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Why did so many babies die?:

Infant mortality and causes of death in Dunedin, 1900-1920

Conrad Schumacher

Preface

This study would not have been possible without the permission of the Registrar-General to view the death certificates in the Dunedin registry. Thanks to the staff at the Births, Deaths and Marriages office in Dunedin for enduring my incessant tapping for a month.

Thanks also to my long-suffering supervisor, Barbara Brookes, in particular for her patience in the face of my somewhat erratic work schedule. Thanks also to Dr Hilda Firth for her crash course in epidemiology and her suggestions.

Finally, a big thank you to all those people who somehow managed to put up with me over the year. You guys know who you are; all I can say is kia ora koutou and thanks for all the support.
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Introduction

Most history is written about big people. This is not surprising as most of the ‘things’ that are considered to be worth remembering, or the thoughts that are thought, or the texts that are written, are the products of adults. Indeed for many, many years ‘history’ was almost exclusively about a group of big people who were considered to be, in one way or another, ‘Great Men’. The experiences of other people who did not fit the set of criteria that historians used to create this class were not considered to be worthy of remembrance and were thus not recorded. In more recent times historians have begun to investigate the experiences of a wider selection of people. Now studies of the experiences of groups and individuals that before would have been deemed insignificant by the historians of previous eras are common place. History is no longer the preserve of ‘big’ people in the metaphorical sense, that is to say the powerful, the rich and the thinkers of big ideas. But history remains almost exclusively about the lives and experiences of adults, and thus about ‘big’ people in the literal sense (although some adults may not be as big as others may).

This dissertation is not about big people. Instead, it is about the littlest people of all. This dissertation is about a group of people who left no memoirs, diaries, or descendants. These little people never had the opportunity to grow up to become big people, to leave some record of their own or even to write history dissertations. Each and every one died before they could celebrate their very first birthday. With the passing of time the very existence of most of these little people is only commemorated by two brief entries in official registers of births and deaths. Some of them died before their parents gave them a name of their own. Seven died alone, abandoned and unnamed, but still carefully recorded by the registrars, who entered what details they could; the place where their body was found, their age and sex, the cause of death and their place of burial, with the names of two witnesses to their little funerals.

These are perhaps the saddest little stories of all, but each record is a little tale of woe all of its own. In most of what follows these little stories are aggregated and categorised, grouped together into rates, statistics and proportions, so that the group as a whole may be described. For while each little story is a tragedy in itself, collectively they are parts of a larger, and happier, tale. Over the years the number of times the registrars had to write ‘a few moments’ or ‘eleven months’ in the age column of their registers grew less as infant mortality declined. And these little stories can provide some explanation as to why this was so.
Today we tend to forget that until around the middle of the nineteenth century the infant mortality rate of every country in the world was somewhere between 150 and 200 per thousand live births. In other terms between one in five or one in six babies born died before the age of one year. Now the infant mortality rate in New Zealand is around twelve per thousand live births and in some countries around six per thousand live births. This decline in infant mortality is perhaps one of the most important demographic processes in history. It has underlain the massive growth in the world’s population since the eighteenth century. It has had profound effects on the age structure of the populations of the countries of the world, and thus the social and economic structures of those countries. It may even have altered the way in which we think.

This dissertation seeks to examine a small part of this larger process over a relatively short period of time. It represents a collection of some 2000 records of the brief lives of little people in Dunedin between 1900 and 1920 and asks why the rate of infant mortality declined over the period. Unfortunately, no one clear answer has emerged. However, what follows does provide some small description of this big process, a process largely concerned with the lives, (and sadly the deaths), of infants, and one that had major effects upon society as a whole.
I.

Nutrition, public health and personal healthcare practices: Some explanations for infant mortality decline.

This chapter examines some of the theories that have been advanced to explain the causes behind infant mortality decline, both internationally and in New Zealand. Broadly speaking three general explanations emerge. However, infant mortality decline was a complex process, and Woods, Watterson and Woodward warn against the temptation to view this complex process in "simple monocausal terms". The complex inter-relationship between the various factors is still not fully understood. What follows therefore is merely a survey of some of the general explanations advanced over the years to explain infant mortality decline.

McKeown, Nutrition, and the 'Modern Rise in Population'.

In his book *The Modern Rise of Population* McKeown set out to investigate the causes behind the great rise in the world’s population from the late eighteenth century onwards. Between 1750 and 1975 McKeown estimated that the world population had risen from 750 million to 1000 million in 1830, 2000 million in 1930, 3000 million in 1960 and 4000 million in 1975, (the present figure is now slightly over 6000 million). "That is to say, it took hundreds of thousands of years for the human population to expand to the first thousand million, the second was added in 100 years, the third in 30 and the fourth in 15." It was this trend that McKeown termed the 'Modern Rise in Population'; "the growth of population which began in the late seventeenth and early twentieth century and has continued to the present day.

