33-51/100 000) to 26/100 000 (95%CI 20-32/100 000) and for high-income children a decline of 48% from 33/100 000 (95%CI 26-39/100 000) to 17/100 000 (95%CI 13-21/100 000) was seen. The middle-income group declined 32% from 28/100 000 (95%CI 22-34/100 000) to 19/100 000 (95%CI 14-25/100 000). For low-income Māori children there was a 22% decline in mortality from 56/100 000 (95%CI 40-71/100 000) to 44/100 000 (95%CI 33-56/100 000), while middle-income Māori children had relatively stable mortality at 44/100 000 (95%CI 29-60/100 000) to 41/100 000 (95%CI 25-58/100 000) (a 7% decline). High-income Māori children had a decline of 38% from 34/100 000 (95%CI 15-54/100 000) to 22/100 000 (95%CI 10-35/100 000).

SRRs in Table 30 indicate the presence of income-mortality relative inequalities in both ethnic groups in all cohorts, although there were wide confidence intervals for Māori children probably due to small numbers of deaths. Absolute differences between the income groups within each ethnic groups were also present, and while the magnitude of these differences seemed to be larger in Māori children compared to non-Māori/non-Pacific children the confidence

intervals were extremely wide. This imprecision was supported in the non-significant test of heterogeneity seen in Table 32.

Table 30 Ethnic specific all-cause mortality rates per 100 000 person years, SRR and SRD, by income both sexes ages 1-14 years.

Cohort	Equivalised Income	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Māori								
1981	Low	75	56	(40 - 71)	1.6	(0.9 - 3.0)	21	(-3 - 46)
	Medium	48	44	(29 - 60)	1.3	(0.7 - 2.5)	10	(-15 - 35)
	High	21	34	(15 - 54)	1.0		0	
1986	Low	57	41	(28 - 54)	0.9	(0.5 - 1.6)	-5	(-32 - 22)
	Medium	36	34	(20 - 48)	0.7	(0.4 - 1.4)	-12	(-39 - 16)
	High	24	46	(22 - 70)	1.0		0	
1991	Low	78	40	(29 - 51)	1.7	(0.7 - 3.8)	16	(-5 - 38)
	Medium	18	20	(6 - 34)	0.9	(0.3 - 2.4)	-4	(-27 - 20)
	High	12	24	(5 - 42)	1.0		0	
1996	Low	87	44	(33 - 56)	2.0	(1.1 - 3.6)	22	(5 - 39)
	Medium	45	41	(25 - 58)	1.9	(0.9 - 3.7)	19	(-2 - 40)
	High	18	22	(10 - 35)	1.0		0	
Non Māori Non Pacific								
1981	Low	135	42	(33 - 51)	1.3	(0.9 - 1.7)	9	(-2 - 20)
	Medium	141	28	(22 - 34)	0.9	(0.6 - 1.1)	-5	(-14 - 4)
	High	159	33	(26 - 39)	1.0		0	
1986	Low	138	40	(32 - 49)	1.6	(1.1 - 2.2)	15	(4 - 25)
	Medium	126	26	(20 - 31)	1.0	(0.7 - 1.4)	-0	(-9 - 8)
	High	96	26	(19 - 32)	1.0		0	
1991	Low	108	31	(24 - 38)	1.6	(1.1 - 2.3)	12	(3 - 20)
	Medium	90	24	(18 - 30)	1.3	(0.9 - 1.8)	5	(-2 - 13)
	High	87	19	(14 - 24)	1.0		0	
1996	Low	93	26	(20 - 32)	1.5	(1.1 - 2.1)	8	(1 - 16)
	Medium	72	19	(14 - 25)	1.1	(0.8 - 1.7)	2	(-5 - 9)
	High	84	17	(13 - 21)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Population measures of inequality showed broadly similar patterns in relative inequality for both Māori and non-Māori/non-Pacific children, i.e. no real change (see Table 31). The SII was suggestive of higher absolute inequality by income in Māori children; however, as noted previously, confidence intervals are so wide as to preclude any firm conclusions. This is supported by the lack of statistical evidence of varying relative or absolute inequalities between ethnic groups at any point in time seen in Table 32.

Table 31 Ethnic specific RII and SII for all-cause mortality by income-both sexes ages 1-14 years.

Cohort	RII	95%CI	SII	95%CI
Māori				
1981	1.7	(0.8 - 3.7)	25	(-3 - 52)
1986	1.0	(0.4 - 2.3)	0	(-29 - 29)
1991	3.3	(0.9 - 11.8)	35	(2 - 68)
1996	1.8	(0.8 - 4.0)	23	(3 - 43)
P (Trend)	*0.68*		*0.76*	
Non Māori Non Pacific				
1981	1.4	(0.9 - 2.2)	11	(-11 - 32)
1986	1.9	(1.2 - 3.2)	19	(1 - 37)
1991	2.0	(1.1 - 3.6)	16	(8 - 25)
1996	1.6	(0.9 - 2.8)	10	(-3 - 22)
P (Trend)	*0.50*		*0.57*	

Age and sex standardised, SII per 100 000 person years.

Table 32 Wald test for ethnic group variations by income.

Cohort	P value for Homogeneity of SRR	P value for Homogeneity of SRD	P value for Homogeneity of RII	P value for Homogeneity of SII
1981	0.45	0.34	0.63	0.42
1986	0.10	0.18	0.18	0.27
1991	0.58	0.56	0.32	0.23
1996	0.42	0.16	0.71	0.28

P values significant when null hypothesis of homogeneity rejected

3 Trends in Mortality by Maternal Education

- Mortality rates declined in lower education groups over the time period, but remained stable in the post school education group.
- There was considerable variation in mortality rates in the post school education group over time, making trends in inequality difficult to interpret.
- Injury mortality shows strong gradients by education.
- There is a strong association between maternal education and mortality in the 1-4 age group.

3.1 Cohort Restriction

The person time of children with available information on maternal education is shown in Table 33. The decline in the number of children whose mothers have no qualifications and increase in the number with school and post school qualifications fit with the secular patterns of education in the two decades under study. The increased proportion of children with missing maternal education qualifications in the first cohort is a function of the way census data were collected in 1981.

Table 33 Person time and number of deaths in each maternal education category by age and cohort.

		Cohort											
		1981			1986			1991			1996		
Education Qualification	Age	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %
Both Sexes													
Nil	1-4	153	201,351	9%	99	157,103	8%	87	151,993	8%	66	135,172	6%
	5-9	120	373,026	17%	63	268,377	13%	63	220,768	11%	60	226,516	11%
	10-14	126	453,953	21%	96	331,664	17%	57	236,897	12%	69	209,934	10%
		399	**1,028,330**	**48%**	**258**	**757,143**	**38%**	**207**	**609,657**	**31%**	**195**	**571,622**	**27%**
School	1-4	63	132,580	6%	75	146,612	7%	66	174,660	9%	81	202,065	10%
	5-9	51	184,406	9%	36	197,938	10%	39	225,666	11%	36	297,257	14%
	10-14	48	151,739	7%	39	169,728	9%	36	188,532	9%	48	241,332	11%
		162	**468,725**	**22%**	**150**	**514,278**	**26%**	**141**	**588,858**	**30%**	**165**	**740,653**	**35%**
Post School	1-4	39	104,619	5%	69	144,750	7%	54	195,417	10%	54	178,808	9%
	5-9	21	154,191	7%	51	208,025	10%	57	268,012	13%	51	275,254	13%
	10-14	30	150,232	7%	60	205,338	10%	54	245,634	12%	51	246,483	12%
		90	**409,042**	**19%**	**180**	**558,113**	**28%**	**165**	**709,063**	**36%**	**156**	**700,545**	**33%**
Missing	1-4	30	42,571	2%	18	27,119	1%	6	13,198	1%	6	13,798	1%
	5-9	21	90,989	4%	18	56,047	3%	6	30,745	2%	9	33,490	2%
	10-14	36	122,581	6%	30	78,001	4%	18	38,614	2%	12	39,868	2%
		87	**256,141**	**12%**	**66**	**161,167**	**8%**	**30**	**82,557**	**4%**	**27**	**87,156**	**4%**
		744	***2,162,238***	***100%***	***648***	***1,990,701***	***100%***	***537***	***1,990,134***	***100%***	***537***	***2,099,976***	***100%***

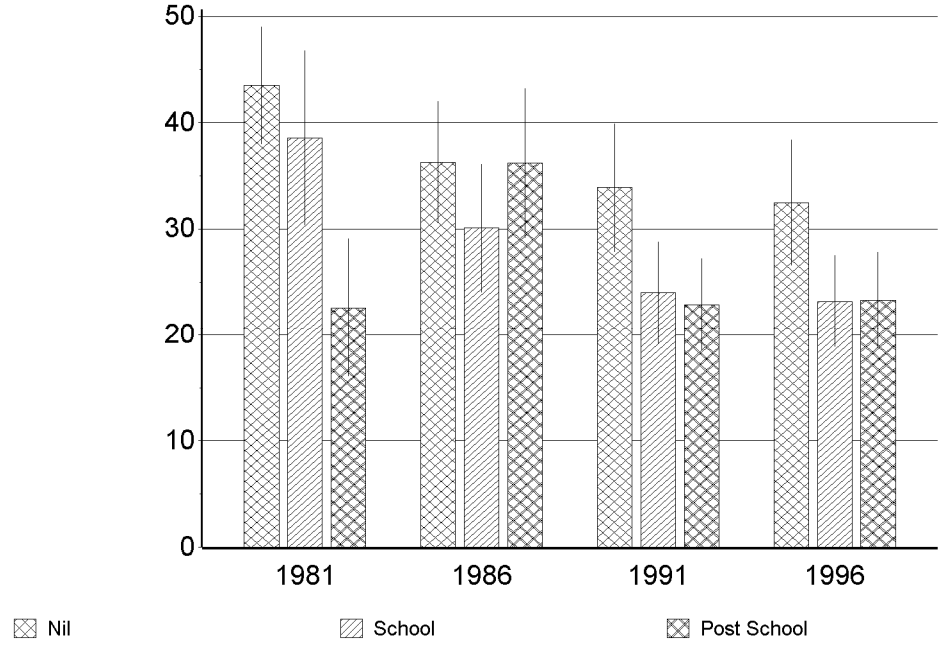
Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

The section on ethnic gradients is not included for maternal education, as due to the small number of Māori children, particularly in the earlier cohorts, and even smaller numbers with mothers in the post school qualifications group, the numbers were extremely unstable and any conclusions on trends could be misleading. Ethnic gradients can be seen in the income and occupational class sections where the numbers were more robust.

3.2 All-Cause Mortality

The changes in mortality by maternal educational qualification were quite similar to those of occupational class described in the following section. Mortality rates have declined in some education groups (see Figure 22) although the post school group has had no decline in mortality.

Figure 22 All-cause mortality by maternal education both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years.

Mortality rates for the post school education group remained stable over the entire time period, at 23/100 000 (95% CI 16-29/100 000) in 1981 and 23/100 000 in 1996 (95% CI 19-28/100 000). However this extremely low mortality rate in 1981 (when compared to both the other education groups in 1981 and the 1986 mortality rates for this group) may be spurious. It certainly makes it more difficult to interpret the data.

The no qualifications group had a mortality decline of 25%, from 44/100 000 (95% CI 38-49/100 000) to 33/100 000 (95% CI 27-38/100 000) and the school

qualifications group declined by 41% from 39/100 000 (95% CI 30-47/100 000) in 1981 to 23/100 000 (95% CI 19-28/100 000) in 1996.

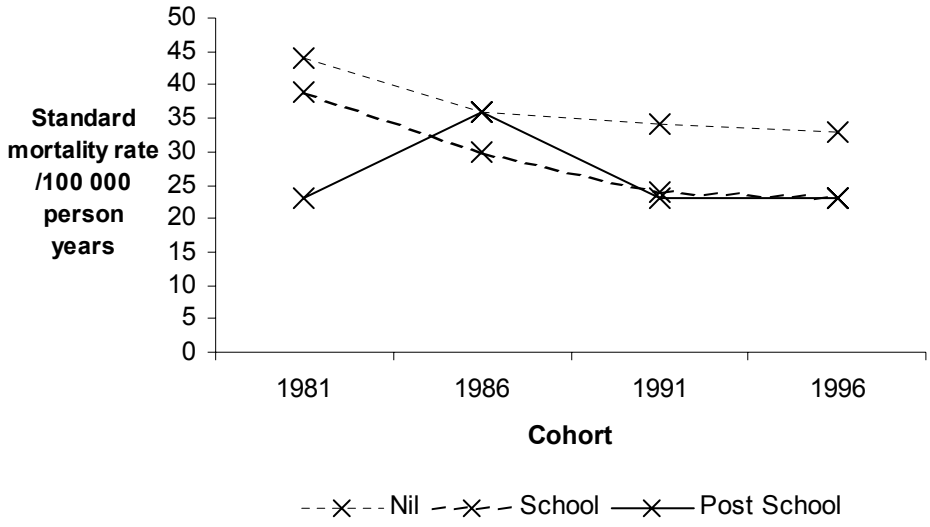
Table 34 confirms the existence of relative and absolute inequalities between the no qualifications and post school qualification groups in all years except 1986, when the increase in mortality in post school group attenuated any differences (this effect is seen well in Figure 23). The level of absolute and relative inequality varied over the cohorts, and trends are best interpreted from the RII and SII given the changing group sizes over time (seen in Table 33). The school qualification group showed a different pattern with similar mortality rates to the post school group in all but the 1981 cohort.

Table 34 All-cause mortality rates per 100 000 person years, SRR and SRD by maternal education both sexes ages 1-14 years.

Cohort	Education Qualification	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1981	Nil	405	44	(38 - 49)	1.9	(1.4 - 2.6)	21	(12 - 29)
	School	159	39	(30 - 47)	1.7	(1.2 - 2.4)	16	(6 - 26)
	Post School	96	23	(16 - 29)	1.0		0	
1986	Nil	258	36	(31 - 42)	1.0	(0.8 - 1.3)	0	(-9 - 9)
	School	144	30	(24 - 36)	0.8	(0.6 - 1.1)	-6	(-15 - 3)
	Post School	180	36	(29 - 43)	1.0		0	
1991	Nil	207	34	(28 - 40)	1.5	(1.1 - 1.9)	11	(4 - 18)
	School	138	24	(19 - 29)	1.1	(0.8 - 1.4)	1	(-5 - 8)
	Post School	165	23	(19 - 27)	1.0		0	
1996	Nil	192	33	(27 - 38)	1.4	(1.1 - 1.8)	9	(2 - 17)
	School	162	23	(19 - 28)	1.0	(0.8 - 1.3)	0	(-6 - 6)
	Post School	153	23	(19 - 28)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Figure 23 Standard mortality rates by maternal education both sexes ages 1-14 years.



Age, sex and ethnicity standardised.

Table 35 shows the RII and SII results. These show an overall decline, with absolute inequalities declining overall from 29/100 000 (95%CI 13-45/100 000) to 13/100 000 (95% CI 3-22/100 000) and relative inequalities also declining from 2.2 (95%CI 1.5-3.4) in 1981 to 1.7 (95%CI 1.1-2.6). However there is considerable variation in both of these measures and simply to take the beginning and end points of non-monotonic changes and conclude a trend from them seems artificial. Of particular note, inequalities were not present in 1986 and a particularly low mortality for the post school group in 1981 drives up the inequality measures for that year. The RII and SII confirm the existence of population absolute and relative inequalities, but it is difficult to determine an overall trend. There may be a suggestion of decreasing inequalities by maternal education but more data from future cohorts are needed.

Table 35 All-cause mortality RII and SII by maternal education both sexes ages 1-14 years.

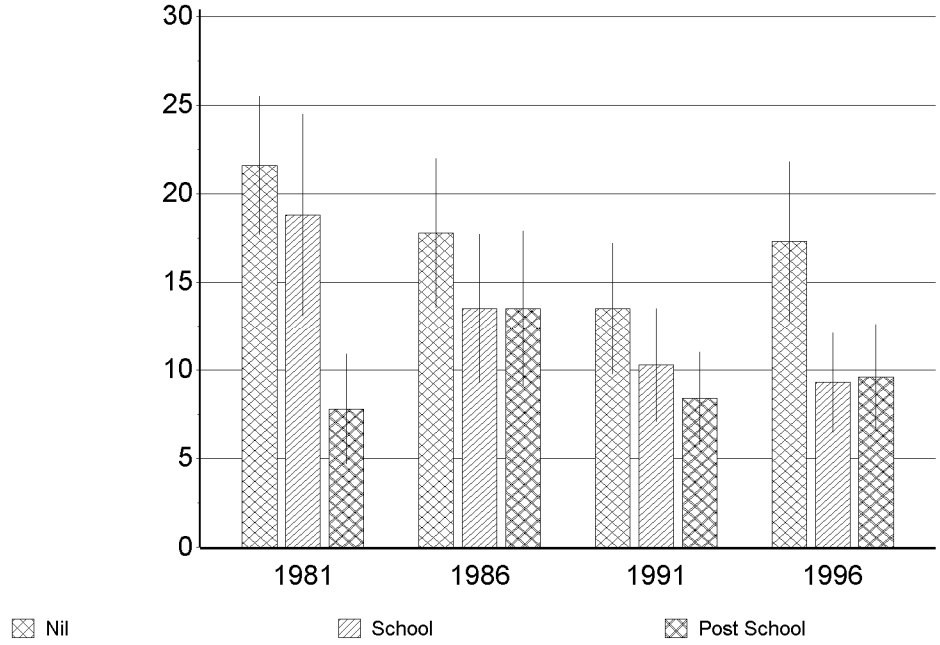
Cohort	RII	95%CI	SII	95%CI
1981	2.2	(1.5 - 3.4)	29	(13 - 45)
1986	1.0	(0.7 - 1.6)	1	(-17 - 19)
1991	2.0	(1.3 - 3.0)	17	(6 - 28)
1996	1.7	(1.1 - 2.6)	13	(3 - 22)
P (Trend)	*0.89*		*0.57*	

Age, sex and ethnicity standardised, SII per 100 000 person years

3.3 Injury Mortality

Injury mortality results are presented here. Cancer, congenital and other mortality tables can be found in Appendix 2.

Figure 24 Injury mortality by maternal education both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years.

Figure 24 shows the trends in injury mortality by maternal education. Standard mortality rates in children with mothers with no qualifications declined 23% from 22/100 000 (95%CI 18-26/100 000) to 17/100 000 (95%CI 13-22/100 000) between 1981 to 1999. Although it should be noted that, in this group, the mortality in the 1996 cohort was actually slightly higher than mortality in the 1991 cohort. Over the same time period mortality in the school qualification group declined 53% from 19/100 000 (95%CI 13-25/100 000) to 9/100 000 (95%CI 7-12/100 000). Mortality in the post school group increased slightly from 8/100 000 (95%CI 5-11/100 000) to 10/100 000 (95%CI 7-13/100 000). The extremely low mortality rates noted in the all-cause post school qualification

group in 1981 is also seen here, thus the seeming increase in injury mortality in this group.

Table 36 shows the relative and absolute inequalities between the school and no qualifications groups and the reference group, which are greatest in the 1981 cohort. Similar to the all-cause mortality group the confidence intervals are quite wide, although all but the 1986 cohort are significant in the no qualifications/post school qualifications rate ratios.

Table 36 Injury mortality rates per 100 000 person years, SRR and SRD, by maternal education both sexes ages 1-14 years.

Cohort	Education Qualification	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1981	Nil	201	22	(18 - 26)	2.8	(1.8 - 4.3)	14	(9 - 19)
	School	75	19	(13 - 25)	2.4	(1.5 - 4.0)	11	(5 - 18)
	Post School	39	8	(5 - 11)	1.0		0	
1986	Nil	129	18	(14 - 22)	1.3	(0.9 - 2.0)	4	(-2 - 10)
	School	63	14	(9 - 18)	1.0	(0.6 - 1.6)	0	(-6 - 6)
	Post School	72	14	(9 - 18)	1.0		0	
1991	Nil	84	14	(10 - 17)	1.6	(1.1 - 2.4)	5	(1 - 10)
	School	60	10	(7 - 14)	1.2	(0.8 - 1.9)	2	(-2 - 6)
	Post School	66	8	(6 - 11)	1.0		0	
1996	Nil	102	17	(13 - 22)	1.8	(1.2 - 2.7)	8	(2 - 13)
	School	63	9	(7 - 12)	1.0	(0.6 - 1.5)	-0	(-4 - 4)
	Post School	63	10	(7 - 13)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

The population inequality measures in Table 37 show that for absolute population inequality by maternal education there has been an overall decline from 18/100 000 (95%CI 3-32/100 000) in 1981 to 9/100 000 (95%CI 0-19/100 000) in 1996. Most of the decline occurred between 1981 and 1986, which may be due to the threshold effect that emerged at that stage between the school and no qualification groups. The RII also showed a small decline from 2.9 (95%CI 1.4-5.9) in 1981 to 2.3 (95%CI 1.0-5.1) in 1996.

Table 37 Injury mortality RII and SII by maternal education both sexes ages 1-14 years.

Cohort	RII	95%CI	SII	95%CI
1981	2.9	(1.4 - 5.9)	18	(3 - 32)
1986	1.8	(1.0 - 3.2)	9	(-4 - 22)
1991	2.0	(1.0 - 4.0)	7	(3 - 11)
1996	2.3	(1.0 - 5.1)	9	(-0 - 19)
P (Trend)	*0.68*		*0.36*	

These trends are again difficult to interpret, partly because of the wide confidence intervals and non significant p values for trend over time in RII and SII.

3.4 Variation in All-cause Mortality by Age

The age variation in all-cause mortality by maternal education was very similar to those seen by income. The key difference was the strength of the inequalities, shown in Table 38. The relative and absolute inequalities are larger than for income, particularly in the 1-4 age group, although, similar to income, the confidence intervals are wide. The other tables and figures are available in Appendix 2 for reference.

Table 38 Age specific RII and SII for all-cause mortality by maternal education both sexes.

Cohort	RII	95%CI	SII	95%CI
1-4 yrs				
1981	3.3	(1.5 - 7.1)	61	(45 - 77)
1986	1.5	(0.9 - 2.6)	22	(-7 - 51)
1991	3.3	(1.4 - 7.6)	41	(17 - 66)
1996	1.7	(0.9 - 3.4)	20	(3 - 37)
P (Trend)	*0.70*		*0.13*	
5-9 yrs				
1981	2.7	(1.2 - 6.0)	26	(8 - 43)
1986	0.8	(0.2 - 2.7)	-5	(-44 - 34)
1991	1.4	(0.7 - 2.7)	7	(-7 - 20)
1996	1.4	(0.6 - 3.4)	6	(-17 - 30)
P (Trend)	*0.42*		*0.19*	
10-14 yrs				
1981	1.1	(0.5 - 2.4)	3	(-28 - 33)
1986	0.7	(0.3 - 1.7)	-10	(-33 - 13)
1991	1.3	(0.7 - 2.6)	5	(-4 - 15)
1996	1.8	(0.9 - 3.6)	13	(-5 - 31)
P (Trend)	*0.27*		*0.24*	

Sex and ethnicity standardised, SII per 100 000 person years.

Table 39 Wald test for age variations by maternal education.

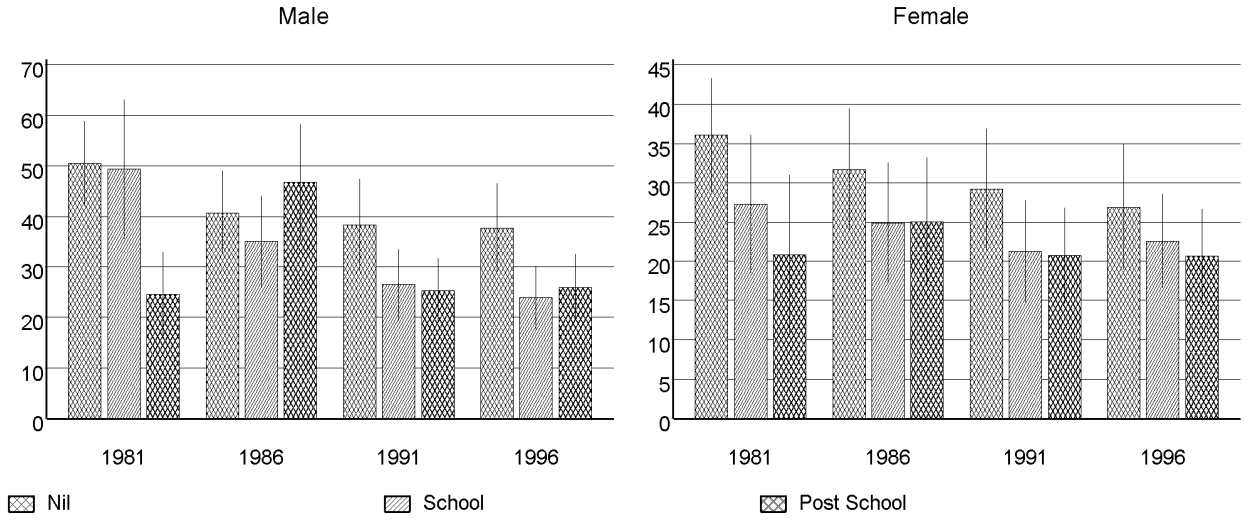
Cohort	P value for Homogeneity of SRR	P value for Homogeneity of SRD	P value for Homogeneity of RII	P value for Homogeneity of SII
1981	0.18	<0.01	0.04	<0.01
1986	0.13	0.12	0.09	0.08
1991	0.02	<0.01	0.05	<0.01
1996	0.88	0.50	0.64	0.32

P values are significant only when the null hypothesis of homogeneity is rejected

3.5 Variation in All-cause Mortality by Sex

Sex specific mortality rates showed declining mortality rates in males and females (see Figure 25).

Figure 25 Sex specific all-cause mortality by maternal education ages 1-14 years.



Age and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years.

There was evidence of inequality by education, for both boys and girls, as seen in Table 40. The magnitude of the absolute and relative inequalities was similar, which is reflected in the null hypothesis not being rejected by tests for homogeneity in Table 41. However there is the suggestion that trends in relative and absolute inequalities by sex are different. Table 40 shows that for females there was a decline in relative and absolute inequality by education, with the RII

of 2.5 (95%CI 1.3-4.7) in 1981 declining to 1.2 (95%CI 0.6-2.4) in 1996 and SII of 26/100 000 (95% CI 17-35/100 000) to 4/100 000 (-15-24/100 000) in 1996 (p trend 0.04). For males stable relative inequality was seen (2.1 (95%CI 1.2-3.6) in 1981 and 2.2 (95%CI 1.2-3.9)) and overall decreasing absolute inequalities (31/100 000 (95%CI 2-61/100 000) to 21/100 000 (95%CI –3-44/100 000)).

Table 40 Sex specific RII and SII for all-cause mortality by maternal education ages 1-14 years.

Cohort	RII	95%CI	SII	95%CI
Male				
1981	2.1	(1.2 - 3.6)	31	(2 - 61)
1986	0.9	(0.6 - 1.4)	-4	(-36 - 28)
1991	2.0	(1.1 - 3.5)	20	(8 - 32)
1996	2.2	(1.2 - 3.9)	21	(-3 - 44)
P (Trend)	*0.70*		*0.98*	
Female				
1981	2.5	(1.3 - 4.7)	26	(17 - 35)
1986	1.2	(0.4 - 3.2)	5	(-40 - 50)
1991	1.9	(1.0 - 3.5)	14	(4 - 25)
1996	1.2	(0.6 - 2.4)	4	(-15 - 24)
P (Trend)	*0.28*		*0.04*	

Age and ethnicity standardised, SII per 100 000 person years

Table 41 Wald test for sex variations by maternal education.

Cohort	P value for Homogeneity of SRR	P value for Homogeneity of SRD	P value for Homogeneity of RII	P value for Homogeneity of SII
1981	0.59	0.22	0.71	0.50
1986	0.16	0.16	0.55	0.73
1991	0.76	0.53	0.90	0.47
1996	0.69	0.46	0.21	0.26

P values are significant only when the null hypothesis of homogeneity is rejected

4 Trends in Mortality by Occupational Class and Labour Force Status

- All-cause mortality declined in all occupational class groups, and in those children with parents not in the labour force. The largest decline in standard mortality rates was seen in the non-labour force group (40%).
- Children with parents not in the labour force had the highest mortality.
- There is evidence suggestive of a gradient in child mortality within the occupational hierarchy, with a slightly increased mortality risk for children in groups 4-6 compared to 1-3 in all except the 1996 cohort. This risk was greatest in 1991 (RR 1.6).
- Overall absolute and relative inequalities by occupational class have declined between 1981 and 1996. The RII declined from 1.7 to 1.4 and the SII from 19–8.
- Divergent trends are seen by sex with reducing relative and absolute inequalities for boys, and increasing relative and absolute differences for girls. It is unclear whether this is a chance finding; however this is different from income.
- Children of farm workers had an increased injury mortality risk in the 1990s compared to children of non-farm workers. This was in the older age group (10-14) in injury mortality.

4.1 Restriction of Cohort

This section presents the results of a hybrid variable, which combines the more 'traditional' occupational class classification with labour force status. Table 42 shows the person time in this variable. The points to note are the increase in person time over the cohorts in the higher Elley Irving groupings (1-3) compared to the decline in the lower occupational class groups (groups 4-6). However the dramatic increase has been in the number of children living in households where no occupational class could be assigned, up from 11% in 1981 to 21% in 1996. The majority of this group are children who are living with parent(s) who are either unemployed or inactive in the labour force. It is important to look at this group, partly because of the known health risks but also because it has become more common over the time period studied. However it is not possible to include this group in regression-based analyses, as they cannot be assigned a place in the occupational hierarchy, hence all RII and SII calculations are on groups 1-3 and 4-6.

Table 42 Person time and number of deaths in each occupational class by age and cohort.

		Cohort											
		1981			1986			1991			1996		
Elley Irving	Age	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %
Both Sexes													
Groups 4-6	1-4	147	233,480	11%	108	207,629	10%	84	174,458	9%	63	171,483	8%
	5-9	96	355,381	16%	63	289,908	15%	45	226,228	11%	54	255,491	12%
	10-14	114	364,038	17%	84	289,811	15%	54	207,542	10%	63	217,656	10%
		357	**952,899**	**44%**	**255**	**787,348**	**40%**	**183**	**608,228**	**31%**	**180**	**644,629**	**31%**
Groups 1-3	1-4	84	191,903	9%	102	206,031	10%	51	215,598	11%	75	233,616	11%
	5-9	84	355,575	16%	75	346,758	17%	57	336,015	17%	54	390,097	19%
	10-14	114	429,946	20%	114	414,036	21%	69	358,078	18%	75	380,534	18%
		282	**977,424**	**45%**	**291**	**966,825**	**49%**	**177**	**909,691**	**46%**	**204**	**1,004,247**	**48%**
Non active	1-4	54	55,739	3%	45	61,924	3%	75	145,211	7%	63	124,744	6%
	5-9	36	91,655	4%	27	93,720	5%	63	182,948	9%	48	186,929	9%
	10-14	15	84,521	4%	27	80,884	4%	39	144,057	7%	45	139,426	7%
		105	**231,914**	**11%**	**99**	**236,528**	**12%**	**177**	**472,215**	**24%**	**156**	**451,100**	**21%**
		744	***2,162,238***	***100%***	***648***	***1,990,701***	***100%***	***537***	***1,990,134***	***100%***	***537***	***2,099,976***	***100%***

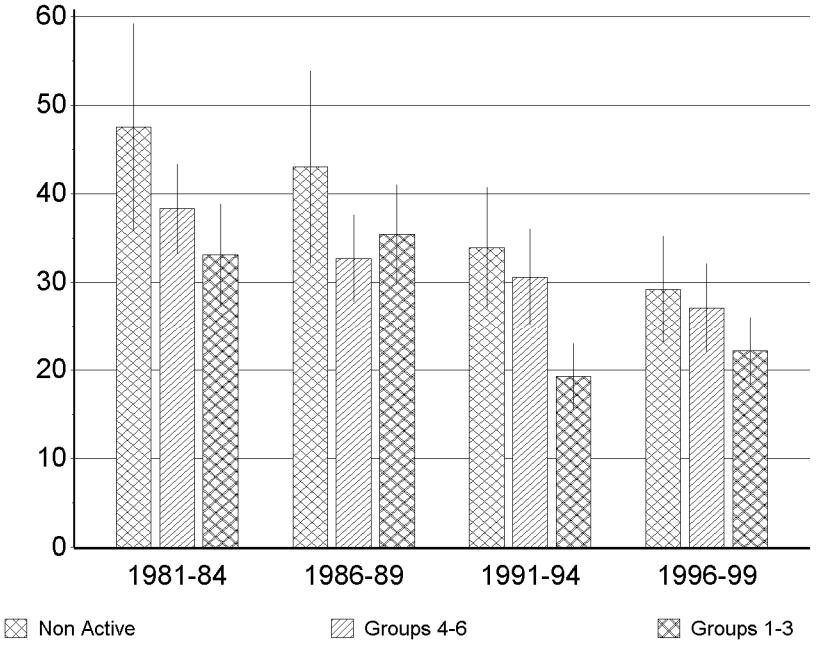
Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Results for cancer, congenital mortality, other mortality and by age and ethnic groups are not presented here, as they are similar to those presented for income and maternal education. Tables are available in Appendix 2.

4.2 All-Cause Mortality

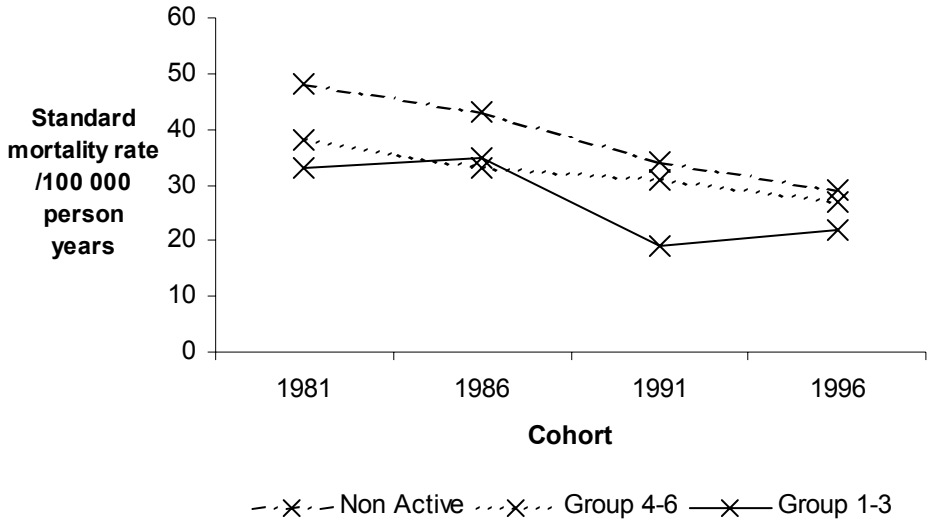
Standard mortality rates showed a similar pattern to those by income (see Figure 26 Figure 27 and Table 43). All groups, in this socioeconomic variable, showed a decline in mortality. The decline was greatest in the non-labour force group at 40%, declining from 48/100 000 (95%CI 36-59/100 000) in 1981 to 29/100 000 (95%CI 23-35/100 000) in 1996. Groups 4-6 (the lower occupational class groups) declined by 29% from 38/100 000 (95%CI 33-43/100 000) in 1981 to 27/100 000 (95%CI 22-32/100 000) in 1996. The higher occupational class declined by 33% from 33/100 000 (95%CI 27-39/100 000) to 22/100 000 (95%CI 18-26/100 000). However latter group did not have a monotonic decline, as in 1986 mortality rates actually increased slightly to 35/100 000 (95%CI 30-41/100 000).

Figure 26 All-cause mortality by occupational class both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years.

Figure 27 Standard mortality rates per 100 000 person years by occupational class both sexes ages 1-14 years.



Age, sex and ethnicity standardised.

The non-labour force group had higher mortality rates than children with parents in the occupational group hierarchy in all cohorts, although in the 1991 and 1996 cohort their mortality rates were only slightly higher than Elley Irving

groups 4-6. For this non-labour force group there was an excess mortality of 20 to 80% compared to the reference group (1-3) over the different cohorts (see Table 43).

The existence of gradients within the occupational hierarchy is not entirely clear. The point estimates in Table 43 are suggestive of a gradient (except in 1986) however confidence intervals around the point estimates are quite wide.

Absolute differences between the groups exist despite falling mortality; although, like relative differences, there does not seem to be any clear pattern in their trend with considerable flux in the absolute differences between group 4-6, the non labour force group and the reference group (see Table 43).

Table 43 All-cause mortality rates per 100 000 person years, SRR and SRD, by occupational class both sexes ages 1-14 years.

Cohort	Elley Irving	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1981	Non Active	105	48	(36 - 59)	1.4	(1.1 - 1.9)	14	(1 - 27)
	Groups 4-6	357	38	(33 - 43)	1.2	(0.9 - 1.4)	5	(-2 - 13)
	Groups 1-3	282	33	(27 - 39)	1.0		0	
1986	Non Active	102	43	(32 - 54)	1.2	(0.9 - 1.6)	8	(-5 - 20)
	Groups 4-6	252	33	(28 - 38)	0.9	(0.7 - 1.1)	-3	(-10 - 5)
	Groups 1-3	294	35	(30 - 41)	1.0		0	
1991	Non Active	177	34	(27 - 41)	1.8	(1.3 - 2.3)	15	(7 - 22)
	Groups 4-6	186	31	(25 - 36)	1.6	(1.2 - 2.1)	11	(5 - 18)
	Groups 1-3	174	19	(16 - 23)	1.0		0	
1996	Non Active	156	29	(23 - 35)	1.3	(1.0 - 1.7)	7	(-0 - 14)
	Groups 4-6	177	27	(22 - 32)	1.2	(0.9 - 1.6)	5	(-1 - 11)
	Groups 1-3	201	22	(18 - 26)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

The population measures of inequality can only be used on the different groupings of occupational class; therefore they do not estimate the effect of those children with no occupational class[3]. The RII and SII confirm the existence of inequalities between the occupational class groups, which was not seen so clearly in the SRR and SRD (see Table 44). However linear patterns in trend are not visible and the variation in the measures combined with relatively

[3] The RII and SII are calculated on 4 groupings of the Elley Irving groups: 1&2, 3, 4, 5&6.

wide confidence intervals mean that more information is needed to determine time changes.

Table 44 All-cause mortality RII and SII by occupational class both sexes ages 1-14 years.

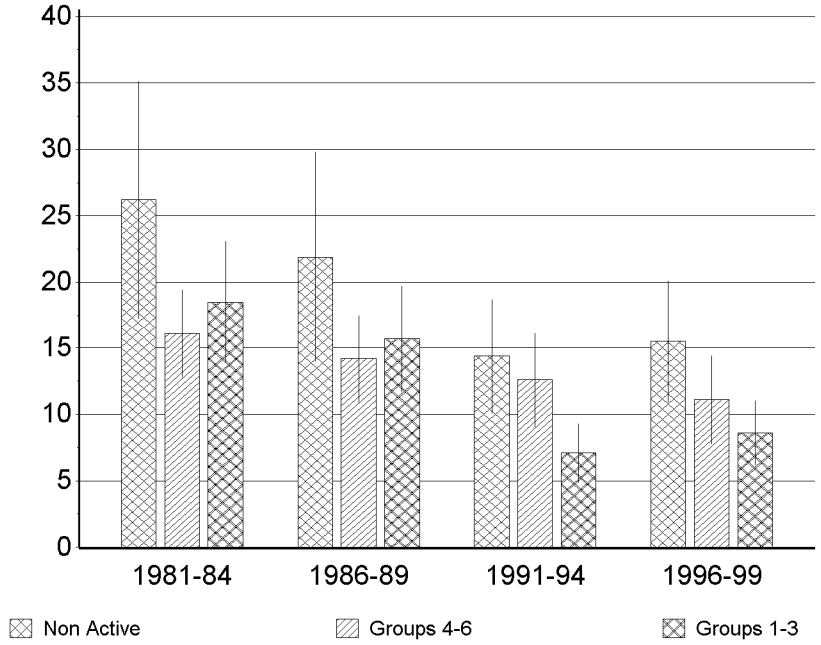
Cohort	RII	95%CI	SII	95%CI
1981	1.7	(1.2 - 2.6)	19	(1 - 36)
1986	0.9	(0.6 - 1.4)	-2	(-10 - 6)
1991	2.4	(1.3 - 4.4)	20	(1 - 38)
1996	1.4	(0.9 - 2.3)	8	(-3 - 19)
P (Trend)	*0.99*		*0.84*	

Age, sex and ethnicity standardised, SII per 100 000 person years

4.3 Injury Mortality

Injury mortality patterns are similar to those discussed in all-cause mortality in the previous section. Standard mortality rates have declined for all groups (see Figure 28 and Table 45). The group of children with parents who were not active in the labour force had higher injury mortality rates than those children whose parents are in the labour force. Within the Elley Irving scale mortality rates are only different in the 1991 and 1996 cohorts.

Figure 28 Injury mortality by occupational class both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years.

Table 45 Injury mortality rates per 100 000 person years, SRR and SRD, by occupational class both sexes ages 1-14 years.

Cohort	Elley Irving	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1981	Non Active	60	26	(17 - 35)	1.4	(0.9 - 2.2)	8	(-2 - 18)
	Groups 4-6	150	16	(13 - 19)	0.9	(0.6 - 1.2)	-2	(-8 - 3)
	Groups 1-3	153	19	(14 - 23)	1.0		0	
1986	Non Active	54	22	(14 - 30)	1.4	(0.9 - 2.2)	6	(-3 - 15)
	Groups 4-6	111	14	(11 - 18)	0.9	(0.6 - 1.3)	-2	(-7 - 4)
	Groups 1-3	129	16	(12 - 20)	1.0		0	
1991	Non Active	75	14	(10 - 19)	2.0	(1.3 - 3.1)	7	(2 - 12)
	Groups 4-6	78	13	(9 - 16)	1.8	(1.2 - 2.7)	5	(1 - 10)
	Groups 1-3	69	7	(5 - 9)	1.0		0	
1996	Non Active	84	16	(11 - 20)	1.8	(1.2 - 2.7)	7	(2 - 12)
	Groups 4-6	75	11	(8 - 14)	1.3	(0.9 - 1.9)	3	(-2 - 7)
	Groups 1-3	81	9	(6 - 11)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

The absolute difference between the non-active group and group 1-3 was stable over the entire period, hence an overall increase in relative inequalities was seen (from 1.4 (95%CI 0.9-2.2) to 1.8 (95%CI 1.2-2.7)).

An increase in relative inequalities is seen in group 4-6 compared to group 1-3 (0.9 in 1981 (95%CI 0.6-1.2) to 1.3 in 1996 (95%CI 0.9-1.9)) as the mortality

differences appear to move from a threshold effect in the 1980s to a gradient in the 1990s (although the 95% confidence intervals include 1 in 1996).

When looking at the RII and SII a similar pattern is seen, a preservation of the absolute inequalities and an overall increase in relative inequalities (see Table 46). However the confidence intervals around these measures are wide and tests for trend are not significant.

Table 46 Injury mortality RII and SII by occupational class both sexes ages 1-14 years

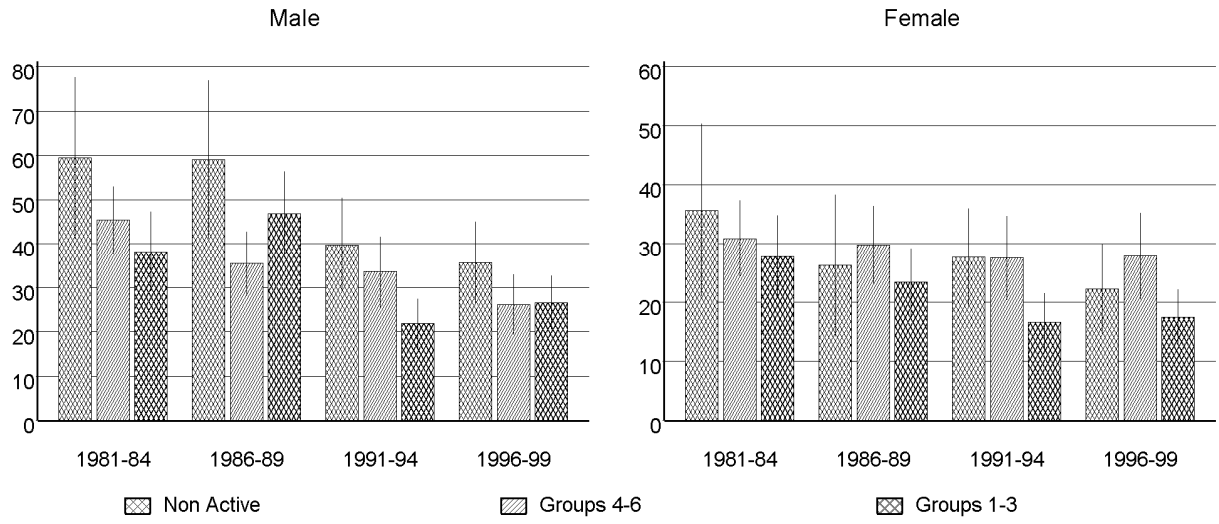
Cohort	RII	95%CI	SII	95%CI
1981	1.7	(0.9 - 3.0)	8	(-23 - 40)
1986	1.2	(0.6 - 2.2)	2	(-12 - 17)
1991	3.2	(1.0 - 10.2)	9	(0 - 19)
1996	1.9	(0.8 - 4.6)	6	(4 - 8)
P (Trend)	*0.60*		*0.98*	

Age, sex and ethnicity standardised, SII per 100 000 person years

4.4 Variation in All-cause Mortality by Sex

Differences by sex are interesting in that all variables have shown different patterns in relative and absolute inequalities. Standard mortality rates are shown in Figure 29, and the table with rates, rate ratios and rate differences can be found Appendix 2.

Figure 29 Sex specific all-cause mortality by occupational class ages 1-14 years.



Age and ethnicity standardised, error bars are 95% confidence intervals.
Y-axis standard mortality rates /100 000 person years.

The results in Table 47 demonstrate the existence of both relative and absolute inequalities by occupational class over this time period (note these values do not include the effect of children who's parents are not in the labour force). The time trends by occupational class are different from those of income and education. For males by occupational class, a decline in both relative and absolute inequality is seen between 1981 and 1999, from 1.9 (95% CI 1.1-3.3) to 0.9 (95% CI 0.5-1.6) and from 24/100 000 (95% CI 2-48/100 000) to 4/100 000 (95% CI –13-4/100 000) respectively. Conversely for females an increase in both the relative and absolute inequalities was seen over the time period. The RII went from 1.6 (95% CI 0.8-2.9) in 1981 to 2.9 (95% CI 1.1-7.4) in 1996 and the SII went from 13/100 000 (95% CI 1-25/100 000) to 21/100 000 (95% CI –3-45/100 000). None of these trends was significant in tests for linear trend, and tests for heterogeneity were significant for these diverging effect measures only in 1996 ($p=0.03$ for RII, $p<0.01$ for SII).

Table 47 Sex specific RII and SII for all-cause mortality by occupational class ages 1-14 years.

Cohort	RII	95%CI	SII	95%CI
Male				
1981	1.9	(1.1 - 3.3)	25	(2 - 48)
1986	0.8	(0.4 - 1.3)	-11	(-41 - 19)
1991	2.7	(1.1 - 6.2)	24	(9 - 38)
1996	0.9	(0.5 - 1.6)	-4	(-13 - 4)
P (Trend)	*0.63*		*0.35*	
Female				
1981	1.6	(0.8 - 2.9)	13	(1 - 25)
1986	1.3	(0.7 - 2.5)	8	(-8 - 23)
1991	2.1	(0.9 - 5.0)	15	(-13 - 42)
1996	2.9	(1.1 - 7.4)	21	(-3 - 45)
P (Trend)	*0.20*		*0.48*	

Age and ethnicity standardised, SII per 100 000 person years

Table 48 Wald test for sex variations by occupational class.

Cohort	P value for Homogeneity of SRR	P value for Homogeneity of SRD	P value for Homogeneity of RII	P value for Homogeneity of SII
1981	0.75	0.58	0.65	0.27
1986	0.02	0.01	0.18	0.16
1991	0.76	0.91	0.71	0.52
1996	0.06	0.09	0.03	<0.01

P values significant when null hypothesis of homogeneity rejected
These tests only include children with an Elley Irving classification.

4.5 Mortality of Children of Farm Workers

In the 1981 and 1986 cohorts no difference in mortality rates between children of farm workers and other children was seen (for both all-cause and injury mortality). However, in the 1991 and 1996 cohorts there was the excess injury mortality risk in children of farm workers, with a SRR 1.9 (95%CI 1.1-1.3) and 1.7 (95% CI 1.0-2.8) respectively. This excess mortality occurred in predominately the 10-14 age group. For cancer and other mortality there was no evidence of any difference in mortality rates or risk for children of farm workers. However, somewhat surprisingly, there was some evidence of a protective effect for children of farm workers for congenital mortality, with a SRR of 0.51 (95%CI 0.28-0.92). The results are presented in tables in Appendix 2.

Chapter 5: Discussion

This first section of this chapter will cover limitations of the study, including the impact of chance, problems with internal validity of the study, and the generalisability to other contexts. The second section will discuss possible reasons for the findings, including how inequalities could be generated, reasons for heterogeneity, the reasons for any changes in inequalities over time, and how these trends correspond with international patterns and explanations. Finally, some conclusions from this study are discussed.

1 Summary of Results

The key findings of this thesis are summarised below:

- Mortality has fallen for children in all socioeconomic groups over the time period studied. However there is some evidence of differential declines by parental socioeconomic position as well as age, sex and ethnic group.
- There is evidence of socioeconomic gradients in injury, congenital and other causes of mortality, but not cancer.
- Younger children have stronger gradients than older children; indeed in early adolescence there is little evidence of gradients at all.
- Male children have higher mortality, but this seems to have fallen more than female mortality; however both male and female children show evidence of socioeconomic differences in mortality.
- Māori children have higher mortality rates than non-Māori children. These differences seem to persist within income groups. Both ethnic groups show evidence of socioeconomic mortality gradients in all-cause mortality.
- Between 1981 and 1991 there is evidence of an increase in relative inequality by income but no clear trend by occupational class and education.