McKeown argued that the modern rise in population was distinguishable from earlier periods of population growth because of its size, continuity and duration. McKeown investigated the rise in population as a whole, attempting to give a comprehensive picture of the causes behind this rise. McKeown criticised the limited focus of earlier studies, as they tended to focus on only

a small part of the wider trend. Medical historians had previously investigated the behaviour of individual diseases instead of the decline of mortality as a whole. Economic historians had focussed upon the relationship between population growth and industrial and economic development. But McKeown recognised that the only ‘facts’ available for his study were the figures derived from national censuses and the civil registration of births and deaths in England and Wales from 1838. Even this data was less than perfect, due to the difficulties of diagnosis, a problem compounded by changes in the ways in which diseases were thought to occur. Before the advent of national registration no meaningful national estimates of mortality could be made.

To investigate population growth fully McKeown argued that more than straight population statistics are required. Birth rates, death rates and the causes of disease are all important in explaining population growth. Population growth could be due to an increase in the birth rate with a stable death rate, but McKeown found that birth rates had been declining in England and Wales during his period. Population growth must then have been linked to a decline in the death rate. A study of the causes of death helps to explain the nature of this decline in mortality. McKeown concluded that the population growth in England and Wales was due to a decline in mortality, particularly mortality from infectious diseases.

The decline in mortality from infectious diseases identified by McKeown had largely taken place prior to the availability of effective medical treatments for infective diseases. Increased birth rates were unlikely to have much effect on population size before 1900 because of the extremely high infant mortality rates of the time; (the infant mortality rate in England and Wales was around 150 per thousand live births in 1900). “As long as mortality from infection remained high, a rise in the birth rate would have relatively little effect on population growth, because a high proportion of children would die soon after birth.”

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6 Ibid., p.4.  
7 Ibid., p.3.  
8 Ibid., p.7.  
9 Ibid., p.10.  
10 Ibid., p.8.  
11 Ibid., p.7.  
12 Ibid.  
13 Ibid., p.15.  
14 Ibid., p.15.  
15 Ibid., p.39.  
16 Ibid., p.41.
Chapter One: Explanations for infant mortality decline

McKeown divided diseases between those attributable to micro organisms and those not attributable to micro organisms. The first category was subdivided into three subcategories according to the mode of transmission of the micro organisms responsible; airborne, food and water borne, and other.17 Of the overall decline in infant mortality between 1848 and 1971 McKeown found that three-quarters of the decline was attributable to a decline in mortality from conditions attributable to micro organisms.18 The decline in deaths associated with airborne micro organisms was responsible for 40 per cent of the decline in infant mortality as a whole. Twenty-one per cent of the total decline was attributable to a decline in deaths from food and water borne micro organisms. Another 13 per cent was attributable to a decline in mortality from other micro organisms.19 McKeown further concluded that the majority of this decline in deaths attributable to micro organisms occurred before 1901, and could therefore not be attributable to advances in medical science. This decline must therefore have been attributable to some other cause. McKeown argued that improvements in living standards in general, and nutritional status in particular, lay behind this decline.

*Szreter, Rice, and Public Health Measures*

McKeown’s work remains the starting point for any discussion of population growth. McKeown’s ‘negative’ conclusion that medicine played little part in any mortality decline before 1930 remains generally accepted.20 McKeown’s positive conclusion, that improved nutrition was the main factor behind the modern rise in population has been subjected to searching criticism from other academics. The difficulty in finding evidence for the long-term improvement in nutrition that McKeown’s thesis rested upon cast doubt upon the theory as a whole.21

In 1988 Szreter reassessed the period studied by McKeown and claimed to find an increased role for public health works in causing the mortality decline.22 Szreter used the same figures as McKeown but questioned some of McKeown’s interpretations of them. Szreter argued that the efforts of public health reformers in the late-nineteenth century provided a better

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17 Ibid., p.53.
18 Ibid., p.56.
19 Ibid.
20 Although Mercer argues that the introduction of small pox vaccination, “seems likely to have been the main cause of the unprecedented population growth “, in early nineteenth century Europe; Alex Mercer, Disease Mortality and Population in Transition: Epidemiological-Demographic Change in England since the Eighteenth Century as part of a Global Phenomenon, Leicester, 1990, p.73.
explanation for mortality decline than questionable figures for nutritional improvements. Szreter saw the gradual adoption of public health measures by local authorities in Britain as slowly leading to the development of a demographically significant “expansion in the health infrastructure”. Szreter saw McKeown’s denial of the efficacy of improvements in medical technology before around 1930 as a justified response to the ‘medical miracle’ interpretation prevalent before 1970. McKeown’s work was effective in, “thoroughly puncturing the inflated claims to importance, on the grounds of a supposed long history of life-saving achievements, of the medical technocrats”.24

However, Szreter felt that McKeown went too far in his explanation of mortality decline as a passive result of improving living standards. McKeown had effectively discounted the effects of any human agency in improving public health. Szreter argued that detailed local studies were necessary to investigate the link between specific public health measures and changing patterns of mortality and morbidity.25 Such studies have had ambiguous results. Barbara Thompson’s study of infant mortality in Bradford showed no immediate decline after the installation of a sewerage system after 1859.26 This failure was attributable to the low proportion of households actually connected to the system before 1900 and the chronic water shortages that tended to render the system inoperable in the summer months.27

Geoffrey Rice’s study of public health in Christchurch is an example of this type of study in a New Zealand context.28 Rice derived the crude death rate figures (deaths per thousand total population) from the Statistics of New Zealand series between 1874 to 1878 and 1883 to 1911 and from the Christchurch Board of Health reports between 1880 and 1884.29 Crude death rates can be a deceptive measure of the health of a population, as they fail to account for changes in the age structure of the population. Using the classificatory schemes employed in those sources Rice then traced the changes in the patterns of mortality from different causes over the period. The resulting figures were then compared with developments in public health and sanitation. Rice found that

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23 Ibid., p.37.
24 Ibid., p.33.
25 Ibid., p.36.
27 Ibid., p.141.
the trends in Christchurch mortality seemed to contradict the ‘sanitation/public health works’ explanation of mortality decline. Often, “an obvious trend in sanitation follows a fall in death rates, and cannot therefore explain it.” Rice’s study demonstrates the complexities and difficulties in studying mortality rates, and the need for massive amounts of data in order to evaluate the effects, if any, of even seemingly ‘obvious’ factors. Mein Smith and Frost also found little direct correlation between sewers and lower infant mortality rates in their study of Adelaide.

**Personal healthcare practices and the Plunket Society**

By 1991 the process that McKeown had called the ‘Modern Rise in Population’ went under the title ‘health transition’. Instead of the growth of populations demographers concentrated on the shift from a state of high fertility and mortality to lower rates. In Europe the critical point, (where infant mortality rates began to ‘drop like a stone’), occurred sometime in the late nineteenth century. This transition led to a dramatic change in the health profiles of the populations that experienced it.

The initial declines in mortality were due to the control of diseases related to deprivation. As these diseases are more prevalent amongst children, age-selective gains in survival follow. This in turn increases the risk of exposure to chronic diseases, whose contribution to mortality increases accordingly. This process has far reaching effects on both the health profile of the population and the age structure of the population. These in turn contribute to changes in the economic and social structures of the population. The changes in age structure militate against the use of crude death rates. Age standardised rates can reveal the changes in mortality patterns during the health transition in a way that crude death rates cannot. Infant mortality rates (defined

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29 Ibid., Table 1, p.92.
32 Also termed the ‘mortality’ or ‘demographic’ transition.
33 Etienne van de Walle, “How do we define health transition?”, in John Caldwell, Sally Findley, Pat Caldwell, Gigi Santow, Wendy Cosford, Jennifer Braid and Daphne Broers-Freeman, eds., *What we know about health transition: The cultural, social and behavioural determinants of health*, vol.1., Canberra, 1990, p.xiv.
34 Ibid., p.xv.
35 Lado Ruzicka and Penny Kane, “Health transition, the course of mortality and morbidity”, in John Caldwell, Sally Findley, Pat Caldwell, Gigi Santow, Wendy Cosford, Jennifer Braid and Daphne Broers-Freeman, eds., *What we know about health transition: The cultural, social and behavioural determinants of health*, vol.1., Canberra, 1990, p.1.
as the number of infant deaths per thousand live births) are a simple example of an age standardised rate. A decline in infant mortality eventually leads to a change in the age distribution of a society and in turn to the collective experience of health and disease. New Zealand is still experiencing the changes that such transformations bring. The progressive ageing of New Zealand's population has had a fundamental effect on society, and is a direct result of changing patterns of mortality and morbidity.