2 Study Limitations

This section considers possible error that may mean the results are incorrect. These are divided into random error, internal validity and generalisability. However initially there is a brief discussion about the limitations of methodology in this study.

2.1 Methodological Problems with RII and SII

The advantages of the RII and SII were discussed in chapters 1 and 2. However there are some problems associated with their use in this thesis. The methodology as explained in chapter 3 involves fitting a regression line through the midpoints of mortality rates (on the y axis) of each value within each SEP measure (on the x axis). This methodology requires 4-5 values within each SEP measure (e.g. income and education were 5 level variables for the RII and SII compared to 3 level variables for the SRR and SRD). The data, however, become quite sparse at this level of stratification, particularly by ethnic group. This means that any outlying values, especially if they are at either end of the SEP spectrum, can excessively influence the regression line (i.e. high or low leverage). This was indeed the case, particularly with Māori children in the 1981 and 1986 cohorts when there were very few children with mothers with tertiary education. Hence the number of deaths in that group (which happened to be incongruously large and small in the different cohorts[4]) had undue influence over the regression line, with consequent anomalous results for the RII and SII. Even though each category is weighted by person time, this does not completely overcome the problem.

[4] Numbers cannot be presented for the individual strata for confidentiality reasons.

Additionally in the SRR and SRD there was the appearance of non-linear trends in the risk of mortality by SEP variable, and the SRD and SRR appeared to demonstrate a threshold effect of low income on mortality risk compared to medium and high income in 1981,1986 and, possibly, 1991) (e.g. Table 17 in section 2.2). This threshold effect was not consistently seen in every variable in all cohorts, but it is of concern as the regression-based measures are based on the assumption of a monotonic increase in mortality risk. However the 5 level variables which the RII and SII are calculated on are more supportive, in some cases, of the assumption of monotonic gradients than the 3 level variables (data not shown).

2.2 Precision and Validity

To understand the concept of validity in epidemiological studies Kleinbaum et al identified a hierarchy of populations in a study. At each level in the hierarchy inferences of statistical, internal and external validity are drawn (Kleinbaum et al., 1982).

The relationship between income and child mortality from this thesis can be used to illustrate this typology. The *study population* in 1981 consisted of the 679 263 children aged 0-14 on census night with an income value available. The actual population is the same as the study population, as this thesis used person time for all children who met the eligibility criteria. This is the level at which statistical inference is drawn, and where problems with precision and random error are assessed. The *target population* for this study was all children in NZ aged 0-14 on census night (886 425 children). This is larger than the actual population due to exclusions, which occurred for a variety of reasons. This is the level at which conclusions about internal validity (e.g. selection bias) are drawn. The *external population* can be thought of as potential biological (and sociological in this case) experience of a broader conceptual population

(even within a total population sample) (Rothman & Greenland, 1998). Study findings are generalised to this population. Similar hierarchies can be identified for education and occupational class.

2.2.1 Random error

Random error occurs when the estimate found in a study differs from the (unknown) true estimate (Kleinbaum et al., 1982). This random variation is an unpredictable part of experience, but the main reason for the error occurs in the process of selecting study subjects (sampling error) (Rothman & Greenland, 1998). While a total population study, such as this, may not appear to have sampling error, as it samples the entire population, the consideration of even the total New Zealand population as a sample of broader experience, as described above, means that it should be considered to have random error (in addition to potential bias) (Rothman & Greenland, 1998).

The traditional methods used to increase precision are not applicable in this study. These are increasing sample size (not possible) and making the study more efficient (also not possible). Hence while this is the biggest study of the effect of individual level SEP measures on child mortality over a continuous time period (that I know of), it still has relatively wide confidence intervals around the point estimates. This is a particular problem when interpreting trends as small changes in the point estimate over time are overshadowed by the wide confidence limits. This problem is not unique to this study, and was seen in much of the literature reviewed.

2.2.2 Internal Validity

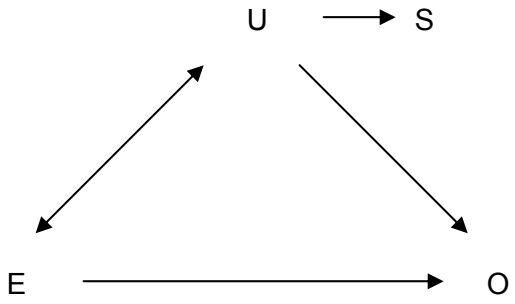
The assessment of internal validity in a study looks at the legitimacy of inferences drawn as they pertain to the members of the *study population* (Rothman & Greenland, 1998). The common types of problems with internal validity that could be present in this thesis are selection bias, confounding and misclassification. These will now be looked at individually.

2.2.2.1 Selection Bias

Selection bias occurs when the association between exposure and disease is different among subjects selected into a study and those excluded from the study. Therefore any effect measure, among selected subjects, is a mixture of the effect of both the exposure and participation on the outcome (Rothman & Greenland, 1998).

Figure 30 illustrates diagrammatically using a directional acyclic graph that in cohort studies selection bias occurs if selection (S) into a study is conditional on a probably unknown, common effect (or cause) (U) between the exposure (E) and outcome (O) (Hernan et al., In press).

Figure 30 Directional acyclic graph illustrating selection bias.



Comparing the association of exposure in the selected and excluded subjects can assess the presence of selection bias. However where selection occurs as a result of a missing exposure variable (as in this study) it is not possible to measure the association of exposure and outcome in the excluded population.

The effect of selection bias on the study estimated effects depends on both the proportion of population excluded and the difference in effect between the selected and excluded population. The results of any selection bias must be seen in the effect measures, either the rate ratio or the rate difference (or possibly in a combination of both).

Looking specifically at this study. There is no information available on the 7-9% of children alive on census night who were excluded for reasons detailed in Table 8 (in section 1.1.2). However for children who did not have a specific SEP variable (e.g. no income information available) some information is available that can be used to create sensitivity analyses to estimate the possible effect of selection bias on the effect measures.

Taking the 1981 cohort as an example. In this cohort it is known that 16% of person time was missing, the mortality rate in the missing group was 37/100

000 and the risk ratio of low/high income group was 1.4. The demographic information available on these children shows that they are equally divided among the sexes but are more likely to be in the 1-4 age group, than in the 10-14 age group. They are also more likely to be of Māori or Pacific ethnicity and have parents with lower educational qualifications (data not presented).

Table 49 examines potential effects if there was selection bias acting on the relative effect measure (the rate ratio). This suggests that quite extreme biases of the rate ratio in the missing group would be needed to affect the observed value.

Table 49 Selection bias of relative effect measure in 1981 cohort.

Person time excluded children (%)	Person time included children (%)	Effect of possible rate ratios of excluded group on overall relative effect measure			
		1.2	1.4	2.0	3.0
16	84	1.4	1.4	1.5	1.7

This uses the formula 16%*U + 84%*1.4=Y where U is unknown rate ratio and Y is unknown 'true' estimate of effect of income on child mortality.

Table 50 shows what would happen if there were bias of the absolute effect measure (standard rate difference). This sensitivity analysis assumes that there is no bias in the relative effect measure (1.4) and it models certain distributions of the income groups within the excluded group. It uses the known mortality rate in the excluded group (37/100 000 in this cohort). This sensitivity analysis shows that the SRD in all these distributions is similar to the observed SRD between low and high-income children in 1981 of 13/100 000, certainly it is within the 95% confidence limits.

Table 50 Selection bias of absolute effect measure in 1981 cohort.

Distribution of income in 'excluded' group (assumed)			Mortality rate in assumed income groups in excluded children			SRD
High Income	Medium Income	Low Income	High	Medium	Low	
33%	34%	33%	31	37	43	12
25%	35%	40%	32	38	44	13
10%	30%	60%	34	40	47	13

When interpreting *trends* selection bias would be a problem if it was differential over time i.e. the amount of selection bias varied by cohort and therefore 'explained away' changes by income over time. This seems unlikely to be a problem with income, given firstly the small effect even quite extreme rate ratios and income distributions have on the overall effect measures, and secondly the static number of children with missing information.

The changing numbers of children with no occupational class variable poses a problem. This is not actually selection bias, as the majority of these children were not eligible for an occupational class classification due to parental non-participation in the labour force. This demographic change means that analyses by occupational class are not ideal for assessing trends in inequality in children.

2.2.2.2 Misclassification Bias

Misclassification occurs as a result of measurement error in epidemiologic studies. There are two broad categories: differential, which is an error that depends on values of other variables, and non-differential, which does not depend on the value of other variables (Rothman & Greenland, 1998). As a general rule differential misclassification can either under or over estimate the effect measure and non-differential misclassification usually (but not always) biases the effect measure towards the null (Rothman & Greenland, 1998).

Misclassification of Exposure

There is potential for misclassification of the exposure of interest in this thesis, however it is difficult to quantify its extent. There are a number of mechanisms by which measurement error could be generated, at a number of stages in the research process (see Table 51).

Table 51 Origin of potential misclassification.

Error Type	Who	Example	Differential or non differential?
Respondent error	Census Respondent	Wrong income box ticked	Unknown
Transcription /Processing error	Staff at Statistics New Zealand	Miscoded occupational class	Probably non differential
Researcher error	NZCMS Researchers	Introduced misclassification through SAS coding of maternal education variable	Differential?

It is difficult to quantify the extent and possible direction of any misclassification. To look at respondent errors, reported income would need to be crosschecked with tax records (which has privacy issues). Transcription errors would require comparison of original paper census records with computer records. To model these effects sensitivity analysis could be undertaken, entering different sensitivities and specificities of a level of variable (e.g. low income) and checking how different changes in the sensitivity and specificity might affect results. That is beyond the scope of this thesis.

Misclassification may also occur in assuming that income on census night is an accurate assessment of socioeconomic position. Census information provides a snapshot and by necessity questions are short and simple, maximising space and time but (possibly) trading off accuracy. Income information, for example, is collected as one question, money earned in the last 12 months (before tax) from all sources. Bin boxes with income ranges are provided to answer the question, and information on wealth is not collected. While we can assume that census information is a reasonable indication of economic situation it is not a complete

assessment. A snapshot does not, as discussed in chapter one, consider the temporal aspects of economic position, which are important for health outcomes.

Misclassification of Outcome

There are two forms for misclassification of outcome that need to be considered in this cohort study. The first is misclassification of vital status; that is children who are deceased are considered alive because the linkage process did not match them. This problem was addressed through the linkage weighting process described in section 1.1.3. This is unlikely to contribute significantly to misclassification bias as all analyses were conducted on weighted deaths, rather than linked deaths. Any residual misclassification of vital status after weights were applied would need to be differential by SEP, which is unlikely as the majority of the initial linkage misclassification (i.e. before the weights were applied) was mainly differential by age, rurality and ethnicity, with only an additional 5-10% by small area deprivation (Fawcett et al., 2002).

Misclassification of cause of death is the second possibility that needs to be considered. The process of assigning cause of death was described in section 1.2.2. The first national review of the accuracy of reporting cause of death in children is currently being undertaken (Child and Youth Mortality Review Committee, 2004). Preliminary results suggest that there may be some misclassification around the areas of SIDS in young children and injury/suicide in older age groups, however it is difficult to determine the extent of this as yet (personal communication Peter Watson -Child and Youth Mortality Review Committee).

However, assuming there is some misclassification of cause of death, this would have to be differential by parental socioeconomic status to change the association seen in this study. In addition, due to the aggregated ICD codes

used in this study misclassification would need to be quite profound to move categories i.e. a small misclassification of exact injury cause would not cause a substantial shift in the SEP/outcome measurement in this thesis.

2.2.2.3 Confounding

The definition of confounding was discussed in section 1.3.4 in chapter one. It is unlikely that the findings in this thesis are a result of differing population structures in the different cohorts, as the effects of differing age, sex and ethnic structures of the child population over time were removed by standardising to the 1991 child population structure when looking at all-cause, and cause specific mortality. Appropriate standardisation was also applied when looking at sex, age and ethnic variations in all-cause mortality.

However other factors, both measured and unmeasured, may confound the results. For example both education and occupational class may confound income-child mortality associations. Other NZCMS studies identified factors that remain significant as predictors of an increased risk of mortality in the 0-14 age group in 1991-94 after multivariable analysis (adjusting for SEP variables). For all-cause mortality these include education (relative risk low/high 1.5 (95%CI 1.1-2.1)), car access (SRR low/high 1.9 (95%CI 1.3-2.7)), area deprivation (SRR low/high 1.8 (95%CI 1.2-2.6)) (Blakely et al., 2003). Looking specifically at injury mortality, the estimated independent effect of parental income on child injury mortality in New Zealand 1991-94 was 1.83 (95%CI 1.02-3.28) (comparing low to high income) (D'Souza, 2004). It has been argued that assessing independent effects in social epidemiology is somewhat irrelevant as all measures of SEP are inextricably linked in 'real' societal structure (Kaufman & Cooper, 1999). However assessing the independent effect of income, despite issues with over controlling for confounding by pulling causal pathway variables

into a model, could be useful given that interventions to reduce inequalities may involve some sort of income redistribution.

The likely existence of confounding does not mean that the findings of this thesis are not salient. Firstly, even though the trends are not independent estimates, the effect of confounding would need to vary between cohorts to make the trends erroneous. Secondly, confounders are also of interest in inequalities, as intervening in them may affect the outcome as well as the other factor under study.

2.2.2.4 Summary of Internal Validity

Table 52 summarises the biases identified, probable magnitude and the likely effect of each bias on point estimates in this thesis.

Table 52 Summary of internal validity.

Bias Type	Extent	Direction
Selection bias	+(?)	Unclear-Differential-?
Misclassification of exposure	++	Non differential-underestimate risk ratio
Misclassification of outcome	None or minimal	-
Confounding	+++	Inflate risk ratio

2.2.3 Generalisability

Generalisability is also known as external validity. Generalisation concerns whether the findings noted in the study population can be extrapolated or inferred to apply to a wider group of individuals (Kleinbaum et al., 1982). Generalisability is dependent on the hypothesis and type of study rather than

the study group simply being representative of the wider population (Rothman & Greenland, 1998).

I would argue that the generalisabilty of the findings of this thesis, to New Zealand children over the time period studied, is dependent on (or equivalent to) the internal validity of the study, as these cohorts consist of an almost total population sample. The number of children not represented at census is extremely small, at 1.7% in 1996, which equates to 14 000 children (Statistics New Zealand, 2002). We would not expect these children to have an experience of mortality and SEP that is not already encompassed within this study, hence the findings would be able to be generalised to these children.

However it is the ability to generalise to other times and places that is probably of interest. Is the experience of socioeconomic position and mortality in children in this study shared by future cohorts? In order to determine this we need to look at the process studied. In the case of the 2001 census cohort of New Zealand children I would suggest that the structure of social stratification which results in differential mortality risk for children has not changed greatly since 1999, so I would expect to find gradients in mortality in the more recent cohort. However the level and specifics of the gradients may be altered (through chance or real change). Generalising the findings to children in other countries is more difficult and perhaps less relevant. Evidence suggests that socioeconomic gradients in child mortality exist in the majority of developed countries where they have been studied; hence in countries where gradients in child mortality have not been measured findings similar to this study should not be unanticipated. However country specific differences (e.g. demographic and structural differences) mean it is not overly useful to apply specific point estimates to other countries.

3 Interpretation of Results

The findings of this thesis suggest that inequalities in child mortality in New Zealand are a persistent feature over the time period studied and over diverse causes of mortality. Assuming that the findings in this thesis cannot be solely explained by chance and are internally valid, is there evidence in this study to support a causal association between SEP and child mortality. What would be the pathways that explain this association? How is it that inequalities are generated and perpetuated when causal pathways to specific mortality causes are so dissimilar? This section will look at the evidence for a causal relationship between SEP and child mortality, and possible explanations.

It has been argued that while social epidemiology has proved the importance of social factors in health, it has failed to explain causal mechanisms (Kaufman & Cooper, 1999). Like many social epidemiological studies, this is an observational study, based on a natural policy experiment in New Zealand in the 1980s and 1990s. One of the criticisms of observational studies is their inability to assess whether altering the exposure can change the risk of outcome (Berkman, 2004). Ideally, to prove causation, randomised controlled trial evidence would be needed, however this is difficult to establish in social epidemiology.

Despite the long history of theory around causation in science (Susser, 2001), perhaps one of the biggest problems in this area is that contemporary paradigms of causation, such as the Bradford Hill criteria (Rothman & Greenland, 1998) and counterfactual models, fail social epidemiology as the basic principles of these theories are violated by the exposures and outcomes that are under study. For example the ‘exposure’ of SEP cannot be randomly

assigned to an individual (Kaufman & Cooper, 1999). However others argue that part of the problem is that the appropriate counterfactual model is not elucidated (Berkman, 2004). Hence interpretation of these results is hampered by these problems. However given the multitude of international observational evidence supporting the existence of socioeconomic gradients in child mortality it is worth considering this as a possible causal association. In addition, there are multiple plausible pathways as discussed in chapter one by which inequalities could arise.

3.1 Genesis of inequalities in child mortality

This section will consider the possible explanations for an association between SEP and child mortality. The first section deals with health selection and the remaining sections use the concepts introduced in the Diderichsen and Hallqvist model, discussed in chapter 2, as explanatory pathways.

3.1.1 Health Selection/ Reverse Causation

This section considers whether it is possible that apparent gradients in mortality are caused by health selection, which is a decline in (parental) SEP *prior* to death (as a result of the child's illness) making it appear as if children with lower (parental) SEP die more than higher SEP. The evidence around health selection as an explanation of socioeconomic gradients in adult mortality is contradictory, and studies that have estimated the magnitude of the effect suggest that it not the primary explanation for gradients (Chandola et al., 2003).

Overall this is unlikely to be an explanation for the findings of this thesis, for a number of reasons. Firstly, it is unlikely that injury mortality, which is the

dominant mortality cause in children, could be susceptible to health selection effects given the unanticipated nature of it. Secondly, in the causes of death where socioeconomic gradients were found (congenital and other mortality), a relationship was seen for both income and education. While income may be susceptible to health selection effects (i.e. a chronically ill child could impose financial hardship on family income) education is very unlikely to by affected by health selection. Cancer mortality did not show any socioeconomic gradient by income or education, despite the substantial financial burden a child with cancer imposes on families (Dockerty et al., 2003).

However the finding that children of farmers are less likely to die of congenital mortality than children of non-farm workers (RR 0.5 95%CI 0.28-0.92) could be a geographic health selection effect. That is, families of children with a severe congenital abnormality may move closer to tertiary hospitals in cities for treatment.

3.1.2 Differential Exposure

Is it possible that children of poorer families have higher exposure to the risk factors for the condition? This includes both direct exposure to risk and less exposure to health promoting resources that other children have access to.

3.1.2.1 Injury Mortality

Possible ways in which increased exposure to risk and decreased exposure to health promoting resources may occur in injury mortality are shown in Table 53. These are condition specific, as pathways to mortality are different for each cause.

Table 53 Possible mechanisms by which differential exposure to injury risk could occur

Injury Type	Poor children have increased exposure to:	Rich children have increased exposure to
Child Pedestrian	• High traffic flow (Roberts et al., 1995b) • Fast traffic speeds (Roberts et al., 1995b) • High density parking (Roberts et al., 1995b) • Walking to school (Roberts & Norton, 1994), • Number of roads crossed while walking to school (Roberts et al., 1996)	• Parental supervision while walking to or from school (Roberts et al., 1996) • Safe places to play (Roberts & Pless, 1995). • Car ownership (Roberts et al., 1996) • Walking school buses (Collins & Kearns, In press) • Speed bumps?
Fire	• Parental smoking (Ministry of Health, 1998) • Mobile homes (Warda et al., 1999) • Rental accommodation (Warda et al., 1999)	• Smoke alarms at home (Roberts, 1995) • Telephones (Warda et al., 1999)
Road Traffic Accident	• Older cars with fewer safety features • More accidents?	• Car seats for infants (Colgan et al., 2004) • Seatbelt use(Colgan et al., 2004)
Bicycle Accidents		• Bicycle helmet use (Farley et al., 1996)
Car Driveway		• Fenced driveways (Roberts et al., 1995a)
Drowning	• Unsafe water environments e.g. unfenced pools	

References indicate this is research proven, otherwise these are my thoughts.

3.1.2.2 Congenital Mortality

The findings in this thesis support an association between socioeconomic position and congenital mortality (e.g. education RR 1.53 95%CI 1.02-2.27). Differential exposure to risks/health promoting resources could be the mechanism for these gradients in congenital mortality. For example there is ample evidence that lower SEP is associated with less awareness and lower intake of folic acid in women, thus increasing risk of primary neural tube defects (NTD) (Centers for Disease Control and Prevention, 2002; de Walle et al., 1998; Russell et al., 1999). There is also evidence that the use of folic acid in

secondary prevention (i.e. after the birth of a child with a NTD) is inversely associated with maternal SEP (Centers for Disease Control and Prevention, 2002). This differential risk to health promoting resources may translate into increased prevalence of NTD in lower SEP and subsequently mortality gradients.

It was noted in chapter one that there is an apparent association between landfills and congenital defects (Dolk et al., 1998; Vrijheid et al., 2002). In New Zealand there is evidence that landfill sites are more likely to be located near poorer neighbourhoods (Salmond et al., 1999). This could be a pathway that inequality in congenital abnormality is embodied.

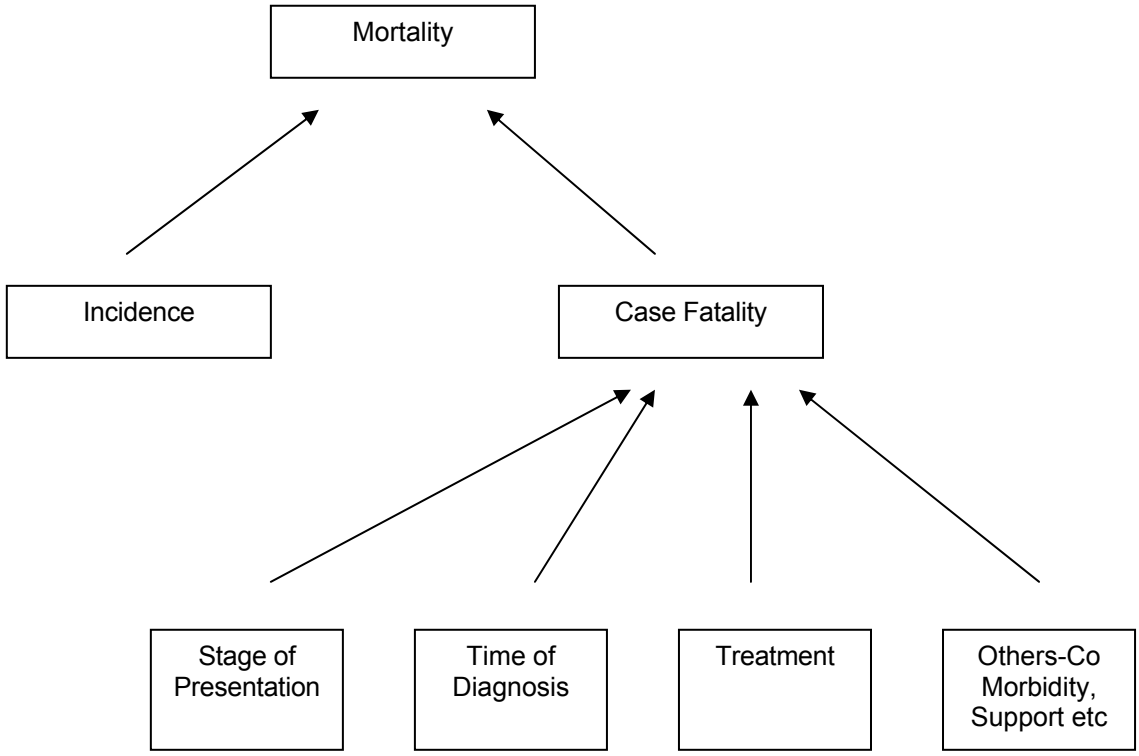
3.1.2.3 Cancer Mortality

There is evidence that there are differences in cancer incidence and survival by SEP in adults (Faggiano et al., 1997; Kogevinas & Porta, 1997), however for childhood cancer this is less clear. Socioeconomic disparities in both incidence and survival of specific leukaemia and solid tumours are not demonstrated (Dockerty et al., 2001; Menegaux et al., 2004; Murray et al., 2002; Schillinger et al., 1999; Swensen et al., 1997). This thesis supported these studies, not finding evidence of socioeconomic gradients in child cancer mortality (income SRR 0.84 95% CI 0.60-1.17). However it is important to remember that childhood cancer covers different cancers with different etiologic pathways. It is possible, although unlikely given the conflicting international evidence, that this grouping obscures cancer specific socioeconomic gradients.

Some studies reviewed did show socioeconomic gradients in cancer mortality (Blakely et al., 2003; Nelson, 1992; Ostberg, 1992). Figure 31 identifies (simplistically) the points at which socioeconomic gradients could be generated

in cancer mortality. It is interesting to look at the studies that did show a relationship between cancer mortality and SEP to determine if there is evidence whether these pathways are relevant. Blakely et al studied the 1991 cohort of children in the NZCMS, which contained fewer deaths than the grouped results in this thesis (Blakely et al., 2003). When looking at the results for that particular cohort (Figure 16 in section 2.3.2 in chapter 4) it is apparent that this is an aberrant finding, not seen in all other cohorts, illustrating the effect of chance on those findings. Ostberg demonstrated a small relationship, only for boys, which may have also been due to chance (Ostberg, 1992). The third study was a U.S.A. study of extremely deprived children (Nelson, 1992). Given the private nature of healthcare delivery in the U.S.A., the differences by SEP could possibly be due to differential case fatality because of inability to access healthcare for cancer treatment

Figure 31 Possible pathways to socioeconomic inequalities in child cancer mortality.



Possibly the reason that there is no consistent convincing evidence of SEP gradients in child cancer mortality is that the risk factors are poorly understood. The few known risk factors for childhood cancers are highly toxic substances such as ionising radiation and chemotherapeutic agents (Pizzo & Poplack, 2002). These risk exposures cause very few child cancers, partly because exposure is tightly controlled. In adult cancer, as less (immediately) toxic risk factors have been elucidated, some, such as tobacco, have become progressively more confined to those in lower SEP (Tomatis, 1997).

3.1.2.4 Other Mortality

Disease related mortality has complex paths. One possible mechanism for SEP gradients may be the increased exposure poor children have to household

overcrowding. Overcrowding is associated with increased risk of infectious diseases such as rheumatic fever, meningococcal disease, TB and pneumonia as well as conditions such as asthma (Baker et al., 2003). An increased incidence could possibly translate into increased mortality. It is important to note that exposures to risk factors, such as crowding, do not occur in isolation. Children of poorer households probably have multiple exposures to risk factors, such as tobacco, crowding, and unsafe physical environments, which may interact with each other.

One of the findings in this study was that children of farm workers aged 10-14 have higher risk of injury mortality than children of non-farmers (1996 RR 1.8 95%CI 1.1-3.2). This phenomenon was only statistically significant in 1996 (although the point estimate was suggestive of a similar effect in the early cohorts the confidence limits included one). This may be an 'exposure to risk' phenomenon. It is possible that children of farmers are exposed to more physical risks, such as dangerous machinery and the use of farm vehicles, than their non-farming compatriots. The 10-14 age group may be particularly affected due to the increasing independence of youth. However the other possibility is differential experience, as children who live on farms are more geographically distanced from trauma services, and therefore may be more likely to die of their injury before receiving medical attention.

3.1.3 Differential Experience of Risk Factor or Condition

There is also the question of whether, once a child is exposed to a risk factor or condition, they then experience it differently depending on their SEP, leading to a different mortality outcome. This may happen in isolation from the exposure (i.e. the same likelihood of exposure but more likely to die) to or in tandem with it (i.e. more likely to be exposed and more likely to die). The factors that influence this have been crudely divided into intrinsic (related to the child) and

extrinsic (not related to the child). While the possibility of differential experience of a condition by SEP needs to be examined, it is unlikely that this contributes substantially to the generation of inequalities in child mortality in New Zealand. This is because the main cause of mortality, injury, is likely to be due to differential incidence rather than differential emergency treatment once in hospital. However it may be important in medical causes of mortality.

3.1.3.1 Intrinsic factors

Intrinsic factors belong to the child, although they may be shaped by environment. This can include host susceptibility, e.g. poor nutrition may make the progress of an infection worse once acquired. There is some evidence that nutrition including vitamin adequacy may vary by SEP in New Zealand children (Ministry of Health, 2003a). Equally there is some evidence from the developing world that susceptibility to disease in children can be mediated by nutritional status (Brooks et al., 2004).

Differential experience of water environments could affect drowning mortality. Children of lower SEP are less likely to know how to swim by age 9 (Langley & Silva, 1986). So for the same level of exposure to unsafe water environments, children of lower SEP may die more frequently because they are unable to swim.

3.1.3.2 Extrinsic factors

The main extrinsic factor that may affect children's experience of a risk factor or condition is health services and their response to children. There is increasing interest in the role of health services in differential mortality outcomes in adult

health (Jeffreys et al., submitted; Kogevinas & Porta, 1997). Although this is probably a minor effect in children, given the relatively smaller contribution that medical causes make to child mortality, the possibility needs to be examined.

It is plausible that there may be differential treatment of children with chronic medical conditions by SEP, which could affect mortality. There is not really much evidence in the area, however studies of asthma have suggested that there are socioeconomic differences in asthma diagnosis (Ng Man Kwong et al., 2002) and in New Zealand there are thought to be ethnic differences in asthma treatment (Mitchell, 1991). This suggests that the health system may not be benign in the genesis of ethnic inequalities in health, and therefore it would warrant investigation as to whether this also applied to SEP.

3.1.4 Structural Determinism

The above section has demonstrated that differential exposure and experience of risk are plausible pathways for the generation of socioeconomic gradients in child mortality. However as can be seen in the original model by Diderichson and Hallqvist (Laflamme & Engstrom, 2002), the distribution of risk factors and the experience of risk do not just occur at the individual level. They occur in the context of societal placement of risk and opportunity. To ignore this is to miss opportunities to intervene to reduce inequalities.

While the effect of massive societal fragmentation (such as war) is an obvious example of the effects of structural determinants of mortality, most influences are not as dramatic as this (Men et al., 2003; Murray & Chen, 1993; Spiegel & Salama, 2000). Most influence on individuals comes from the configuration of policies and practices in a society. Changes in society are often incremental, although the net results can be quite dramatic, as was seen in Table 5 in

section 3.1. This section does not purport to list all the ways that wider society influences risk of child mortality, but some examples from New Zealand will be provided. It should be noted that proving causation rather than association is even more difficult at this level of removal from the child (i.e. it is difficult to prove the level of family income is causally associated with child mortality, let alone the determinants of income).

The broad range of policies that governmental structures enact and enforce governs family social experience. This includes taxation level (e.g. flat or progressive), and benefit distribution. The 1991 New Zealand benefit cuts differentially impacted on families with children, decreasing income more for parents on the unemployment benefit than working parents (Stephens, 1999). This change in income would have substantially altered the ability of parents to pay for the necessities of life for children, and this was reflected in the subsequent increase in the use of food banks (Stephens, 1999). Presumably this may also have impacted on the ability of parents to pay for the material goods that promote safety, such as car seats.

Policy can affect the differential exposure of children to risks. In Table 53 many of the factors identified as possibly generating gradients are under direct governmental control, mainly through transport and safety policy, e.g. traffic speed is controlled by legislated and enforced speed restrictions through residential and school areas. The distribution of folic acid is determined by individual knowledge and ability to access and pay for supplements and/or food high in folic acid. However a societal choice has been made not to fortify foods, although this may be under policy review (Ministry of Health, 2003b).

Neighbourhood level factors may be of particular importance to children; in one study area of residence was assessed to have an effect independent of parental SEP for all-cause mortality, with a RR 1.8 (95%CI 1.2-2.6) comparing the highest deprivation quintile with the lowest (Blakely et al., 2003). While some of

this apparent effect may be due to residual confounding by individual SEP, there is increasing evidence that place of residence has a small, but significant, effect on health above and beyond individual SEP (Macintyre et al., 2002). The actual neighbourhood factors that affect child health have not been identified, but consideration should be given to school and park placement, types of roads, and the presence of toxic materials such as landfills. Many of these factors are under the control of national and local body legislation.

3.2 Trends

The hypothesis of this thesis was that child mortality inequality would have changed (most likely increased) in New Zealand between 1981 and 1999. This hypothesis was based on evidence of an increase in inequality in adults over this time period, and that the social determinants of health have changed for New Zealand children with an increase in social inequality (Ajwani et al., 2003; University of Otago & Ministry of Health, 2004) (see Table 5 and Table 6 in chapter 2).

However time trends in child mortality inequality are difficult to interpret. Firstly, the (relatively) small number of deaths means that confidence intervals are wide, lowering the precision of the point estimates; this is particularly problematic when interpreting small changes in time trends of relative and absolute inequalities. Secondly, intrinsic features of the measures of socioeconomic position used in this thesis mean that trends are inconsistent (although given the different social meaning of each measure complete similarity should not be anticipated). For example the measurement and meaning of income was relatively stable over time, in contrast to education where large secular changes in distribution and social meaning were seen. These are probably only partly overcome by the use of regression-based measures. It is difficult to be confident that any trends seen by occupational

class are an accurate representation of overall trends in socioeconomic inequalities in child mortality in New Zealand, due to the large increase in the number of children with no parent(s) in the workforce (from 11% in the 1981 cohort to 21% in 1996 cohort). Finally small or large numbers in some of the five level strata used in calculating the RII and SII may cause undue instability or even leverage of the estimates. This was not an issue in the income variable, as there was equal distribution of children in each tertile or quintile.

Despite these problems it is worth discussing some of the findings, as some interesting points arise out of the data from these cohorts and in anticipation of data from future cohorts. It is also worth noting that limitations experienced in this study are likely to be replicated in future New Zealand and international studies.

By income all-cause mortality trends showed an increase in relative inequality over time. This was of borderline statistical significance with p for trend of 0.06, but is more persuasive when considered in context of the stability of the measure of SEP (i.e. income) and societal changes in income since 1981. Changes in relative income (an increase in poverty), absolute income (a decline in the absolute income of some families) and income inequality (a rise in the Gini coefficient) were all experienced over this time period. Hence if one were to expect a change in inequalities, then one might expect to see them by income. Arguing against this is the study by Bremberg, which considered trends in inequality over a time period where incomes declined rapidly and substantially in Sweden (Bremberg, 2003). He did not find evidence of increasing inequality, but the exposure was area level socioeconomic factors not individual income introducing substantial misclassification of income as the exposure.

By education there was no clear (linear) trend, although it is possible that data from future cohorts may make any emerging trends clearer. It is interesting to consider whether changes in distribution of maternal education in younger

(childbearing) cohorts may mean that inequality by maternal education appears to decline reflecting the increase in homogeneity of this 'exposure'. Although it has also been argued that changing distribution makes inequalities more obvious (Arntzen et al., 2004).

The majority of international studies are by occupational class. As mentioned previously there was no clear trend in increasing or decreasing inequality in New Zealand by occupational class. Parental participation in the labour force is probably a more important determinant of child mortality then actual position within the labour force.

There idea of a commonality of process determining adult inequality trends has been discussed (Koskinen, 2003; Leon, 1998, 2001). This is due to a broad trend of increasing socioeconomic inequality (despite cause specific inequality trends being diverse) (Mackenbach et al., 2003; Martikainen et al., 2001; Pappas et al., 1993). This dialogue is not possible with child literature due to the diverse methodology used in assessing trends (as discussed in section 1.1 in chapter 2). For example a broad trend of increasing relative inequality in all-cause child mortality over time was seen some studies (Botting, 1997; DiLiberti, 2000), but not others (Bremberg, 2003; Ostberg, 1992). In another study differing trends were found by sex (Turrell & Mathers, 2001). Socioeconomic position in these studies is variously measured by occupational class, phone access, area level SEP, (different) occupational class and (different) area level SEP.

Trends in socioeconomic gradients in all cause child mortality are mainly driven by injury mortality. Injury mortality has declined overall, but (by income) an increase in relative inequality was seen due to the preservation of absolute differences (tests for trend were not significant, most likely due to non linear change). These trends are consistent with some of the international child injury

mortality literature reviewed (Roberts & Power, 1996; Turrell & Mathers, 2001), although not others (Morrison et al., 1999a).

The preservation of absolute differences between groups is thought to be due to differential declines in mortality among the socioeconomic groups. Some researchers consider this to be a possible explanation as to why inequalities might increase over time (Haines, 1995; Roberts & Power, 1996). In this thesis greater decline in mortality among the high-income group (56%) compared to the low-income group (38%) was seen in injury mortality. However 'differential decline' is not an explanation of why groups that have the most 'need' (as defined by the highest mortality rate), are not able to access health promoting materials at the same rate, let alone a higher rate, than those who are better off.

The inverse equity hypothesis one theory of why differential declines and an increase in inequality may occur. This theory suggests that as a health intervention moves through the population increasing relative inequality is inevitable due to quicker uptake of the intervention by higher socioeconomic groups. It was originally illustrated in an example of child morbidity and mortality inequities in Brazil (Victora et al., 2000).

It seems unlikely that one injury prevention programme could cause the different rates of decline in injury mortality by SEP group; however these results could perhaps arise from many programmes that systematically favour children of richer parents. Another option is that the direct (material) effects of income could be causing this pattern. Children of wealthier parents are able to afford access to the physical resources that decrease the risk of injury mortality. Ability to pay means that benefits (e.g. safe cars) filter down to poorer children at a slower rate compared to richer children.

The trends in inequality by income in this thesis are not statistically incontrovertible. They represent one period of time, and it is possible that as time continues different trends in inequalities will emerge. Monitoring trends by income and education in the future will add to our understanding.

3.3 Heterogeneity by age, sex and ethnicity

Some of the interesting findings in this thesis were around the differences in socioeconomic gradients in mortality by sex, age and ethnicity.

3.3.1 Age differences

This thesis found that there were differences in the existence of socioeconomic gradients in mortality by age group. For income and maternal education, in all cohorts except 1996, the steepest gradients (both relative and absolute) were found in the 1-4 age group, followed by more attenuated gradients in the 5-9 age group. The 10-14 age group showed little evidence of relative or absolute inequality in the first three cohorts. In 1996 relative inequality in all age groups converged (due to the emergence of socioeconomic gradients in the10-14 age group), although absolute inequality was different (see Table 25 and Table 38in chapter 4). It is unclear whether the emergence of socioeconomic gradients in the10-14 age group in the 1996 cohort is chance or the emergence of a new trend in that age group. For occupational class no clear pattern was seen (Table 67 in appendix 2).

The findings in the first three cohorts are consistent with West's hypothesis of flattening of inequalities in youth (Macintyre & West, 1991; West, 1988, 1997). The international evidence highlighted in the literature review is inconsistent around this theory (see Table 3 in section 2.2). However these studies had

different measures of SEP and precision problems, with overlapping confidence intervals. Precision was also a problem in this thesis, but as already stated a reasonable consistent pattern emerged from both education and income.

West speculated that the flattening in youth was due the effects of age and peer group culture associated with that age cutting across social class differences to equalise mortality. He loosely defines youth as 10-19 (although noting this is a shifting definition), however West's own data suggest the emergence of socioeconomic mortality gradients in 16-19 year olds (West, 1997). This is consistent with other data from the NZCMS (Blakely et al., 2003).

The criticism of West's hypothesis is that the data excluded the group of children who did not have an occupational class (Judge & Benzeval, 1993). This thesis does not have that problem; the flattening of gradients was seen in both income and education and, in fact, was less clear in the occupational class measure.

The converse of West's question is why younger children would have steeper socioeconomic gradients in mortality than older children. Is it possible that parental SEP 'matters' more for mortality at younger ages? Is the model of receptivity to health messages, as measured by maternal education, of more relevance when a child is under the predominant influence of the parent? Does school act to mitigate the effect of parental SEP?

This thesis also found that over time children aged 10-14 did not have the same declines in mortality compared to younger children, with low and middle-income children having little or no decline in mortality but the high-income group mortality declining by 41%. This is in contrast to younger children, when all income groups had declines in mortality. The reason for these differences remains unclear.

3.3.2 Ethnicity

The main finding of differences by ethnic group was that Māori children have higher mortality in (almost) all income groups compared to non-Māori/non-Pacific children. Both groups have had a decline in mortality, although the absolute differences have remained. The poorer position of indigenous or minority children compared to children of the dominant/colonial culture is not unique to New Zealand. Aboriginal children in Australia and black American children have higher mortality than their peers (Alessandri et al., 1999; Singh & Yu, 1996). This thesis demonstrates that these differences are unlikely to be accounted for by poorer socioeconomic profile of Māori children (as a group) (Statistics New Zealand, 1999a). Comparing high-income children of both ethnic groups, Māori children still had higher mortality (although confidence intervals are wide).

Routine data indicate that Māori children have higher rates of injury deaths, respiratory and infectious disease mortality compared to non-Māori/non-Pacific children (Ministry of Health, 1998). It may be possible that at the same level of SEP Māori children are exposed to more risks of infectious and respiratory disease and injury risk factors than non-Māori/non-Pacific children. There is some evidence to support this hypothesis. For example in families whose main source of income is the domestic purposes benefit Māori households are more likely to be crowded than non-Māori/non-Pacific households (46% and 32% respectively) (Statistics New Zealand, 2003). Māori children are likely to have higher exposure to passive smoking, even when socioeconomic position is considered (Crampton et al., 2000). Hence there may be an additive effect of risk factors that measurement of SEP does not capture.

Another explanation is that the measures used are not capturing the true SEP of Māori children, i.e. they may be more likely to have been exposed to prolonged periods of poverty and thus be at an increased risk of mortality compared to non-Māori/non-Pacific children in the same current SEP group. For example Roberts reported that within the same social class Māori and Pacific parents in Auckland had fewer resources such as cars or house tenure compared to non-Māori/non-Pacific (Roberts, 1994). However the underlying structural question of why Māori children occupy a less advantaged place in society compared to non-Māori/non-Pacific needs to be addressed as well.

3.3.3 Sex

The main finding by sex was the higher rates of deaths that boys experienced. Figure 7 in section 2.6 shows that sex differences have been preserved while mortality rates have declined in New Zealand.

This thesis is suggestive of the existence of socioeconomic gradients within both sexes. Point estimates indicated 30-50% excess mortality for female children of poorer families and a 40-80% excess mortality for boys of poorer families, although confidence intervals include 1 for many of these values. International studies have not shown consistent patterns on whether the effect measures are stronger for boys or girls (Botting, 1997; Ostberg, 1992; Vagero & Ostberg, 1989). The point estimates, in these studies, varied with age and time period, suggesting gradients by sex are dynamic and affected by falling mortality and causal dominance of mortality (assuming variations are due to more than just chance).

For children in these cohorts the majority of sex differences in mortality come from the increased male rates of injury mortality and other mortality (which

includes SIDS, violent deaths and suicide). The most likely explanation of differences is a combination of biological and acquired risk, although there may be gendered differences in healthcare access if parents respond to children differently by sex (Emslie et al., 1999). It is possible that different causes of death have different emphasis on each of these explanations.

There is evidence of different trends in socioeconomic inequality over time between the sexes. For boys, by income, the absolute differences between income groups were retained while relative inequalities increased (RII 2.5 in 1996 compared to 1.5 in 1981 p-trend 0.05). For girls the absolute differences have declined and relative differences have diminished slightly also (although all confidence intervals on RII for girls include 1). These divergent trends could be due to chance, however if they are true this suggests that the effect of SEP on the sexes is different.

4 Conclusions and Implications

This study has shown that child mortality inequalities by socioeconomic position exist in New Zealand. These inequalities are consistent across most causes of death, by age group, ethnicity and sex. There is some evidence that by income socioeconomic inequalities increased between 1981 and 1999, when dramatic social and economic changes occurred in New Zealand.

The pathways to disease and death from socioeconomic position are related to the differential exposure to risk that low SEP children experience. These exposures spread across all dimensions of health (not just those exposures that lead to mortality) and there is increasing evidence that socioeconomic disadvantage in childhood leads to the accumulation of adult health disadvantage thus embodying privilege in certain groups (Galobardes et al., 2004; Jackson et al., 2004; Poulton et al., 2002).

Despite their pervasiveness, inequalities are not inevitable; they are the result of deliberate decisions (Woodward & Kawachi, 2000). These decisions may be acts of omission as well as conscious choices. These socioeconomic inequalities in mortality are unjust, and their description is simply a foundation for action.

There are many frameworks to understand the genesis of inequalities and to conceptualise and conduct action to eliminate them. These include (among others) the model by Diderichson and Hallqvist used in this thesis (Laflamme & Diderichsen, 2000), the Mackenbach model used by the Ministry of Health in New Zealand (Ministry of Health, 2002b), the Ottawa Charter (World Health Organization, 1986), inverse equity theory (Victora et al., 2000),

population/targeted interventions (Rose, 2001), equity lens (Public Health Consultancy and Te Ropu Rangahau Hauora a Eru Pomare, 2004), and health impact assessment (Public Health Advisory Committee, 2004). Each has strengths and limitations. Each would need to be focused on children.

Despite the plethora of strategies and frameworks, actual solutions to reducing inequalities are elusive, as the effectiveness of most population public health interventions (let alone action to reduce inequalities) remains unclear (Roberts, 2004). There are signs that evaluation of public health interventions is improving (DiGuiseppi et al., 2002; Duperrex et al., 2002; Tester et al., 2004; Toroyan et al., 2003), and any interventions specifically aimed at reducing inequalities also need to be monitored and evaluated appropriately. There are moral (as well as financial) obligations to do this.

There is no single solution to eliminate inequalities; action is likely to be needed in multiple places on the Diderichson and Hallqvist model (Laflamme & Diderichsen, 2000). For example even the eradication of child poverty is unlikely to completely remove injury inequalities (D'Souza, 2004). Injury mortality needs particular focus, as it accounts for the majority of deaths in children in New Zealand and generates most of the inequalities. Solutions to this may be unpalatable (to adults), as they are likely to include environmental modification of traffic and transport.

Finally, this thesis has highlighted that most of the factors that determine child mortality inequalities are not in the jurisdiction of the health sector. Therefore the most important aspect to ensure success in eliminating inequalities is population and government commitment.

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Appendix 1 - Cohort Weighting

Previous weighting had been done on the 0-14 age group, where as the analyses that this thesis was concerned with involved finer age bands and different causes of death as reflected in child mortality trends. Hence investigation of how well the current weights based on age and ethnicity, age and deprivation, age and ICD code was undertaken.

The results showed that while the actual number of deaths and the weighted number of deaths were in accordance for broad categories (see Table 54), there were some differences to these numbers once finer categories were analysed (see Table 55). This was of concern as the small number of deaths in some cells for children mean that inaccuracy of these numbers might alter results and their interpretation significantly.

Table 54 Original weighting for all deaths ages 0-14 on census night.

Cohort	Total deaths	Base Weight	Age Ethnicity Weight	Deprivation Weight	ICD Weight
1981	1092	1092	1092	1092	1083
1986	1020	1020	1017	1017	1017
1991	831	831	831	828	825
1996	810	810	807	807	807

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Table 55 Original weighting for 1986 when broken down by age, sex and cause of death.

Sex	Age at Census	Cause of Death	Total deaths	Base Weight	Age Ethnicity Weight	Deprivation Weight	ICD Weight
Males	0 yrs	Injury	27	18	21	21	21
		Congenital	21	18	21	21	18
		Cancer	6	6	6	6	6
		Other Deaths	60	60	63	60	57
	1-4 yrs	Injury	78	69	75	72	81
		Congenital	9	12	12	12	12
		Cancer	24	21	21	21	21
		Other Deaths	45	45	48	51	48
	5-9 yrs	Injury	54	57	60	57	54
		Congenital	6	6	6	6	6
		Cancer	24	24	24	24	21
		Other Deaths	15	12	12	9	15
	10-14 yrs	Injury	120	135	126	123	120
		Congenital	12	9	9	9	12
		Cancer	33	36	33	36	36
		Other Deaths	90	90	84	84	90
			624	**618**	**621**	**612**	**618**
Females	0 yrs	Injury	18	15	15	15	15
		Congenital	15	15	15	15	12
		Cancer	6	6	6	6	6
		Other Deaths	42	48	45	45	42
	1-4 yrs	Injury	42	42	39	42	45
		Congenital	18	18	18	18	18
		Cancer	18	21	21	21	18
		Other Deaths	21	21	18	18	18
	5-9 yrs	Injury	27	27	24	27	27
		Congenital	9	9	9	9	9
		Cancer	12	12	12	12	12
		Other Deaths	24	30	27	27	27
	10-14 yrs	Injury	72	72	75	72	72
		Congenital	9	15	15	15	15
		Cancer	24	21	21	21	21
		Other Deaths	51	45	48	48	51
			408	**417**	**408**	**411**	**408**
			1032	***1035***	***1029***	***1023***	***1026***

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

A process of re-weighting child deaths was undertaken, which is summarized below. For more detail on this process see Fawcett et al (Fawcett et al., 2002).

Step 1-Creating the Base Weight

A base weight was created on files containing death records that were submitted for the linkage process during NZCMS cohort formation. Information contained on this file included demographic details, causes of death and a flag indicating whether they were linked during the data matching process. This weight was specific to children who were between 0-14 on census night.

Each cohort was weighted separately. Stratifying the deaths into year, age, sex and ethnic group and then, within each of these cells, dividing the total deaths by the linked deaths created a base weight. A simple example of this is if there were 13 deaths in one strata (i.e. 1981, Māori boys aged 5-9 at census) but only 10 of them had been linked then the base weight would be 1.3 (13/10).

There was a process of merging some of the above cells at this stage. Merging only occurred if there were less than 3 children in each cell and/or less than 66% linkage. Cells were merged in a prioritized fashion: Māori and Pacific ethnic groups were combined, and then if the above rule was not met they were merged with the "other" ethnic group. Sex had to be merged twice, for children aged 5-9 years who died of cancer in 1981 and 1986. Cohort and age were never merged.