While the general causes of mortality decline are widely agreed upon, their relative importance is hotly disputed. It seems that some combination of economic and social factors underlie the health transition. But debates continue as to the correlation (if any) between mortality decline and gross national income per head of population, the development of new medical practices, literacy rates and daily calorie consumption. As yet no definitive explanation exists that can adequately account for the varied experiences of the health transition in different countries. Differentials in infant mortality rates with respect to the parents' social or occupational class do exist, even in the developed countries. The disparities between countries are far wider. In 1985 three per cent of all the infants born worldwide were born in countries with an infant mortality rate of above 150 per thousand. A similar proportion lived in countries whose infant mortality rates were around 6 per thousand.

Aside from the factors outlined by McKeown and Szreter, some researchers argue that changes in changes personal health care practices may have contributed to the decline in infant mortality. Ewbank and Preston investigated the possible influence of behavioural changes in the decline in infant and child mortality in the United States between 1900 and 1930. In order to do so Ewbank and Preston looked at contemporary medical and public health literature, recording the emphasis placed on behaviour, the efforts to change it, and the changes purportedly achieved. Then Ewbank and Preston studied the mortality records of the time to see whether the changes in infant and child mortality were consistent with the change in behaviour hypothesis. Ewbank and Preston hypothesised that occupational differentials would be useful in assessing the effects of increased knowledge in reducing infant mortality, as 'professional and managerial

36 Ibid.
37 Ibid., p.5.
38 Ibid., p.9.
39 Ibid., p.21.
40 Douglas C Ewbank and Samuel H Preston, "Personal health behaviour and the decline in infant and child mortality: the United States 1900-1930", in Caldwell et. al., p.117.
41 Ibid.
classes’ were more likely to adopt the new techniques earlier.\footnote{Ibid, p.129.} An earlier study by Preston had already found that the differential in mortality between social classes was somewhat less in the United States around 1900 than was the case in England and Wales.\footnote{Ibid, p.118.}

Ewbank and Preston found that the infant mortality rates for the children of certain groups had declined substantially relative to the national average. Two groups in particular stood out. One group consisted of the children of physicians, dentists and veterinarians; the other of lawyers, judges and teachers. In 1900 both these groups had infant mortality rates some 10 per cent lower than the national average. By 1920 they were between 30 and 40 per cent lower.\footnote{Ibid, p.133.} Ewbank and Preston accept that other socio-economic factors may have been involved, but argue that these figures show that the adoption of better child care practices were responsible for at least some of the decline in infant mortality over the period. While the evidence was not conclusive it was at least consistent with Ewbank and Preston’s hypothesis that the decline in infant mortality rates was not solely a passive response to improving living standards and public health projects.\footnote{Ibid, p.143.}

The adoption of an active attitude against infant mortality was linked to the adoption of the germ theory of disease. Tomes illustrates how the new ‘Gospel of the Germ’ created an optimistic belief that infant mortality could be controlled by individual families.\footnote{Nancy Tomes, The Gospel of Germs: Men, Women and the Microbe in American Life, Cambridge (Massachusetts), 1998, p. 52.} Successive editions of Diseases of Children, (a British technical publication), illustrate the changing view of infant disease. In 1889 infant diarrhoea had been attributed to “the use of sour milk, unripe fruit, the inhalation of sewer gas, emanations from the soil etc”. By 1899 infant diarrhoea was said to be the result of an “infection of the alimentary canal by various toxin-producing bacteria”.\footnote{Ewbank and Preston, p.119.} Popular magazines and newspapers took up the task of educating Americans about infant health. By 1912 columns on baby care appeared in nearly every major American newspaper.\footnote{Ibid, p.125.} But Tomes’ assertion that the importance of personal health care practices had often been overlooked when assessing the causes of mortality decline may not hold true for the New Zealand experience.\footnote{Tomes, p.15.}
Fig. 1.1. New Zealand Infant Mortality Rates, 1872 to 1978

Sources:
For years 1872 to 1922; 1924 New Zealand Official Year-Book, Wellington, 1925, p.144.
For years 1923 to 1931; 1932 New Zealand Official Year-Book, Wellington, 1933, p.135.
For years 1942-1979; John Hyslop, Jan Dowland and Janet Hickling, Health Facts New Zealand, Wellington, 1983, p.50.
Chapter One: Explanations for infant mortality decline

Infant mortality decline in New Zealand

In order to place the results of the study in context it is necessary to consider the national rates of infant mortality over a longer period. The infant mortality rates for New Zealand as a whole between 1872 and 1978 can be seen in figure 1.1. The figures are for non-Maori deaths before 1950. Maori births and deaths were not subject to compulsory registration until 1913, and were not included in the national infant mortality rate until 1950. This would have had the effect of underestimating the total infant mortality rate, as the infant mortality rate amongst Maori was likely to be higher than among non-Maori. The steady decline in infant mortality between 1872 and 1892 was interrupted between 1892 and 1896. Maclean attributes this to a measles epidemic in 1893 and whooping cough epidemics in 1892, 1894 and 1895. The decline in infant mortality between 1907 and 1920 was almost entirely due to a reduction in post neonatal mortality rates. Maclean credited the Plunket society with playing an important role in this decline since its foundation in 1907.