Step 2 Scaling the Base Weight for Cause of Death

This base weight was attached to the "linked" children and then they were re-stratified according year, age, sex and ethnic group **and** cause of death (injury, congenital mortality, cancer and other). The total of these weighted deaths was then compared to the known total of deaths in each strata to determine how well this base weight performed on cause of death. Each child who had died and was linked would have a value of more than 1 and this total was compared to the known total within each strata.

The known sum was then divided by the weighted sum to scale the weight slightly for cause of death. This scaled weight was then attached back to the linked children and the base weight dropped.

An example of this is in 1991 females other ethnic group aged 1-4 injury deaths if there were 20 known deaths, of which 15 were linked and the base weight attached to those linked children summed to 19 then the base weight for individuals in that strata was scaled by 1.05 (20/19).

Step 3 Attaching weights to original file by SNZ

These weights were then attached back to individuals on the cohorts by Statistics New Zealand staff, thus preserving confidentiality.

Step 4 Weighting down original cohort

Once the weights were attached to the cohorts the remainder of the cohort were then weighted down slightly. This was to remove the effect of the unlinked children, so that the total of the weighted deaths and those that were registered as alive at the end of follow up equaled the known number of children at census.

Step 5 Checking the new weight

It was possible to determine how well the new weight performed at different levels of detail. Table 56 shows the new weight in comparison to the actual numbers of deaths. It functioned well for breakdowns by age, sex and cause of death. However when breaking the figures down by ethnic group, the weights show more differences and therefore become less robust (see Table 57).

Table 56 Comparison of total and weighted deaths with new weight by sex, age and cause of death.

			Cohort							
			1981		1986		1991		1996	
Sex	Age at Census	Cause of Death	Total Deaths	Weighted Deaths	Total Deaths	Weighted Deaths	Total Deaths	Weighted Deaths	Total Deaths	Weighted Deaths
Males	0 yrs	Injury	33	33	27	27	15	15	21	21
		Congenital	12	12	21	21	18	18	18	18
		Cancer	6	6	6	6	6	6	6	6
		Other Deaths	60	60	60	60	69	69	45	45
	1-4 yrs	Injury	75	75	78	78	57	57	57	57
		Congenital	15	15	12	12	9	9	12	12
		Cancer	27	27	24	24	15	15	24	24
		Other Deaths	45	45	45	45	33	33	27	27
	5-9 yrs	Injury	69	69	54	54	33	33	42	42
		Congenital	9	9	6	6	12	12	6	6
		Cancer	24	24	24	24	21	21	21	21
		Other Deaths	27	27	12	12	24	24	15	15
	10-14 yrs	Injury	147	147	120	120	93	93	81	81
		Congenital	6	6	12	12	6	6	6	6
		Cancer	30	30	36	36	18	18	21	21
		Other Deaths	78	78	90	90	66	66	75	75
Females	0 yrs	Injury	15	15	18	18	18	18	15	15
		Congenital	12	12	12	12	9	9	15	15
		Cancer	6	6	6	6	6	6	6	6
		Other Deaths	54	54	45	45	42	42	27	27
	1-4 yrs	Injury	51	51	42	42	30	30	33	33
		Congenital	18	18	18	18	15	15	9	9
		Cancer	15	15	18	18	15	15	18	18
		Other Deaths	33	33	18	18	30	30	30	30
	5-9 yrs	Injury	39	39	27	27	30	30	27	27
		Congenital	6	6	9	9	9	9	6	6
		Cancer	15	15	12	12	12	12	15	15
		Other Deaths	27	27	27	27	24	24	15	15
	10-14 yrs	Injury	57	57	72	72	45	45	48	48
		Congenital	9	9	9	9	6	6	9	9
		Cancer	21	21	24	24	12	12	21	21
		Other Deaths	39	39	51	51	33	33	51	51

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Table 57 Comparison of total and weighted deaths with new weight by sex, age and ethnic group.

			Cohort							
			1981		1986		1991		1996	
Sex	Age at Census	Ethnic Group	Total Deaths	Weighted Deaths	Total Deaths	Weighted Deaths	Total Deaths	Weighted Deaths	Total Deaths	Weighted Deaths
Males	0 yrs	Māori	21	21	18	15	18	18	33	33
		Pacific People	6	6	6	9	12	12	9	9
		Other	90	90	84	87	78	78	42	42
	1-4 yrs	Māori	21	21	15	15	6	6	48	48
		Pacific People	6	6	6	6	9	9	9	9
		Other	138	138	135	135	99	99	60	60
	5-9 yrs	Māori	9	9	9	9	12	12	21	21
		Pacific People	6	6	6	6	6	6	6	6
		Other	114	117	84	84	72	72	54	54
	10-14 yrs	Māori	27	27	30	30	18	12	69	54
		Pacific People	6	6	6	6	6	6	15	12
		Other	225	225	222	222	162	168	105	120
Females	0 yrs	Māori	15	15	9	9	12	12	27	27
		Pacific People	6	6	6	6	6	6	6	6
		Other	69	69	72	72	60	60	27	27
	1-4 yrs	Māori	12	12	12	12	9	9	33	36
		Pacific People	6	6	9	9	6	6	6	6
		Other	108	108	75	75	78	78	48	48
	5-9 yrs	Māori	12	12	6	6	9	9	12	12
		Pacific People	6	6	6	6	6	6	6	6
		Other	72	72	63	66	60	60	42	42
	10-14 yrs	Māori	15	15	18	18	9	9	42	45
		Pacific People	6	6	6	6	9	9	9	9
		Other	111	111	132	132	78	78	78	78

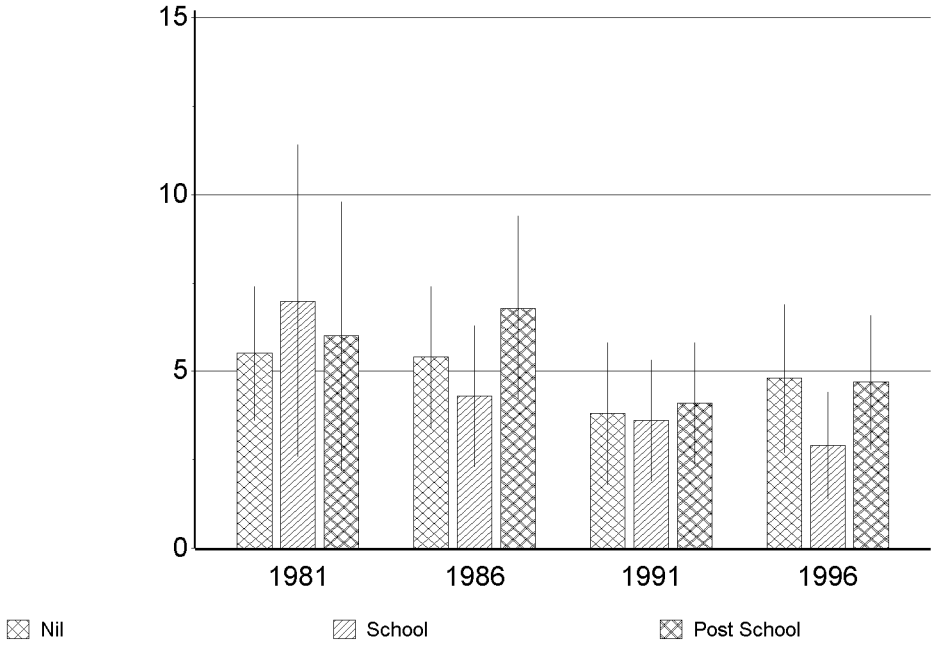
Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Appendix 2 - Further Results

4.1 Figures and Tables from Maternal Education Results

4.1.1 Cancer

Figure 32 Cancer mortality by maternal education both sexes, ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years

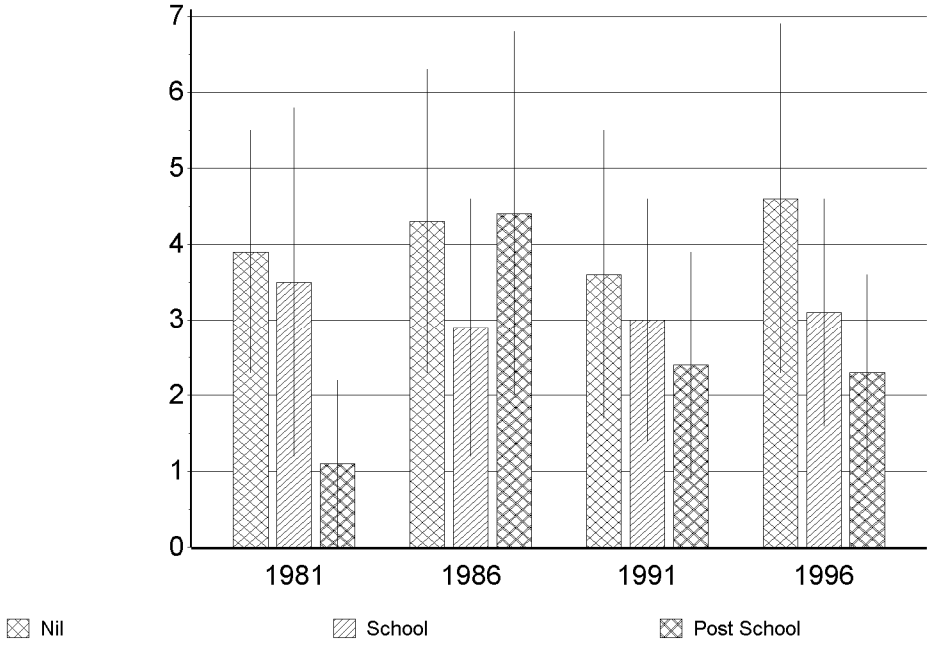
Table 58 All years cancer mortality rates per 100 000 person years SRR and SRD, by maternal education both sexes ages 1-14 years.

Education Qualification	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Nil	153	5.0	(4.0 - 6.0)	0.95	(0.71 - 1.28)	-0.3	(-1.8 - 1.3)
School	96	4.1	(3.1 - 5.2)	0.79	(0.57 - 1.09)	-1.1	(-2.6 - 0.4)
Post School	120	5.2	(4.1 - 6.4)	1.00		0.0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.1.2 Congenital

Figure 33 Congenital mortality by maternal education both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.

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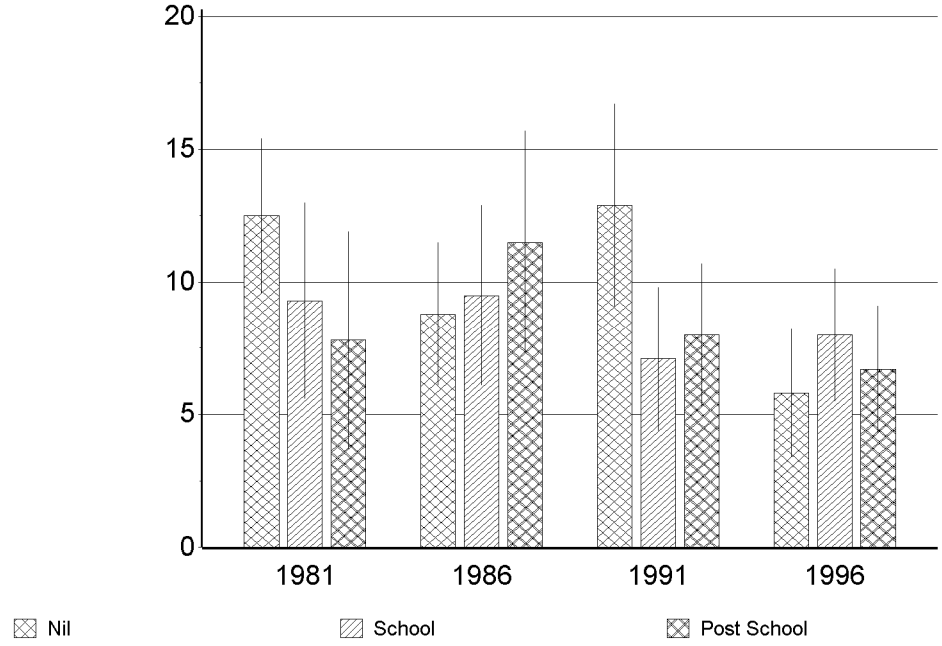
Table All years congenital mortality rates per 100 000 person years SRR and SRD, by maternal education both sexes ages 1-14 years.

Education Qualification	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Nil	99	4.0	(3.1 - 4.9)	1.53	(1.03 - 2.27)	1.4	(0.1 - 2.6)
School	69	3.1	(2.2 - 3.9)	1.17	(0.77 - 1.79)	0.4	(-0.7 - 1.6)
Post School	60	2.6	(1.8 - 3.4)	1.00		0.0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.1.3 Other

Figure 34 Other mortality by maternal education both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years

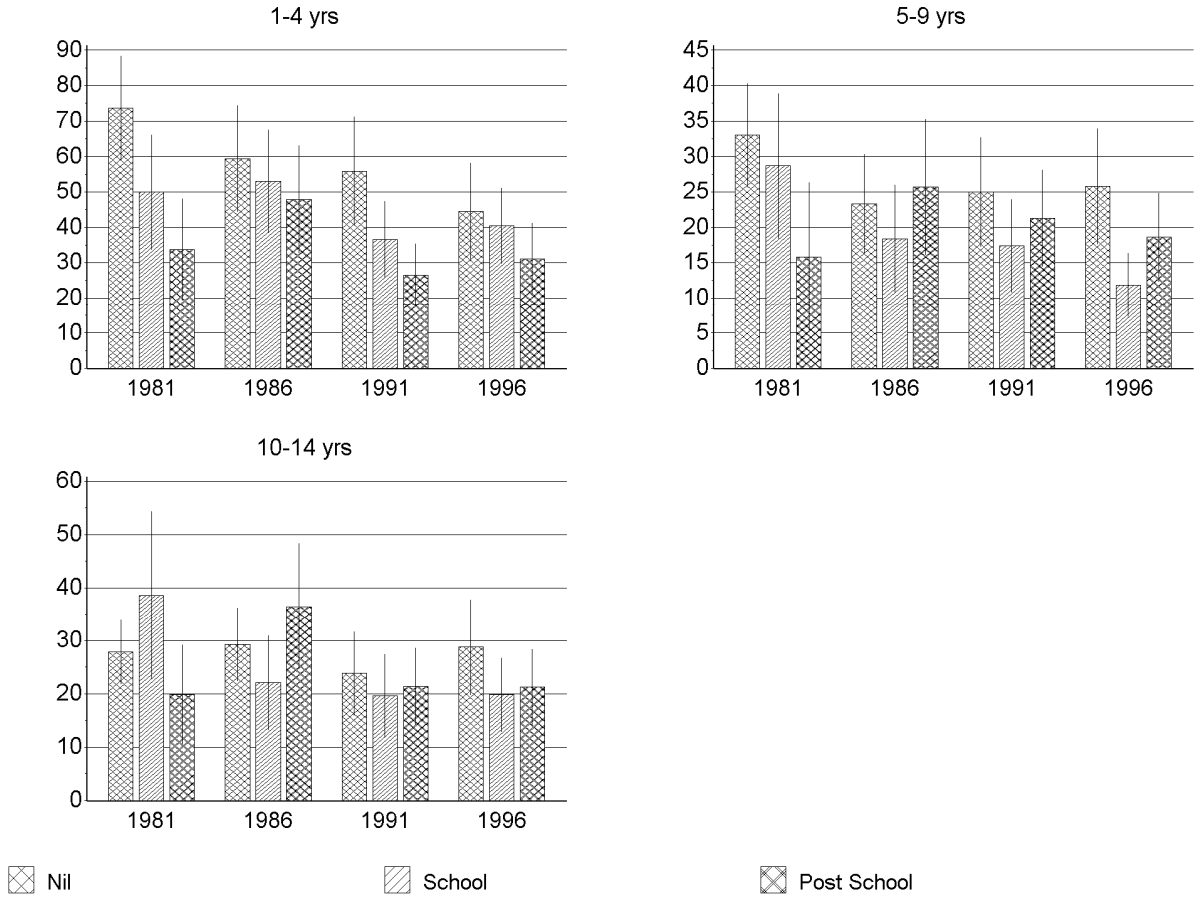
Table 59 All years other mortality rates per 100 000 person years SRR and SRD, by maternal education both sexes ages 1-14 years.

Education Qualification	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Nil	291	10.2	(8.7 - 11.7)	1.25	(0.98 - 1.58)	2.0	(-0.1 - 4.2)
School	183	8.4	(6.9 - 9.9)	1.02	(0.79 - 1.32)	0.2	(-2.0 - 2.3)
Post School	177	8.2	(6.7 - 9.8)	1.00		0.0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.1.4 Variation in All-cause Mortality by Age

Figure 35 Age specific all-cause mortality by maternal education both sexes.



Sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years

Table 60 Age specific all-cause mortality rates per 100 000 person years, SRR and SRD, by maternal education both sexes.

Cohort	Education Qualification	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1-4 yrs								
1981	Nil	153	74	(59 - 88)	2.2	(1.4 - 3.5)	40	(20 - 60)
	School	63	50	(34 - 66)	1.5	(0.9 - 2.5)	16	(-5 - 38)
	Post School	39	34	(20 - 48)	1.0		0	
1986	Nil	99	59	(45 - 74)	1.2	(0.8 - 1.9)	12	(-10 - 33)
	School	75	53	(39 - 68)	1.1	(0.7 - 1.7)	5	(-16 - 26)
	Post School	69	48	(33 - 63)	1.0		0	
1991	Nil	87	56	(41 - 71)	2.1	(1.4 - 3.2)	29	(12 - 47)
	School	66	37	(26 - 47)	1.4	(0.9 - 2.1)	10	(-4 - 24)
	Post School	54	27	(18 - 35)	1.0		0	
1996	Nil	66	45	(31 - 58)	1.4	(0.9 - 2.2)	13	(-4 - 30)
	School	81	41	(30 - 51)	1.3	(0.9 - 2.0)	9	(-5 - 24)
	Post School	54	31	(21 - 41)	1.0		0	
5-9 yrs								
1981	Nil	120	33	(26 - 40)	2.1	(1.0 - 4.2)	17	(4 - 30)
	School	51	29	(19 - 39)	1.8	(0.9 - 3.9)	13	(-2 - 28)
	Post School	21	16	(5 - 26)	1.0		0	
1986	Nil	63	23	(16 - 30)	0.9	(0.6 - 1.5)	-2	(-14 - 10)
	School	36	18	(11 - 26)	0.7	(0.4 - 1.2)	-7	(-20 - 5)
	Post School	51	26	(16 - 35)	1.0		0	
1991	Nil	63	25	(17 - 33)	1.2	(0.8 - 1.8)	4	(-7 - 14)
	School	39	17	(11 - 24)	0.8	(0.5 - 1.3)	-4	(-13 - 5)
	Post School	57	21	(15 - 28)	1.0		0	
1996	Nil	60	26	(18 - 34)	1.4	(0.9 - 2.2)	7	(-3 - 17)
	School	36	12	(7 - 16)	0.6	(0.4 - 1.0)	-7	(-15 - 1)
	Post School	51	19	(12 - 25)	1.0		0	
10-14 yrs								
1981	Nil	126	28	(22 - 34)	1.4	(0.8 - 2.3)	8	(-3 - 19)
	School	48	39	(23 - 54)	1.9	(1.0 - 3.6)	19	(1 - 37)
	Post School	30	20	(11 - 29)	1.0		0	
1986	Nil	96	29	(22 - 36)	0.8	(0.5 - 1.2)	-7	(-21 - 7)
	School	39	22	(13 - 31)	0.6	(0.4 - 1.0)	-14	(-29 - 0)
	Post School	60	37	(25 - 48)	1.0		0	
1991	Nil	57	24	(16 - 32)	1.1	(0.7 - 1.8)	2	(-8 - 13)
	School	36	20	(12 - 27)	0.9	(0.5 - 1.5)	-2	(-13 - 9)
	Post School	54	21	(14 - 29)	1.0		0	
1996	Nil	69	29	(20 - 38)	1.4	(0.9 - 2.1)	8	(-4 - 19)
	School	48	20	(13 - 27)	0.9	(0.6 - 1.5)	-1	(-11 - 9)
	Post School	51	21	(14 - 28)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.1.5 Variation in All-cause Mortality by Sex

Table 61 Sex specific all-cause mortality rates per 100 000 person years, SRR and SRD, by maternal education ages 1-14 years.

Cohort	Education Qualification	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Male								
1981	Nil	243	51	(42 - 59)	2.1	(1.4 - 3.0)	26	(14 - 38)
	School	102	49	(36 - 63)	2.0	(1.3 - 3.1)	25	(9 - 41)
	Post School	60	25	(16 - 33)	1.0		0	
1986	Nil	153	41	(33 - 49)	0.9	(0.6 - 1.2)	-6	(-20 - 8)
	School	84	35	(26 - 44)	0.8	(0.5 - 1.1)	-12	(-26 - 3)
	Post School	117	47	(35 - 58)	1.0		0	
1991	Nil	117	38	(29 - 48)	1.5	(1.1 - 2.2)	13	(2 - 24)
	School	78	27	(19 - 34)	1.1	(0.7 - 1.5)	1	(-8 - 11)
	Post School	96	25	(19 - 32)	1.0		0	
1996	Nil	120	38	(29 - 47)	1.5	(1.0 - 2.1)	12	(1 - 23)
	School	87	24	(18 - 30)	0.9	(0.6 - 1.3)	-2	(-11 - 7)
	Post School	90	26	(19 - 32)	1.0		0	
Female								
1981	Nil	162	36	(29 - 43)	1.7	(1.0 - 2.9)	15	(3 - 28)
	School	60	27	(19 - 36)	1.3	(0.7 - 2.4)	6	(-7 - 20)
	Post School	39	21	(11 - 31)	1.0		0	
1986	Nil	105	32	(24 - 39)	1.3	(0.8 - 1.9)	7	(-5 - 18)
	School	60	25	(17 - 33)	1.0	(0.6 - 1.5)	-0	(-11 - 11)
	Post School	60	25	(17 - 33)	1.0		0	
1991	Nil	90	29	(22 - 37)	1.4	(0.9 - 2.1)	9	(-1 - 18)
	School	60	21	(15 - 28)	1.0	(0.7 - 1.6)	1	(-8 - 10)
	Post School	69	21	(15 - 27)	1.0		0	
1996	Nil	75	27	(19 - 35)	1.3	(0.9 - 2.0)	6	(-4 - 16)
	School	78	23	(17 - 29)	1.1	(0.7 - 1.6)	2	(-7 - 11)
	Post School	63	21	(15 - 27)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.1.6 Variation in All-cause Mortality by Ethnic Group

Table 62 Ethnic specific all-cause mortality rates per 100 000 person years, SRR and SRD, by maternal education both sexes ages 1-14 years.

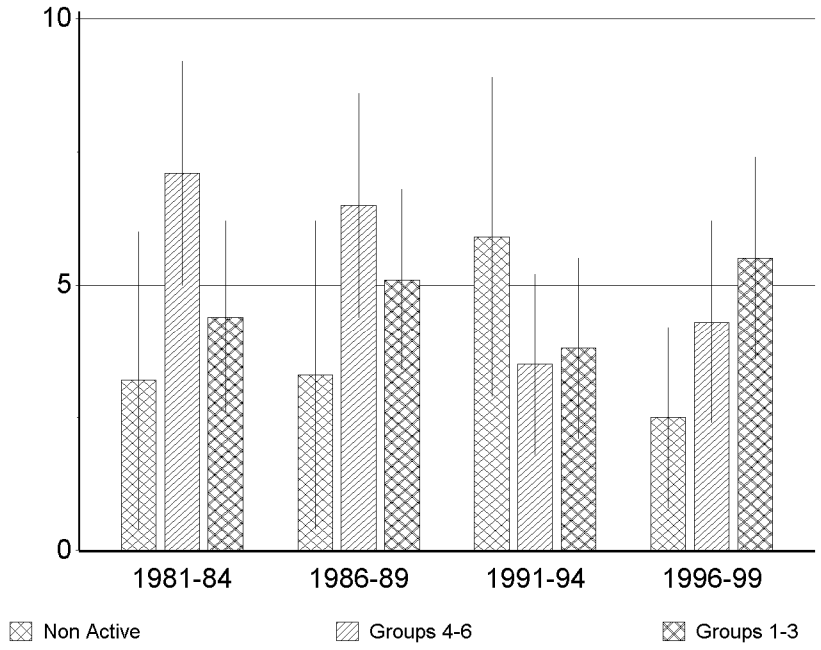
Cohort	Education Qualification	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Māori								
1981	Nil	126	52	(41 - 64)	3.0	(1.0 - 9.6)	35	(13 - 58)
	School	33	55	(30 - 79)	3.2	(0.9 - 10.6)	37	(6 - 68)
	Post School	6	17	(6 - 54)	1.0		0	
1986	Nil	93	44	(33 - 55)	0.8	(0.5 - 1.4)	-9	(-36 - 17)
	School	30	37	(21 - 54)	0.7	(0.4 - 1.3)	-16	(-45 - 13)
	Post School	27	53	(29 - 77)	1.0		0	
1991	Nil	90	45	(33 - 56)	3.3	(1.6 - 7.0)	31	(16 - 46)
	School	24	26	(14 - 38)	2.0	(0.8 - 4.5)	13	(-3 - 28)
	Post School	12	13	(4 - 23)	1.0		0	
1996	Nil	96	47	(35 - 60)	1.2	(0.8 - 1.9)	8	(-11 - 27)
	School	51	34	(23 - 46)	0.9	(0.5 - 1.4)	-5	(-24 - 13)
	Post School	42	40	(25 - 54)	1.0		0	
Non Māori Non Pacific								
1981	Nil	246	41	(34 - 47)	1.6	(1.2 - 2.2)	16	(6 - 25)
	School	114	30	(23 - 37)	1.2	(0.8 - 1.7)	5	(-4 - 14)
	Post School	87	25	(19 - 31)	1.0		0	
1986	Nil	153	35	(28 - 42)	1.1	(0.9 - 1.5)	4	(-5 - 13)
	School	105	26	(20 - 32)	0.9	(0.6 - 1.2)	-4	(-13 - 4)
	Post School	144	31	(25 - 37)	1.0		0	
1991	Nil	105	32	(24 - 39)	1.3	(1.0 - 1.8)	7	(-2 - 16)
	School	87	20	(15 - 25)	0.8	(0.6 - 1.1)	-5	(-12 - 2)
	Post School	144	25	(20 - 30)	1.0		0	
1996	Nil	81	29	(22 - 36)	1.5	(1.1 - 2.1)	10	(1 - 18)
	School	99	19	(15 - 24)	1.0	(0.7 - 1.4)	0	(-6 - 6)
	Post School	105	19	(15 - 23)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.2 Figures and Tables from Occupational Class and Labour Force Status Results

4.2.1 Cancer

Figure 36 Cancer mortality by occupational class both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years

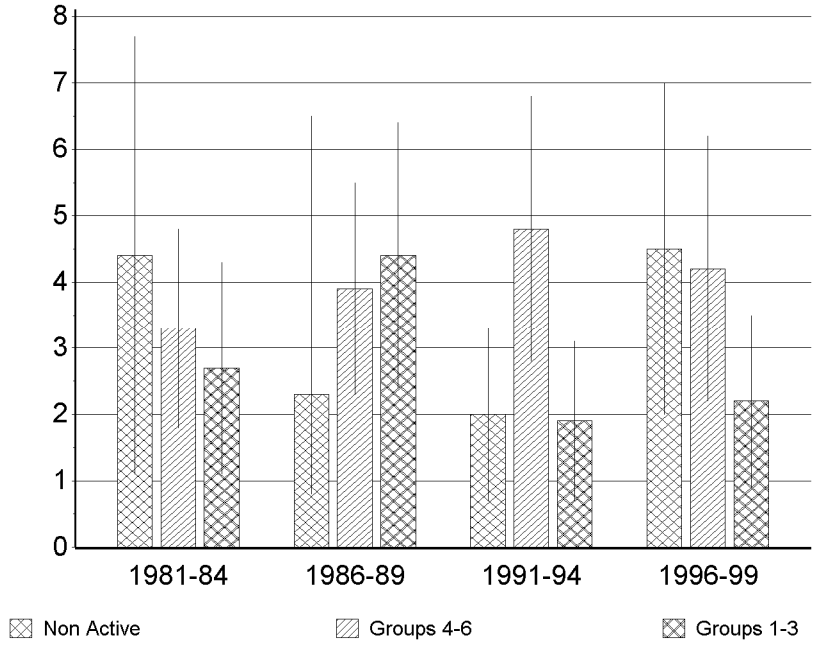
Table 63 All years cancer mortality rates per 100 000 person years SRR and SRD, by occupational class both sexes ages 1-14 years.

Elley Irving	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Non Active	57	4.0	(2.6 - 5.3)	0.81	(0.55 - 1.20)	-0.9	(-2.5 - 0.7)
Groups 4-6	171	5.6	(4.6 - 6.6)	1.15	(0.88 - 1.50)	0.7	(-0.6 - 2.1)
Groups 1-3	174	4.9	(3.9 - 5.8)	1.00		0.0	

Number of deaths rounded to base 3, with minimum cell size of 6 as per Statistics New Zealand protocol.

4.2.2 Congenital

Figure 37 Congenital mortality by occupational class- both sexes ages1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years

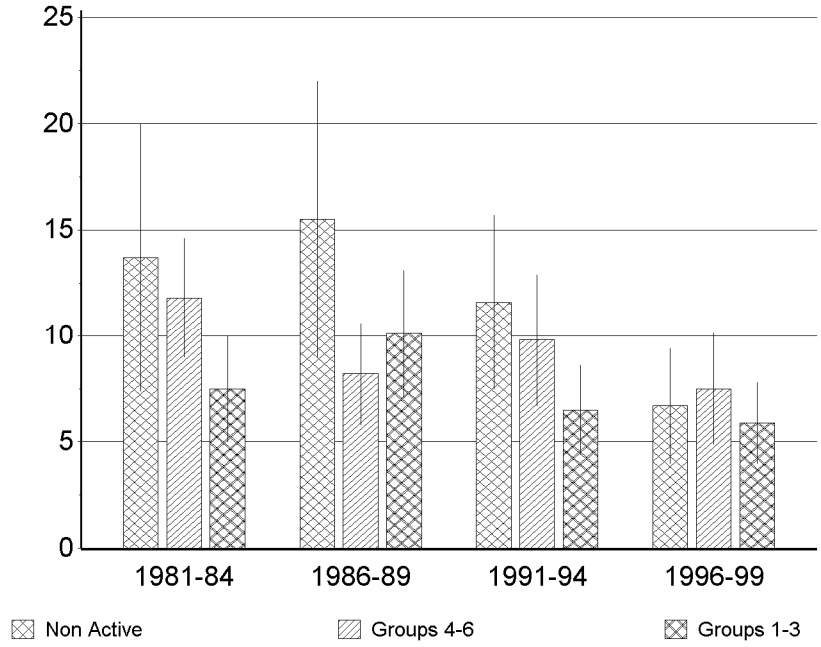
Table 64 All years congenital mortality rates per 100 000 person years SRR and SRD, by occupational class both sexes ages 1-14 years.

Elley Irving	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Non Active	51	3.2	(2.1 - 4.3)	1.14	(0.73 - 1.76)	0.4	(-1.0 - 1.7)
Groups 4-6	105	3.9	(3.1 - 4.8)	1.39	(0.98 - 1.98)	1.1	(0.0 - 2.3)
Groups 1-3	87	2.8	(2.0 - 3.6)	1.00		0.0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.2.3 Other Causes

Figure 38 Other mortality by occupational class both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years

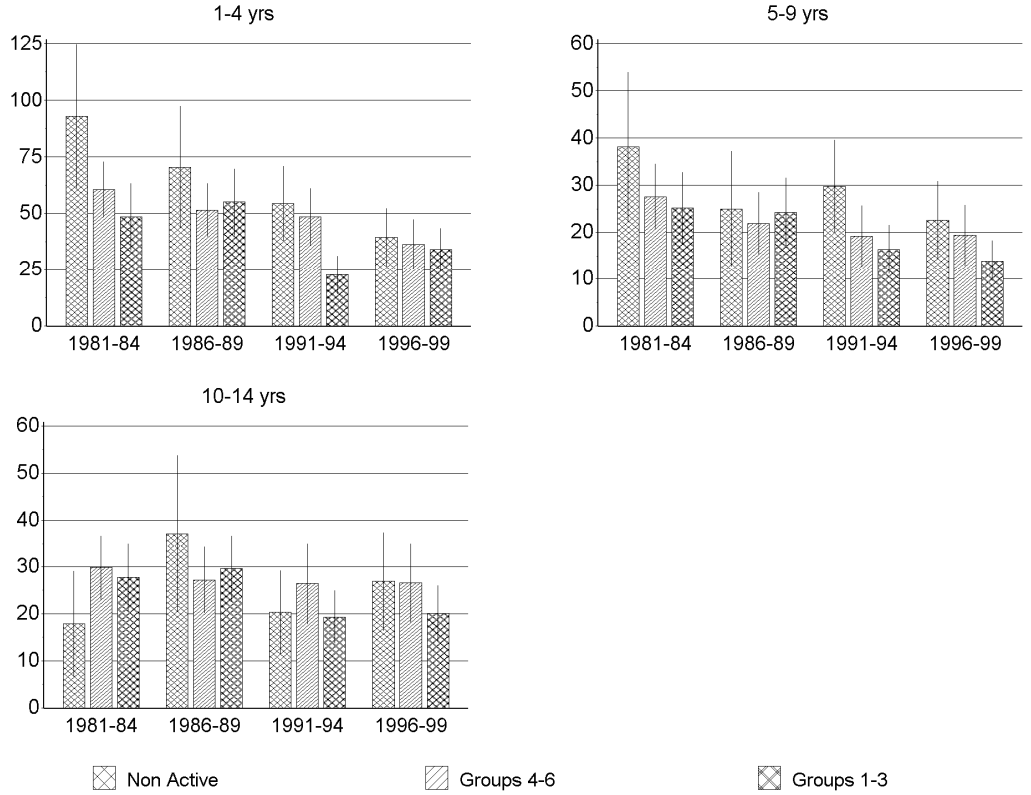
Table 65 All years other mortality rates per 100 000 person years SRR and SRD, by occupational class both sexes ages 1-14 years.

Elley Irving	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Non Active	162	11.2	(8.9 - 13.4)	1.52	(1.17 - 1.96)	3.8	(1.3 - 6.3)
Groups 4-6	279	9.4	(8.1 - 10.8)	1.28	(1.03 - 1.59)	2.1	(0.3 - 3.8)
Groups 1-3	267	7.3	(6.2 - 8.5)	1.00		0.0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.2.4 Variation in All-cause Mortality by Age

Figure 39 Age specific all-cause mortality by occupational class both sexes.



Sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years

Table 66 Age specific all-cause mortality rates per 100 000 person years, SRR and SRD, by occupational class both sexes.

Cohort	Elley Irving	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1-4 yrs								
1981	Non Active	54	93	(61 - 125)	1.9	(1.2 - 3.0)	45	(10 - 79)
	Groups 4-6	147	61	(49 - 73)	1.3	(0.9 - 1.8)	12	(-7 - 31)
	Groups 1-3	84	49	(34 - 63)	1.0		0	
1986	Non Active	45	71	(44 - 97)	1.3	(0.8 - 2.0)	15	(-15 - 46)
	Groups 4-6	108	52	(40 - 63)	0.9	(0.7 - 1.3)	-4	(-22 - 15)
	Groups 1-3	102	55	(41 - 70)	1.0		0	
1991	Non Active	75	54	(38 - 71)	2.4	(1.5 - 3.9)	32	(13 - 50)
	Groups 4-6	84	49	(36 - 61)	2.1	(1.4 - 3.4)	26	(11 - 41)
	Groups 1-3	51	23	(14 - 31)	1.0		0	
1996	Non Active	63	40	(27 - 52)	1.2	(0.8 - 1.8)	6	(-10 - 21)
	Groups 4-6	63	36	(26 - 47)	1.1	(0.7 - 1.6)	2	(-12 - 17)
	Groups 1-3	75	34	(25 - 43)	1.0		0	
5-9 yrs								
1981	Non Active	36	38	(22 - 54)	1.5	(0.9 - 2.5)	13	(-5 - 30)
	Groups 4-6	96	28	(21 - 35)	1.1	(0.7 - 1.6)	2	(-8 - 13)
	Groups 1-3	84	25	(18 - 33)	1.0		0	
1986	Non Active	27	25	(13 - 37)	1.0	(0.6 - 1.8)	1	(-13 - 15)
	Groups 4-6	63	22	(15 - 29)	0.9	(0.6 - 1.4)	-2	(-12 - 7)
	Groups 1-3	75	24	(17 - 32)	1.0		0	
1991	Non Active	63	30	(20 - 40)	1.8	(1.1 - 2.9)	13	(2 - 25)
	Groups 4-6	45	19	(13 - 26)	1.2	(0.7 - 1.9)	3	(-6 - 11)
	Groups 1-3	57	16	(11 - 22)	1.0		0	
1996	Non Active	48	23	(14 - 31)	1.6	(1.0 - 2.6)	9	(-1 - 18)
	Groups 4-6	54	19	(13 - 26)	1.4	(0.9 - 2.2)	6	(-2 - 13)
	Groups 1-3	54	14	(10 - 18)	1.0		0	
10-14 yrs								
1981	Non Active	15	18	(7 - 29)	0.7	(0.3 - 1.3)	-10	(-23 - 4)
	Groups 4-6	114	30	(23 - 37)	1.1	(0.8 - 1.5)	2	(-8 - 12)
	Groups 1-3	114	28	(20 - 35)	1.0		0	
1986	Non Active	27	37	(21 - 54)	1.3	(0.8 - 2.1)	8	(-11 - 26)
	Groups 4-6	84	27	(20 - 34)	0.9	(0.6 - 1.3)	-2	(-12 - 8)
	Groups 1-3	114	30	(23 - 37)	1.0		0	
1991	Non Active	39	20	(11 - 29)	1.1	(0.6 - 1.8)	1	(-9 - 12)
	Groups 4-6	54	26	(18 - 35)	1.4	(0.9 - 2.1)	7	(-3 - 17)
	Groups 1-3	69	19	(14 - 25)	1.0		0	
1996	Non Active	45	27	(16 - 37)	1.3	(0.8 - 2.2)	7	(-5 - 19)
	Groups 4-6	63	27	(18 - 35)	1.3	(0.9 - 2.0)	6	(-4 - 17)
	Groups 1-3	75	20	(14 - 26)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Table 67 Age specific RII and SII for all-cause mortality by occupational class both sexes.

Cohort	RII	95%CI	SII	95%CI
1-4 yrs				
1981	2.1	(1.1 - 4.3)	39	(11 - 68)
1986	1.1	(0.6 - 2.0)	3	(-23 - 29)
1991	5.2	(1.0 - 26.8)	46	(27 - 65)
1996	1.1	(0.5 - 2.4)	4	(-30 - 39)
P (Trend)	*0.64*		*0.95*	
5-9 yrs				
1981	1.2	(0.6 - 2.5)	5	(-3 - 14)
1986	0.8	(0.4 - 1.8)	-5	(-10 - 1)
1991	0.9	(0.4 - 2.1)	-2	(-35 - 30)
1996	1.8	(0.7 - 4.4)	9	(-2 - 20)
P (Trend)	*0.69*		*0.69*	
10-14 yrs				
1981	1.7	(0.9 - 3.2)	14	(-14 - 41)
1986	0.9	(0.5 - 1.8)	-2	(-20 - 15)
1991	2.5	(0.9 - 7.1)	19	(3 - 35)
1996	1.7	(0.7 - 4.0)	12	(6 - 17)
P (Trend)	*0.75*		*0.63*	

Age and Ethnicity Standardised, SII per 100 000 person years

Table 68 Wald test for age variations by occupational class.

Cohort	P value for Homogeneity of SRR	P value for Homogeneity of SRD	P value for Homogeneity of RII	P value for Homogeneity of SII
1981	0.52	0.28	0.27	<0.01
1986	0.87	0.90	0.61	0.29
1991	0.05	<0.01	0.01	<0.01
1996	0.36	0.64	0.39	0.13

P values significant when null hypothesis of homogeneity rejected

4.2.5 Variation in All-cause Mortality by Sex

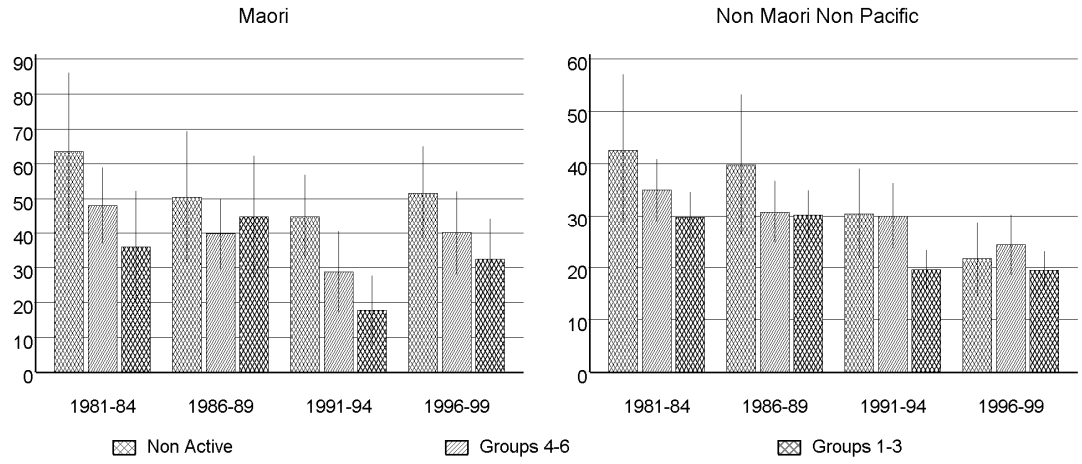
Table 69 Sex specific all-cause mortality rates per 100 000 person years, SRR and SRD, by occupational class ages 1-14 years.

Cohort	Elley Irving	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Male								
1981	Non Active	69	59	(41 - 78)	1.6	(1.1 - 2.3)	21	(1 - 42)
	Groups 4-6	219	45	(38 - 53)	1.2	(0.9 - 1.6)	7	(-5 - 19)
	Groups 1-3	162	38	(29 - 47)	1.0		0	
1986	Non Active	69	59	(41 - 77)	1.3	(0.9 - 1.8)	12	(-8 - 33)
	Groups 4-6	141	36	(28 - 43)	0.8	(0.6 - 1.0)	-11	(-23 - 1)
	Groups 1-3	186	47	(37 - 56)	1.0		0	
1991	Non Active	102	40	(29 - 50)	1.8	(1.3 - 2.6)	18	(6 - 30)
	Groups 4-6	102	34	(26 - 42)	1.5	(1.1 - 2.2)	12	(2 - 21)
	Groups 1-3	105	22	(16 - 27)	1.0		0	
1996	Non Active	99	36	(26 - 45)	1.3	(1.0 - 1.9)	9	(-2 - 20)
	Groups 4-6	87	26	(19 - 33)	1.0	(0.7 - 1.4)	-0	(-10 - 9)
	Groups 1-3	126	27	(21 - 33)	1.0		0	
Female								
1981	Non Active	36	36	(21 - 50)	1.3	(0.8 - 2.1)	8	(-8 - 24)
	Groups 4-6	135	31	(24 - 37)	1.1	(0.8 - 1.5)	3	(-7 - 13)
	Groups 1-3	120	28	(21 - 35)	1.0		0	
1986	Non Active	33	26	(14 - 38)	1.1	(0.7 - 1.9)	3	(-11 - 16)
	Groups 4-6	111	30	(23 - 36)	1.3	(0.9 - 1.8)	6	(-2 - 15)
	Groups 1-3	105	23	(18 - 29)	1.0		0	
1991	Non Active	75	28	(19 - 36)	1.7	(1.1 - 2.5)	11	(2 - 21)
	Groups 4-6	84	28	(21 - 35)	1.7	(1.1 - 2.5)	11	(2 - 20)
	Groups 1-3	72	17	(12 - 22)	1.0		0	
1996	Non Active	57	22	(15 - 30)	1.3	(0.8 - 2.0)	5	(-4 - 14)
	Groups 4-6	90	28	(21 - 35)	1.6	(1.1 - 2.3)	11	(2 - 19)
	Groups 1-3	75	18	(13 - 22)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.2.6 Variation in All-cause Mortality by Ethnic Group

Figure 40 Ethnic specific all-cause mortality by occupational class both sexes , 1-14 years .



Age and sex standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years

Table 70 Ethnic specific all-cause mortality rates per 100 000 person years, SRR and SRD, by occupational class both sexes ages 1-14 years.

Cohort	Elley Irving	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Māori								
1981	Non Active	42	64	(41 - 86)	1.8	(1.0 - 3.1)	28	(-0 - 55)
	Groups 4-6	117	48	(37 - 59)	1.3	(0.8 - 2.2)	12	(-8 - 32)
	Groups 1-3	27	36	(20 - 52)	1.0		0	
1986	Non Active	42	51	(32 - 69)	1.1	(0.7 - 1.9)	6	(-20 - 31)
	Groups 4-6	84	40	(30 - 50)	0.9	(0.6 - 1.4)	-5	(-25 - 15)
	Groups 1-3	42	45	(27 - 62)	1.0		0	
1991	Non Active	84	45	(33 - 57)	2.5	(1.4 - 4.7)	27	(12 - 43)
	Groups 4-6	36	29	(17 - 40)	1.6	(0.8 - 3.2)	11	(-4 - 26)
	Groups 1-3	18	18	(8 - 28)	1.0		0	
1996	Non Active	90	52	(38 - 65)	1.6	(1.0 - 2.5)	19	(1 - 37)
	Groups 4-6	72	40	(28 - 52)	1.2	(0.8 - 2.0)	8	(-9 - 25)
	Groups 1-3	45	32	(21 - 44)	1.0		0	
Non Māori Non Pacific								
1981	Non Active	54	43	(28 - 57)	1.4	(1.0 - 2.1)	13	(-2 - 28)
	Groups 4-6	210	35	(29 - 41)	1.2	(0.9 - 1.5)	5	(-2 - 13)
	Groups 1-3	243	30	(25 - 35)	1.0		0	
1986	Non Active	51	40	(26 - 53)	1.3	(0.9 - 1.9)	10	(-5 - 24)
	Groups 4-6	150	31	(25 - 37)	1.0	(0.8 - 1.3)	1	(-7 - 8)
	Groups 1-3	240	30	(26 - 35)	1.0		0	
1991	Non Active	72	31	(22 - 39)	1.6	(1.1 - 2.2)	11	(2 - 20)
	Groups 4-6	123	30	(24 - 36)	1.5	(1.2 - 2.0)	10	(3 - 18)
	Groups 1-3	153	20	(16 - 23)	1.0		0	
1996	Non Active	48	22	(15 - 29)	1.1	(0.8 - 1.6)	2	(-6 - 10)
	Groups 4-6	93	24	(19 - 30)	1.3	(0.9 - 1.7)	5	(-2 - 12)
	Groups 1-3	153	20	(16 - 23)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Table 71 Ethnic specific RII and SII for all-cause mortality by occupational class -both sexes ages 1-14 years.

Cohort	RII	95%CI	SII	95%CI
Māori				
1981	2.3	(1.0 - 5.3)	35	(2 - 68)
1986	0.7	(0.3 - 1.7)	-12	(-31 - 6)
1991	2.0	(0.5 - 8.2)	16	(-24 - 56)
1996	1.6	(0.7 - 3.7)	16	(9 - 23)
P (Trend)	*0.89*		*0.57*	
Non Māori Non Pacific				
1981	1.5	(0.9 - 2.3)	12	(1 - 23)
1986	1.2	(0.8 - 1.8)	4	(-4 - 13)
1991	2.1	(1.1 - 3.8)	16	(-1 - 34)
1996	1.3	(0.7 - 2.3)	5	(-10 - 20)
P (Trend)	*0.90*		*0.76*	

Age and sex standardised, SII per 100 000 person years
Y axis standard mortality rates /100 000 person years

Table 72 Wald test for ethnic variations by occupational class.

Cohort	P value for Homogeneity of SRR	P value for Homogeneity of SRD	P value for Homogeneity of RII	P value for Homogeneity of SII
1981	0.58	0.49	0.31	0.12
1986	0.40	0.37	0.27	0.08
1991	0.79	0.80	0.59	0.62
1996	0.86	0.75	0.71	0.03

P values significant when null hypothesis of homogeneity rejected

4.2.7 Farmer Information

4.2.7.1 Person Time

Table 73 Person time and number of deaths by farming status by age and cohort

		Cohort											
		1981			1986			1991			1996		
	Age	Deaths	Person Time	PT %	Deaths	Person Time	PT %	Deaths	Person Time	PT %	Deaths	Person Time	PT %
Both Sexes													
Farmer	1-4	39	59,339	3%	27	57,253	3%	18	44,642	2%	18	44,112	2%
	5-9	18	101,477	5%	15	91,319	5%	18	70,172	4%	12	75,723	4%
	10-14	39	113,863	5%	39	102,189	5%	27	71,105	4%	27	73,991	4%
		96	**274,678**	**13%**	**81**	**250,762**	**13%**	**63**	**185,919**	**9%**	**57**	**193,826**	**9%**
No Farmer	1-4	249	421,783	20%	231	418,331	21%	192	490,625	25%	186	485,731	23%
	5-9	198	701,135	32%	147	639,068	32%	147	675,019	34%	144	756,794	36%
	10-14	204	764,642	35%	186	682,541	34%	138	638,571	32%	150	663,625	32%
		651	**1,887,559**	**87%**	**564**	**1,739,939**	**87%**	**477**	**1,804,215**	**91%**	**480**	**1,906,150**	**91%**
		744	***2,162,238***	***100%***	***648***	***1,990,701***	***100%***	***537***	***1,990,134***	***100%***	***537***	***2,099,976***	***100%***

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.2.7.2 All-cause Mortality

Table 74 All-cause standard mortality rates per 100 000 person years SRR and SRD, by farming status, both sexes ages 1-14 years.

Cohort	Farmer	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1981	Farmer	96	36	(26 - 45)	1.0	(0.7 - 1.3)	-2	(-12 - 8)
	No Farmer	648	37	(34 - 41)	1.0		0	
1986	Farmer	81	33	(24 - 42)	1.0	(0.7 - 1.3)	-2	(-11 - 8)
	No Farmer	567	35	(31 - 38)	1.0		0	
1991	Farmer	60	35	(23 - 47)	1.3	(0.9 - 1.9)	8	(-5 - 20)
	No Farmer	477	27	(24 - 30)	1.0		0	
1996	Farmer	54	33	(22 - 44)	1.3	(0.9 - 1.9)	8	(-4 - 19)
	No Farmer	480	25	(23 - 28)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Injury

Table 75 Injury standard mortality rates per 100 000 person years SRR and SRD, by farming status, both sexes ages 1-14 years.

Cohort	Farmer	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1981	Farmer	57	21	(14 - 28)	1.2	(0.9 - 1.8)	4	(-4 - 12)
	No Farmer	303	17	(15 - 19)	1.0		0	
1986	Farmer	42	17	(10 - 24)	1.1	(0.7 - 1.7)	2	(-6 - 9)
	No Farmer	252	16	(13 - 18)	1.0		0	
1991	Farmer	33	21	(10 - 31)	1.9	(1.1 - 3.3)	10	(-0 - 20)
	No Farmer	192	11	(9 - 13)	1.0		0	
1996	Farmer	30	18	(9 - 27)	1.7	(1.0 - 2.8)	7	(-2 - 16)
	No Farmer	210	11	(9 - 12)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Cancer

Table 76 All years cancer mortality rates per 100 000 person years SRR and SRD, by farming status both sexes ages 1-14 years.

Farmer	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
No Farmer	360	5.0	(4.3 - 5.6)	1.00		0.0	
Farmer	45	5.0	(3.1 - 6.8)	1.00	(0.68 - 1.48)	0.0	(-1.9 - 1.9)

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Congenital Mortality

Table 77 All years congenital mortality rates per 100 000 person years SRR and SRD, by farming status both sexes ages 1-14 years.

Farmer	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
No Farmer	228	3.4	(2.9 - 3.9)	1.00		0.0	
Farmer	18	1.7	(0.7 - 2.7)	0.51	(0.28 - 0.92)	-1.7	(-2.8 - -0.6)

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Other Mortality

Table 78 All years other mortality rates per 100 000 person years SRR and SRD, by farming status both sexes ages 1-14 years.

Farmer	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
No Farmer	639	9.0	(8.2 - 9.9)	1.00		0.0	
Farmer	72	8.3	(5.8 - 10.8)	0.92	(0.67 - 1.26)	-0.7	(-3.4 - 1.9)

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.2.7.4 Variation in All-cause Mortality by Age

Table 79 Age specific all-cause mortality rates per 100 000 person years, SRR and SRD, by farming status both sexes.

Cohort	Farmer	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1-4								
1981	Farmer	39	62	(38 - 87)	1.1	(0.7 - 1.6)	3	(-23 - 29)
	No Farmer	249	59	(50 - 68)	1.0		0	
1986	Farmer	27	46	(25 - 67)	0.8	(0.5 - 1.3)	-10	(-32 - 13)
	No Farmer	231	56	(47 - 65)	1.0		0	
1991	Farmer	18	45	(15 - 76)	1.2	(0.6 - 2.3)	7	(-25 - 38)
	No Farmer	192	39	(32 - 46)	1.0		0	
1996	Farmer	18	39	(16 - 61)	1.0	(0.6 - 1.9)	2	(-22 - 25)
	No Farmer	186	37	(31 - 44)	1.0		0	
5-9								
1981	Farmer	18	16	(6 - 25)	0.6	(0.3 - 1.0)	-13	(-23 - -2)
	No Farmer	198	28	(24 - 33)	1.0		0	
1986	Farmer	15	15	(6 - 24)	0.7	(0.4 - 1.2)	-8	(-18 - 2)
	No Farmer	147	23	(19 - 28)	1.0		0	
1991	Farmer	18	24	(9 - 38)	1.1	(0.6 - 2.1)	2	(-13 - 17)
	No Farmer	147	22	(17 - 26)	1.0		0	
1996	Farmer	12	20	(5 - 36)	1.1	(0.5 - 2.4)	2	(-14 - 18)
	No Farmer	144	18	(15 - 22)	1.0		0	
10-14								
1981	Farmer	39	32	(19 - 45)	1.2	(0.8 - 1.8)	5	(-9 - 18)
	No Farmer	204	27	(23 - 32)	1.0		0	
1986	Farmer	39	39	(23 - 55)	1.4	(0.9 - 2.2)	11	(-5 - 28)
	No Farmer	186	28	(23 - 32)	1.0		0	
1991	Farmer	27	36	(18 - 54)	1.7	(1.0 - 2.8)	14	(-4 - 33)
	No Farmer	138	22	(17 - 26)	1.0		0	
1996	Farmer	27	40	(20 - 61)	1.8	(1.1 - 3.2)	18	(-3 - 39)
	No Farmer	150	22	(18 - 26)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.2.7.5 Variation in All-cause Mortality by Sex

Table 80 Sex specific all-cause mortality rates per 100 000 person years, SRR and SRD, by farming status ages 1-14 years.

Cohort	Farmer	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Male								
1981	No Farmer	393	44	(39 - 50)	1.0		0	
	Farmer	57	41	(27 - 54)	0.9	(0.6 - 1.3)	-3	(-18 - 11)
1986	No Farmer	348	41	(36 - 47)	1.0		0	
	Farmer	51	39	(25 - 53)	0.9	(0.6 - 1.4)	-3	(-17 - 12)
1991	No Farmer	267	30	(26 - 34)	1.0		0	
	Farmer	39	47	(26 - 68)	1.6	(1.0 - 2.5)	17	(-4 - 39)
1996	No Farmer	282	29	(25 - 33)	1.0		0	
	Farmer	33	35	(19 - 50)	1.2	(0.8 - 1.9)	6	(-10 - 22)
Female								
1981	No Farmer	258	30	(26 - 35)	1.0		0	
	Farmer	36	30	(18 - 43)	1.0	(0.6 - 1.6)	0	(-13 - 13)
1986	No Farmer	219	28	(23 - 32)	1.0		0	
	Farmer	30	27	(15 - 38)	1.0	(0.6 - 1.5)	-1	(-13 - 12)
1991	No Farmer	207	24	(20 - 27)	1.0		0	
	Farmer	21	21	(10 - 33)	0.9	(0.5 - 1.6)	-2	(-15 - 10)
1996	No Farmer	198	22	(18 - 25)	1.0		0	
	Farmer	24	30	(14 - 47)	1.4	(0.8 - 2.5)	9	(-8 - 26)

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.2.7.6 Variation in All-cause Mortality by Ethnic Group

Table 81 Ethnic specific all-cause mortality rates per 100 000 person years, SRR and SRD, by farming status both sexes ages 1-14 years.

Cohort	Farmer	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Māori								
1981	No Farmer	177	50	(41 - 59)	1.0		0	
	Farmer	15	36	(13 - 59)	0.7	(0.4 - 1.4)	-14	(-39 - 10)
1986	No Farmer	153	44	(36 - 53)	1.0		0	
	Farmer	12	34	(12 - 56)	0.8	(0.4 - 1.5)	-10	(-34 - 14)
1991	No Farmer	126	33	(26 - 40)	1.0		0	
	Farmer	9	43	(11 - 75)	1.3	(0.6 - 2.8)	10	(-23 - 43)
1996	No Farmer	195	42	(35 - 50)	1.0		0	
	Farmer	12	38	(11 - 65)	0.9	(0.4 - 1.9)	-4	(-32 - 24)
Non Māori Non Pacific								
1981	No Farmer	426	32	(28 - 36)	1.0		0	
	Farmer	78	39	(28 - 50)	1.2	(0.9 - 1.6)	7	(-5 - 18)
1986	No Farmer	372	31	(27 - 35)	1.0		0	
	Farmer	66	34	(24 - 44)	1.1	(0.8 - 1.5)	3	(-8 - 13)
1991	No Farmer	300	24	(21 - 27)	1.0		0	
	Farmer	48	31	(20 - 41)	1.3	(0.9 - 1.8)	7	(-4 - 18)
1996	No Farmer	252	21	(18 - 23)	1.0		0	
	Farmer	42	26	(16 - 36)	1.3	(0.9 - 1.9)	6	(-5 - 16)

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Appendix 3 - Alternative Education Variable

This appendix includes results for child mortality by highest educational qualification in the household. These will mainly, but not exclusively, be parents as discussed in the methods section.

4.3 Cohort Restriction

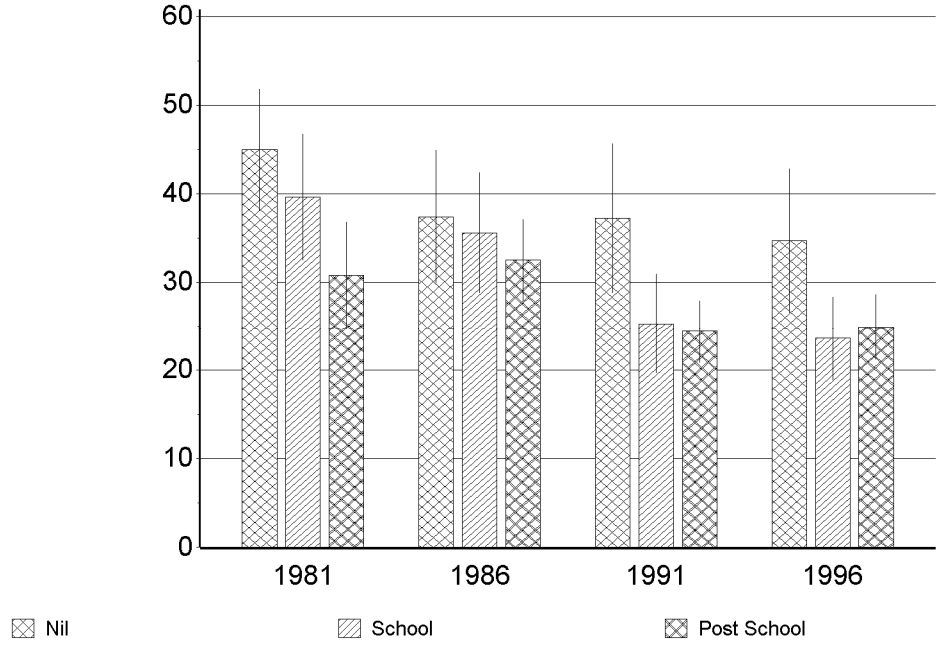
Table 82 Person time and number of deaths in each education category by age and cohort.

		Cohort											
		1981			1986			1991			1996		
Education Qualification	Age	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %	Deaths	Person Time (yrs)	PT %
Both Sexes													
Nil	1-4	120	147,737	7%	66	102,259	5%	63	100,494	5%	51	90,477	4%
	5-9	90	277,046	13%	39	173,912	9%	42	144,944	7%	36	151,110	7%
	10-14	96	326,751	15%	66	200,822	10%	42	146,115	7%	48	129,839	6%
		306	**751,534**	**35%**	**171**	**476,993**	**24%**	**147**	**391,553**	**20%**	**135**	**371,426**	**18%**
School	1-4	72	126,909	6%	75	115,866	6%	48	124,499	6%	54	157,581	8%
	5-9	54	192,925	9%	33	167,257	8%	30	165,758	8%	45	240,843	11%
	10-14	63	204,530	9%	48	171,956	9%	36	150,451	8%	45	207,437	10%
		189	**524,364**	**24%**	**156**	**455,078**	**23%**	**114**	**440,707**	**22%**	**144**	**605,862**	**29%**
Post School	1-4	87	191,632	9%	114	250,911	13%	99	308,403	15%	96	280,230	13%
	5-9	69	302,714	14%	81	376,438	19%	93	429,366	22%	75	437,751	21%
	10-14	75	310,953	14%	111	396,935	20%	87	408,745	21%	84	397,841	19%
		231	**805,299**	**37%**	**306**	**1,024,283**	**51%**	**279**	**1,146,514**	**58%**	**255**	**1,115,822**	**53%**
Missing	1-4	9	14,844	1%	6	6,548	0%	6	1,872	0%	6	1,555	0%
	5-9	6	29,926	1%	12	12,780	1%	6	5,123	0%	6	2,813	0%
	10-14	12	36,271	2%	6	15,018	1%	6	4,365	0%	6	2,499	0%
		27	**81,041**	**4%**	**24**	**34,346**	**2%**	**18**	**11,360**	**1%**	**18**	**6,866**	**0%**
		744	***2,162,238***	***100%***	***648***	***1,990,701***	***100%***	***537***	***1,990,134***	***100%***	***537***	***2,099,976***	***100%***

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.4 All-cause Mortality

Figure 41 All-cause mortality by highest education both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years.

Table 83 All-cause mortality rates per 100 000 person years, SRR and SRD, by highest education both sexes ages 1-14 years.

Cohort	Education Qualification	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1981	Nil	303	45	(38 - 52)	1.5	(1.1 - 1.9)	14	(5 - 23)
	School	189	40	(33 - 47)	1.3	(1.0 - 1.7)	9	(-1 - 18)
	Post School	231	31	(25 - 37)	1.0		0	
1986	Nil	171	37	(30 - 45)	1.2	(0.9 - 1.5)	5	(-4 - 14)
	School	153	36	(29 - 42)	1.1	(0.9 - 1.4)	3	(-5 - 11)
	Post School	309	33	(28 - 37)	1.0		0	
1991	Nil	144	37	(29 - 46)	1.5	(1.2 - 2.0)	13	(4 - 22)
	School	114	25	(20 - 31)	1.0	(0.8 - 1.3)	1	(-6 - 7)
	Post School	279	24	(21 - 28)	1.0		0	
1996	Nil	135	35	(27 - 43)	1.4	(1.1 - 1.8)	10	(1 - 19)
	School	144	24	(19 - 28)	1.0	(0.7 - 1.2)	-1	(-7 - 5)
	Post School	255	25	(21 - 29)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Table 84 All-cause mortality RII and SII by highest education both sexes ages 1-14 years.

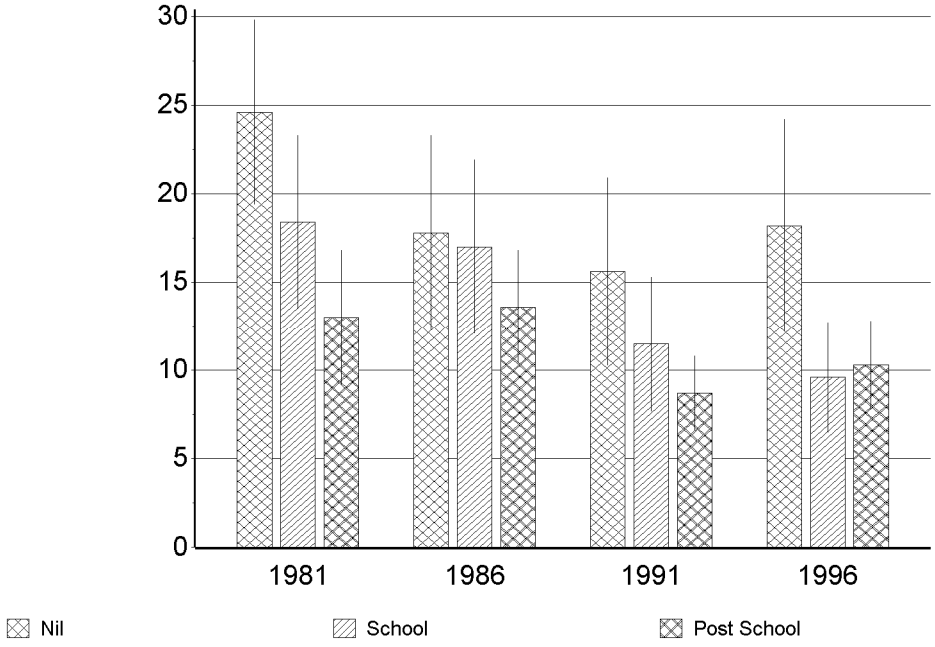
Cohort	RII	95%CI	SII	95%CI
1981	2.1	(1.4 - 3.0)	26	(7 - 45)
1986	1.2	(0.8 - 1.8)	5	(-1 - 12)
1991	2.0	(1.3 - 3.1)	18	(5 - 31)
1996	1.7	(1.1 - 2.6)	13	(-1 - 28)
P (Trend)	*0.92*		*0.83*	

Age, sex and ethnicity standardised, SII per 100 000 person years

4.5 Variation in Mortality by Cause of Death

4.5.1 Injury

Figure 42 Injury mortality by highest education, both sexes and ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years.

Table 85 Injury mortality rates per 100 000 person years SRR and SRD, by highest education both sexes ages 1-14 years.

Cohort	Education Qualification	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1981	Nil	159	25	(19 - 30)	1.9	(1.3 - 2.7)	12	(5 - 18)
	School	87	18	(14 - 23)	1.4	(1.0 - 2.1)	5	(-1 - 12)
	Post School	99	13	(9 - 17)	1.0		0	
1986	Nil	81	18	(12 - 23)	1.3	(0.9 - 1.9)	4	(-2 - 11)
	School	75	17	(12 - 22)	1.2	(0.9 - 1.8)	3	(-3 - 9)
	Post School	132	14	(10 - 17)	1.0		0	
1991	Nil	66	16	(10 - 21)	1.8	(1.2 - 2.7)	7	(1 - 13)
	School	51	12	(8 - 15)	1.3	(0.9 - 2.0)	3	(-2 - 7)
	Post School	102	9	(7 - 11)	1.0		0	
1996	Nil	72	18	(12 - 24)	1.8	(1.2 - 2.7)	8	(1 - 14)
	School	60	10	(7 - 13)	0.9	(0.6 - 1.4)	-1	(-5 - 3)
	Post School	108	10	(8 - 13)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

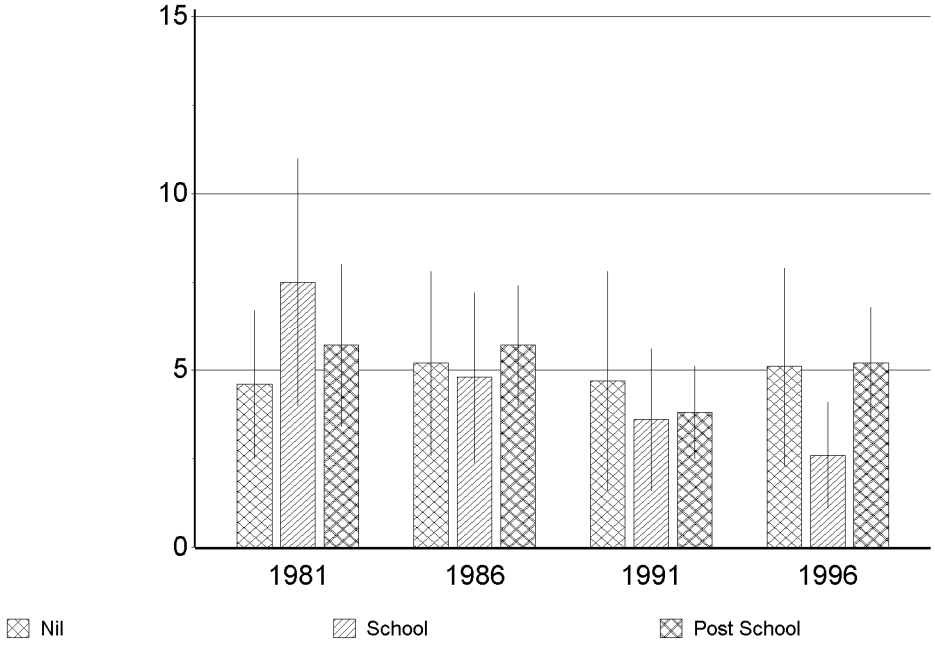
Table 86 Injury mortality RII and SII by highest education both sexes ages 1-14 years.

Cohort	RII	95%CI	SII	95%CI
1981	3.2	(1.6 - 6.4)	19	(12 - 28)
1986	1.9	(1.0 - 3.4)	9	(2 - 17)
1991	2.9	(1.3 - 6.7)	10	(5 - 16)
1996	2.1	(1.0 - 4.5)	8	(-3 - 19)
P (Trend)	*0.62*		*0.21*	

Age, sex and ethnicity standardised, SII per 100 000 person years

4.5.2 Cancer

Figure 43 Cancer mortality by highest education both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years.

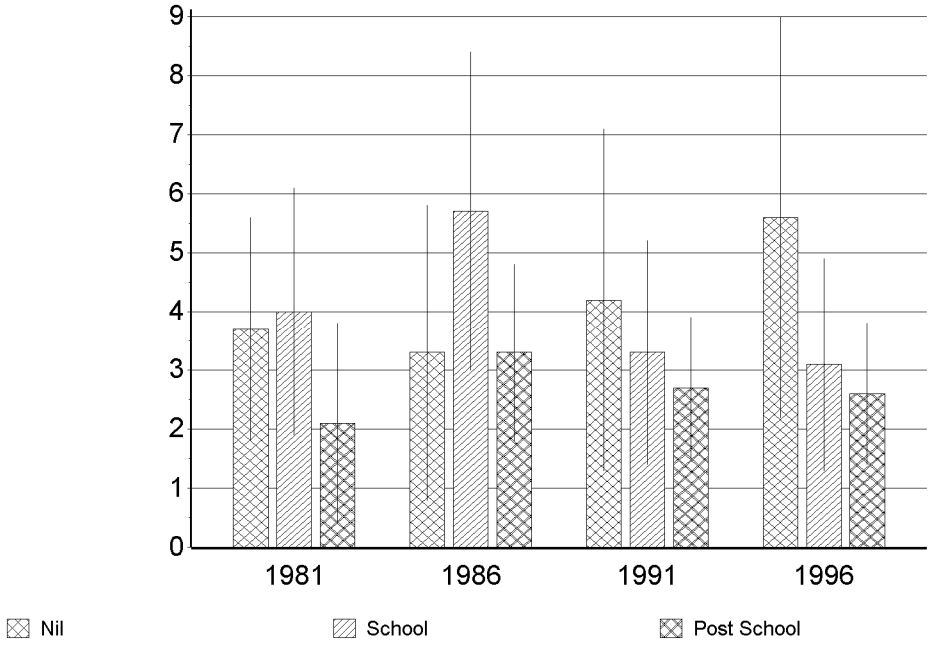
Table 87 All years cancer mortality rates per 100 000 person years SRR and SRD, by highest education both sexes ages 1-14 years.

Education Qualification	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Nil	102	4.9	(3.6 - 6.1)	0.95	(0.70 - 1.30)	-0.2	(-1.8 - 1.3)
School	90	4.4	(3.3 - 5.6)	0.87	(0.64 - 1.17)	-0.7	(-2.1 - 0.7)
Post School	207	5.1	(4.3 - 6.0)	1.00		0.0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.5.3 Congenital

Figure 44 Congenital mortality by highest education both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years.

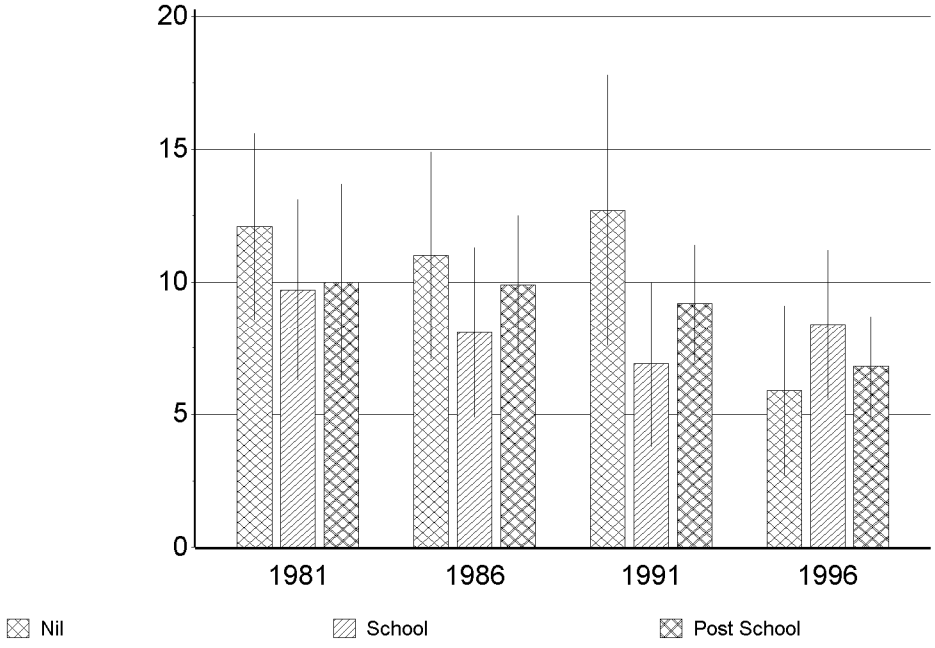
Table 88 All years congenital mortality rates per 100 000 person years SRR and SRD, by highest education both sexes ages 1-14 years.

Education Qualification	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Nil	63	3.9	(2.7 - 5.2)	1.48	(1.00 - 2.20)	1.3	(-0.1 - 2.7)
School	75	3.9	(2.9 - 5.0)	1.48	(1.03 - 2.11)	1.3	(0.0 - 2.5)
Post School	99	2.7	(2.0 - 3.3)	1.00		0.0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.5.4 Other

Figure 45 Other mortality by highest education both sexes ages 1-14 years.



Age, sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years.

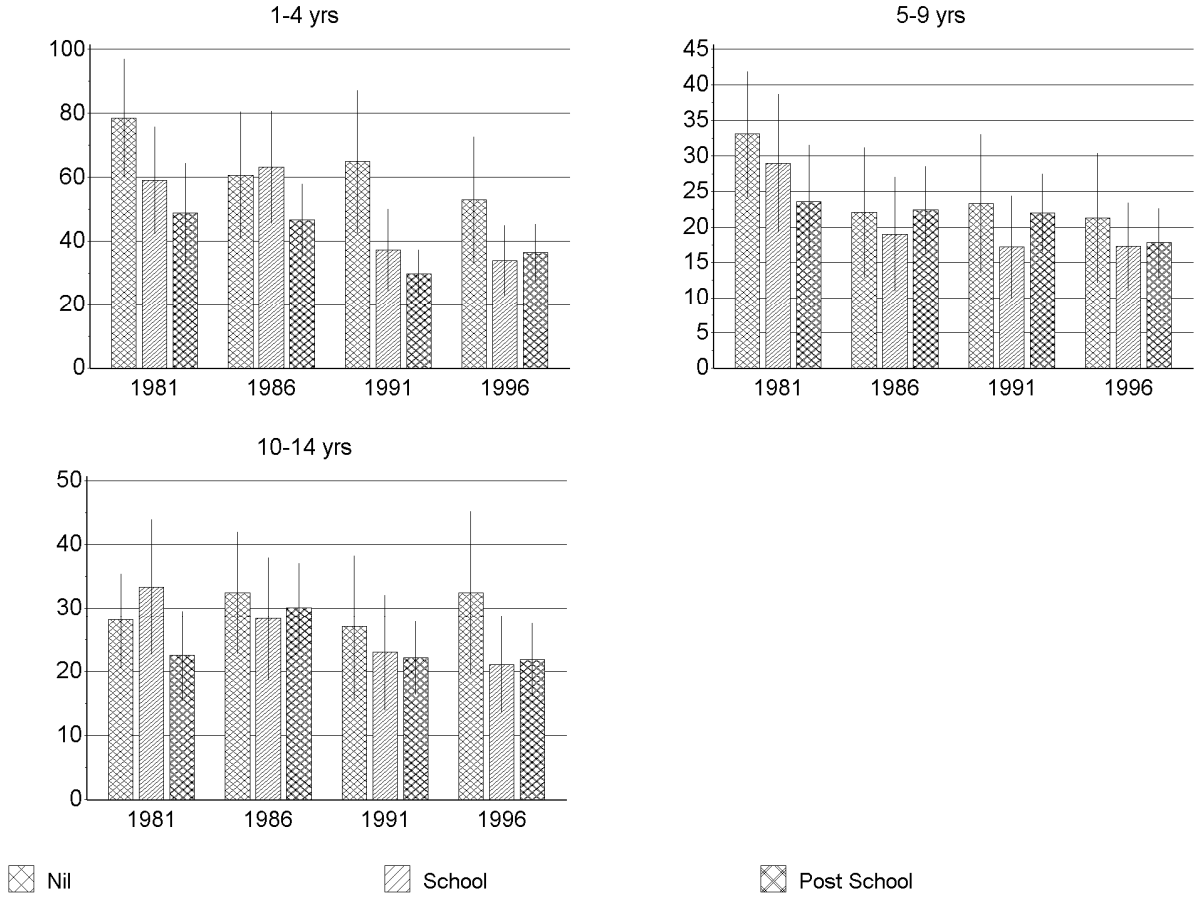
Table 89 All years other mortality rates per 100 000 person years SRR and SRD, by highest education both sexes ages 1-14 years.

Education Qualification	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Nil	204	10.7	(8.7 - 12.6)	1.21	(0.96 - 1.52)	1.8	(-0.5 - 4.1)
School	165	8.4	(6.8 - 9.9)	0.95	(0.75 - 1.19)	-0.5	(-2.4 - 1.5)
Post School	333	8.8	(7.6 - 10.0)	1.00		0.0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

4.6 Variation in All-cause Mortality by Age

Figure 46 Age specific all-cause mortality by highest education both sexes.



Sex and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years.

Table 90 Age specific all-cause mortality rates per 100 000 person years, SRR and SRD, by highest education both sexes.

Cohort	Education Qualification	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
1-4 yrs								
1981	Nil	120	79	(60 - 97)	1.6	(1.1 - 2.4)	30	(6 - 54)
	School	72	59	(42 - 76)	1.2	(0.8 - 1.9)	10	(-13 - 33)
	Post School	87	49	(33 - 64)	1.0		0	
1986	Nil	66	61	(41 - 80)	1.3	(0.9 - 1.9)	14	(-9 - 37)
	School	75	63	(46 - 81)	1.4	(0.9 - 1.9)	17	(-4 - 37)
	Post School	114	47	(36 - 58)	1.0		0	
1991	Nil	63	65	(43 - 87)	2.2	(1.4 - 3.3)	35	(12 - 58)
	School	48	37	(25 - 50)	1.3	(0.8 - 1.9)	7	(-7 - 22)
	Post School	99	30	(23 - 37)	1.0		0	
1996	Nil	51	53	(33 - 73)	1.5	(0.9 - 2.3)	16	(-5 - 38)
	School	54	34	(23 - 45)	0.9	(0.6 - 1.4)	-3	(-17 - 12)
	Post School	96	37	(28 - 45)	1.0		0	
5-9 yrs								
1981	Nil	90	33	(24 - 42)	1.4	(0.9 - 2.2)	10	(-2 - 21)
	School	54	29	(19 - 39)	1.2	(0.8 - 2.0)	5	(-7 - 18)
	Post School	69	24	(16 - 32)	1.0		0	
1986	Nil	39	22	(13 - 31)	1.0	(0.6 - 1.6)	-0	(-11 - 11)
	School	33	19	(11 - 27)	0.9	(0.5 - 1.4)	-3	(-13 - 7)
	Post School	81	22	(16 - 29)	1.0		0	
1991	Nil	42	23	(14 - 33)	1.1	(0.7 - 1.7)	1	(-10 - 12)
	School	30	17	(10 - 24)	0.8	(0.5 - 1.3)	-5	(-14 - 4)
	Post School	93	22	(17 - 28)	1.0		0	
1996	Nil	36	21	(12 - 30)	1.2	(0.7 - 2.0)	3	(-7 - 14)
	School	45	17	(11 - 23)	1.0	(0.6 - 1.5)	-1	(-8 - 7)
	Post School	75	18	(13 - 23)	1.0		0	
10-14 yrs								
1981	Nil	96	28	(21 - 35)	1.3	(0.8 - 1.9)	6	(-5 - 16)
	School	63	33	(23 - 44)	1.5	(0.9 - 2.3)	11	(-2 - 24)
	Post School	75	23	(16 - 30)	1.0		0	
1986	Nil	66	32	(23 - 42)	1.1	(0.7 - 1.6)	2	(-10 - 14)
	School	48	28	(19 - 38)	0.9	(0.6 - 1.4)	-2	(-14 - 10)
	Post School	111	30	(23 - 37)	1.0		0	
1991	Nil	42	27	(16 - 38)	1.2	(0.8 - 2.0)	5	(-8 - 18)
	School	36	23	(14 - 32)	1.0	(0.7 - 1.7)	1	(-10 - 12)
	Post School	87	22	(16 - 28)	1.0		0	
1996	Nil	48	32	(20 - 45)	1.5	(0.9 - 2.4)	11	(-3 - 25)
	School	45	21	(14 - 29)	1.0	(0.6 - 1.5)	-1	(-10 - 9)
	Post School	84	22	(16 - 28)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

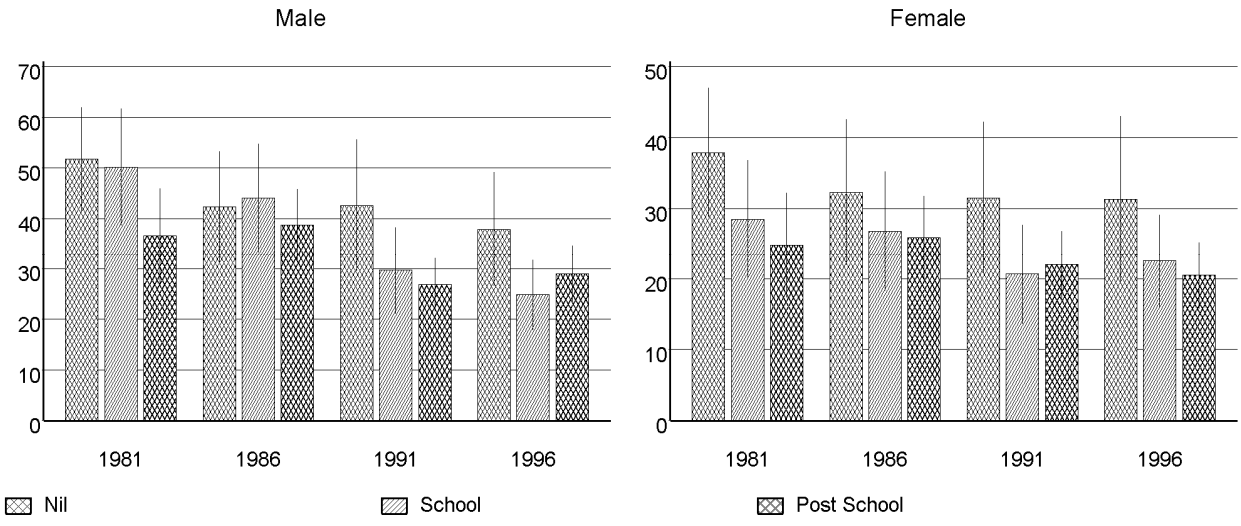
Table 91 Age specific RII and SII for all-cause mortality by highest education-both sexes.

Cohort	RII	95%CI	SII	95%CI
1-4 yrs				
1981	2.6	(1.4 - 5.2)	54	(22 - 87)
1986	1.9	(1.0 - 3.5)	33	(9 - 57)
1991	3.3	(1.3 - 8.5)	41	(13 - 69)
1996	1.8	(0.9 - 3.6)	21	(0 - 42)
P (Trend)	*0.58*		*0.16*	
5-9 yrs				
1981	2.1	(1.1 - 4.1)	20	(3 - 36)
1986	0.7	(0.2 - 1.8)	-9	(-29 - 11)
1991	1.3	(0.7 - 2.5)	6	(-15 - 26)
1996	1.2	(0.6 - 2.7)	4	(-13 - 20)
P (Trend)	*0.56*		*0.55*	
10-14 yrs				
1981	1.4	(0.7 - 2.6)	9	(-21 - 38)
1986	0.9	(0.4 - 2.0)	-2	(-15 - 11)
1991	1.6	(0.8 - 3.2)	10	(3 - 17)
1996	2.0	(1.0 - 4.4)	15	(-0 - 31)
P (Trend)	*0.37*		*0.21*	

Sex and ethnicity standardised, SII per 100 000 person years

4.7 Variation in All-cause Mortality by Sex

Figure 47 Sex specific all-cause mortality by highest education ages 1-14 years.



Age and ethnicity standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years.

Table 92 Sex specific all-cause mortality rates per 100 000 person years, SRR and SRD, by highest education ages 1-14 years.

Cohort	Education Qualification	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Male								
1981	Nil	180	52	(42 - 62)	1.4	(1.0 - 2.0)	15	(1 - 29)
	School	120	50	(39 - 62)	1.4	(1.0 - 1.9)	14	(-1 - 28)
	Post School	138	37	(27 - 46)	1.0		0	
1986	Nil	105	42	(31 - 53)	1.1	(0.8 - 1.5)	4	(-10 - 17)
	School	96	44	(33 - 55)	1.1	(0.8 - 1.5)	5	(-7 - 18)
	Post School	189	39	(32 - 46)	1.0		0	
1991	Nil	84	43	(30 - 56)	1.6	(1.1 - 2.3)	16	(2 - 30)
	School	69	30	(21 - 38)	1.1	(0.8 - 1.6)	3	(-7 - 13)
	Post School	159	27	(22 - 32)	1.0		0	
1996	Nil	84	38	(27 - 49)	1.3	(0.9 - 1.9)	9	(-4 - 22)
	School	75	25	(18 - 32)	0.9	(0.6 - 1.2)	-4	(-13 - 5)
	Post School	153	29	(23 - 35)	1.0		0	
Female								
1981	Nil	123	38	(29 - 47)	1.5	(1.0 - 2.2)	13	(1 - 25)
	School	69	29	(20 - 37)	1.2	(0.8 - 1.7)	4	(-8 - 15)
	Post School	93	25	(17 - 32)	1.0		0	
1986	Nil	66	32	(22 - 43)	1.3	(0.8 - 1.8)	6	(-6 - 18)
	School	57	27	(18 - 35)	1.0	(0.7 - 1.5)	1	(-9 - 11)
	Post School	117	26	(20 - 32)	1.0		0	
1991	Nil	63	32	(21 - 42)	1.4	(1.0 - 2.1)	10	(-2 - 21)
	School	48	21	(14 - 28)	0.9	(0.6 - 1.4)	-1	(-10 - 7)
	Post School	117	22	(17 - 27)	1.0		0	
1996	Nil	51	31	(20 - 43)	1.5	(1.0 - 2.4)	11	(-2 - 23)
	School	66	23	(16 - 29)	1.1	(0.8 - 1.6)	2	(-6 - 10)
	Post School	102	21	(16 - 25)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

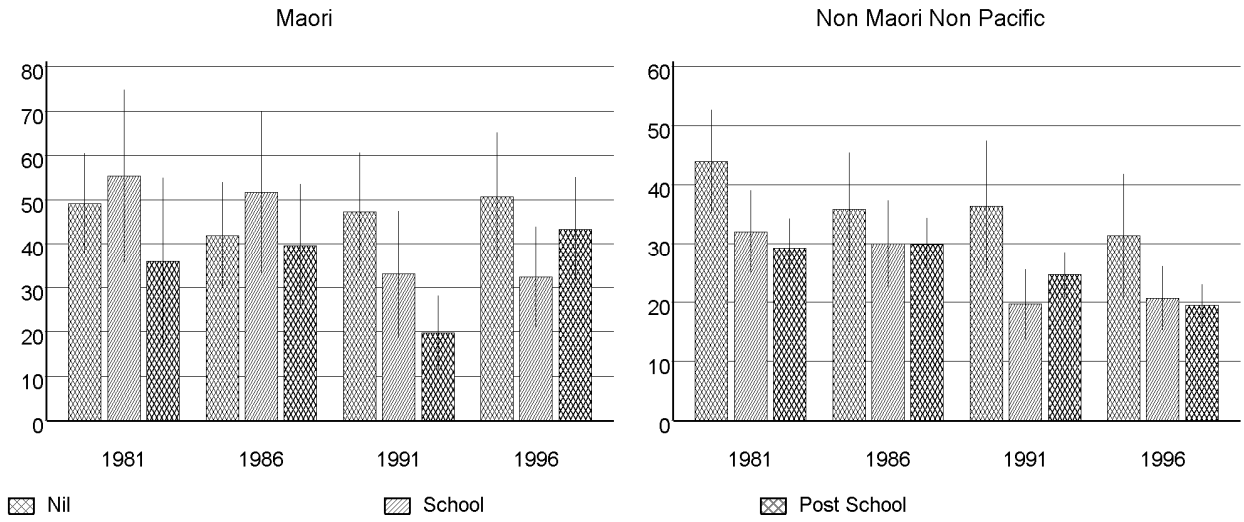
Table 93 Sex specific RII and SII for all-cause mortality by highest education 1-14.

Cohort	RII	95%CI	SII	95%CI
Male				
1981	1.9	(1.2 - 3.1)	28	(2 - 54)
1986	1.4	(0.9 - 2.1)	12	(-8 - 32)
1991	2.0	(1.1 - 3.6)	20	(4 - 36)
1996	1.7	(1.0 - 3.0)	15	(-14 - 44)
P (Trend)	*0.98*		*0.63*	
Female				
1981	2.3	(1.3 - 4.3)	24	(8 - 39)
1986	0.9	(0.4 - 2.0)	-2	(-34 - 30)
1991	2.1	(1.1 - 4.0)	16	(-2 - 35)
1996	1.6	(0.8 - 3.3)	11	(-2 - 24)
P (Trend)	*0.82*		*0.35*	

Age and ethnicity standardised, SII per 100 000 person years

4.8 Variation in All-cause Mortality by Ethnic Group

Figure 48 Ethnic specific all-cause mortality by highest education both sexes ages 1-14 years.



Age and sex standardised, error bars are 95% confidence intervals.
Y axis standard mortality rates /100 000 person years.

Table 94 Ethnic specific all-cause mortality rates per 100 000 person years, SRR and SRD, by highest education both sexes ages 1-14 years.

Cohort	Education Qualification	Weighted Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
Māori								
1981	Nil	108	49	(38 - 61)	1.4	(0.8 - 2.4)	13	(-9 - 35)
	School	48	55	(36 - 75)	1.5	(0.8 - 2.9)	20	(-8 - 47)
	Post School	24	36	(17 - 55)	1.0		0	
1986	Nil	69	42	(30 - 54)	1.1	(0.7 - 1.7)	2	(-16 - 21)
	School	48	52	(33 - 70)	1.3	(0.8 - 2.1)	12	(-11 - 35)
	Post School	45	40	(26 - 54)	1.0		0	
1991	Nil	75	47	(34 - 61)	2.4	(1.4 - 3.9)	27	(12 - 43)
	School	33	33	(19 - 47)	1.7	(0.9 - 3.0)	13	(-3 - 30)
	Post School	30	20	(12 - 28)	1.0		0	
1996	Nil	78	51	(36 - 65)	1.2	(0.8 - 1.7)	7	(-11 - 26)
	School	48	32	(21 - 44)	0.8	(0.5 - 1.2)	-11	(-27 - 6)
	Post School	78	43	(32 - 55)	1.0		0	
Non Māori Non Pacific								
1981	Nil	171	44	(35 - 53)	1.5	(1.2 - 2.0)	15	(5 - 25)
	School	123	32	(25 - 39)	1.1	(0.8 - 1.4)	3	(-6 - 11)
	Post School	204	29	(24 - 34)	1.0		0	
1986	Nil	84	36	(26 - 46)	1.2	(0.9 - 1.6)	6	(-5 - 17)
	School	93	30	(23 - 37)	1.0	(0.8 - 1.3)	0	(-9 - 9)
	Post School	252	30	(25 - 34)	1.0		0	
1991	Nil	63	36	(25 - 48)	1.5	(1.1 - 2.1)	12	(0 - 24)
	School	60	20	(14 - 26)	0.8	(0.6 - 1.1)	-5	(-12 - 2)
	Post School	228	25	(21 - 29)	1.0		0	
1996	Nil	48	31	(21 - 42)	1.6	(1.1 - 2.3)	12	(1 - 23)
	School	78	21	(15 - 26)	1.1	(0.8 - 1.5)	1	(-5 - 8)
	Post School	165	20	(16 - 23)	1.0		0	

Numbers of deaths are rounded to base 3, with a minimum cell size of 6 as per Statistics New Zealand protocol.

Table 95 Ethnic specific RII and SII for all-cause mortality by highest education-both sexes ages 1-14 years.

Cohort	RII	95%CI	SII	95%CI
Māori				
1981	1.3	(0.7 - 2.5)	12	(-23 - 46)
1986	1.0	(0.5 - 2.2)	1	(-45 - 47)
1991	5.3	(1.4 - 20.1)	46	(36 - 56)
1996	1.4	(0.8 - 2.5)	13	(-22 - 48)
P (Trend)	*0.76*		*0.58*	
Non Māori Non Pacific				
1981	2.0	(1.3 - 3.2)	24	(6 - 41)
1986	1.3	(0.8 - 2.0)	8	(-3 - 19)
1991	1.4	(0.9 - 2.2)	8	(-12 - 28)
1996	1.6	(0.9 - 2.7)	10	(-5 - 24)
P (Trend)	*0.50*		*0.49*	

Age and sex standardised, SII per 100 000 person years

Appendix 4 - Executive Summary

Socio-economic inequalities in child mortality have been described in many developed countries in recent decades, however the changing trends of these inequalities have not received much attention. Data from the New Zealand Census-Mortality Study is used in this thesis to determine differences and trends in child mortality by income, education and occupational class. Results for each of the 1981-84, 1986-89, 1991-94 and 1996-99 periods are presented. The differences in mortality are measured in both *absolute* and *relative* terms. Absolute differences are differences in mortality rates between low and high-income people. In this thesis absolute differences are measured using standard rate differences (SRD) and slope index of inequality (SII). Relative differences are the ratio of these mortality rates for low compared to high-income people. In this thesis relative differences are measured using standard rate ratios (SRR) and relative index of inequality (RII). The key results found for child mortality are listed below:

- Mortality rates for children in all socioeconomic groups have fallen over the study period.
- Children of parents with lower socioeconomic position have higher mortality than children of parents with higher socioeconomic position. This was a consistent finding over all periods and all measures of socioeconomic position.
- For income there is evidence that as mortality rates have declined overall, the absolute differences between income groups have tended to be preserved and thus the relative inequalities have increased.
- However trends over time in both absolute and relative inequality by both education and occupational class are not entirely consistent with those found for income. It is not clear whether these variations in trends by education, occupational class and income are due to chance or are real.
- The main cause of death that contributes to these differences, by income, is injury mortality, although other causes of death, except cancer, also contribute to these gradients

All-cause Mortality

Mortality has declined in all income groups in the time period under study, however despite this overall decline in mortality low-income children almost always had higher mortality than children from high-income households. In the most recent cohort (1996-1999) low-income children had higher absolute rates of mortality (29/100 000 compared to 19/100 000) and therefore 60% higher risk of dying than high-income children for all-cause mortality. Children in middle-income households usually had mortality rates midway between the other groups (a gradient) but sometimes their mortality rates were more similar to the

high-income group (a threshold). For income there is evidence that as mortality rates have declined overall, the absolute differences between income groups have been preserved and thus the relative inequalities have increased (for example the relative index of inequality (a relative risk type measure) increased from 1.5 in 1981, to 1.8 in 1996, p-trend 0.06). However when looking at trends by education and occupational class clear trends are not visible for either relative or absolute inequalities.

Injury mortality

Injury mortality rates declined in all income groups over the time period studied. For example low-income children had injury mortality rates of 25/100 000 in 1981, whereas by 1996 this had fallen to 15/100 000. There is a higher mortality risk in the low income, education or occupational class groups compared to the highest group for all these variables. In 1996 low-income children had 130% excess risk of dying from injury compared to high-income children. As injury rates have fallen there have been stable absolute inequalities by income and consequently increasing relative inequalities (e.g. the RII increased from 2.0 in 1981 to 3.0 in 1996). However trends by education and occupational class show different patterns

Other causes of mortality

There were relatively small numbers of deaths in causes of mortality other than injury, so for each category all data were combined to give a pooled risk over all years. These results show there is no evidence that low-income children have an increased risk of cancer mortality (standard rate ratio of low income children compared to high income children 0.84 (95%CI 0.60-1.17)). However there is some evidence supporting a gradient between socioeconomic position and

congenital and 'other' causes of mortality (SRR 1.52 (95%CI 0.95-2.42) and SRR 1.69 (95%CI 1.28-2.63) respectively).

Variation by age in all-cause mortality

There was evidence that mortality declines between 1981 and 1999 seen in the overall results occurred mainly in the 1-4 and 5-9 year olds and there was little decline in mortality rates in the 10-14 age group. Gradients in mortality between high income and low-income children were present in the 1-4 and 5-9 age groups in all socioeconomic variables. These were particularly strong for maternal education. There was little supporting evidence of gradients by any socioeconomic variable in the 10-14 age group, except in 1996 when gradients seemed to emerge. Between 1981 and 1996 population measures of relative and absolute inequality (RII and SII) did not show any clear pattern by any socioeconomic variable in 1-4 and 5-9 year olds, however in the 10-14 age group there was the emergence of a socioeconomic gradient in the 1996 cohort in all measures of SEP.

Variation by sex in all-cause mortality

Mortality rates are higher in males than females, however male mortality has declined faster than female mortality over the time period under study. Differences in mortality between high and low-income children are seen for both boys and girls. Over time by income divergent trends in relative and absolute inequality were seen between boys and girls, however this finding was not replicated in the other measures of SEP.

Variation by ethnic group in all-cause mortality

Māori children have higher mortality rates than non-Māori children. These differences persist within income groups, although there is statistical uncertainty about this because of small numbers. Both ethnic groups show evidence of socioeconomic mortality gradients in all-cause mortality in income, education and occupational class. The changes in these inequalities are difficult to interpret due to wide confidence intervals.

Conclusions and implications

Socioeconomic inequalities in child mortality exist in New Zealand, by most causes and for 1-4 and 5-9 year old children, both sexes and both ethnic groups. These inequalities persisted over the period from 1981-1999. They have not diminished and there is some evidence that by income inequalities have increased, although no clear trends are seen by occupational class and education. Reduction in socioeconomic inequalities in child mortality requires action to eliminate differential exposure to the risk factors of the causes of child mortality, particularly injury mortality, and further exploration of the pathways through which these inequalities are generated.