From its foundation in 1907 the Plunket Society was devoted to changing personal health care practices amongst New Zealand mothers. Truby King saw the main causes of infant mortality to be maternal ignorance, a decline in the rate of breast feeding, and generally bad methods of infant care. His solution was to educate mothers in a strict regimen of baby care through the establishment of a national society of nurses trained in Truby King’s methods and publications. Until 1923 the Plunket Society was the only national organisation in New Zealand that was expressly devoted to infant care.

Mein Smith has commented upon the link between Truby King’s views of infant care and his eugenic ideals. By drawing his graphs from 1905 or 1907 Truby King masked the fall in infant mortality before the foundation of the Plunket society. The twelve rules to infant care

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51 Ibid., p.183.
52 Ibid., p.182.
54 Ibid., p.181.
55 Ibid., p.177.
57 Ibid., p. 34.
formed a strict regimen for mothers to follow.\textsuperscript{58} Most of Truby King's methods were derived from American examples, as both Mein Smith and Maclean note.\textsuperscript{59} Mein Smith describes the debate over 'humanized milk' in Australia, propounded as essential by Truby King but which many contemporary Australian doctors saw as unnecessary or absurd.\textsuperscript{60} Mein Smith also raises the important point that ultimately mothers adopted or rejected the various prescriptions of infant care as they saw fit.\textsuperscript{61} This 'maternal autonomy' may have had a greater role in behavioural changes than the efforts of the infant welfare societies.\textsuperscript{62} But regardless of Truby King's motives or personality the official statistics seemed to show that infant mortality had declined at a faster rate in New Zealand after the foundation of the Plunket Society and contemporary observers gave Plunket the credit for that decline.

In 1947 Helen Deem, the medical adviser to the Council of the Plunket society reviewed the infant mortality figures from 1905 to 1946.\textsuperscript{63} Deem also gave the Plunket society the credit for the decline in post-neonatal mortality that had occurred. Deem, like Truby King, made most of her comparisons from 1905 or 1907, the year Plunket was founded, and 1946.\textsuperscript{64} As 1907 was a year of unusually high infant mortality in New Zealand this made the decline appear to be more dramatic. It also masked the 'pre-Plunket' decline in the infant mortality rate.

By 1963 the post-neonatal mortality rate had declined to five per thousand live births. Maclean concluded that no further improvement could be achieved by the methods of the Plunket Society.\textsuperscript{65} Any further improvement would have to come from a decline in neonatal mortality rates. Ford and Pearce found that the majority of infant mortality decline between 1950 and 1990 was due to a reduction in the neonatal mortality rate, in line with Maclean's 1964 prediction.\textsuperscript{66} Ford and Pearce concluded that a static post-neonatal mortality rate now underlies behind New Zealand's declining infant mortality rate.

\textsuperscript{58} Ibid., p. 30; Olssen has called the Plunket regimen a 'prescriptive ideology', see Erik Olssen, "Truby King and the Plunket Society: An Analysis of a Prescriptive Ideology", \textit{New Zealand Journal of History}, 15(1), (1981), pp.3-23.
\textsuperscript{59} Mein, Smith, "Truby King in Australia", p.31; Maclean, p.182.
\textsuperscript{60} Mein Smith, "Truby King in Australia", p.31.
\textsuperscript{62} Ibid., p.191.
\textsuperscript{64} See for example Charts 1 and 2, \textit{Ibid.}, pp. 476-477.
\textsuperscript{65} Maclean, p. 181.
Zealand's comparatively high infant mortality rate. This high post-neonatal rate, "is due entirely to the high and increasing incidence of Suspected Cot Death".

A contemporary opinion: Robert Morse Woodbury

Until 1950 New Zealand had the lowest infant mortality rate for any country in the world. This attracted international attention. Julia Lathrop, head of the United States Children's Bureau, "continually held up [New Zealand's] approach to infant welfare as a model which the United States might emulate." In 1920 Robert Morse Woodbury, the director of statistical research at the United States Children's Bureau, visited New Zealand to study the conditions affecting infant mortality in this country. Woodbury presented his conclusions in a chapter in his book *Infant Mortality and its Causes*. Woodbury noted the decline of infant mortality in New Zealand, and that the decline was largely due to improvements in post neonatal mortality. Woodbury drew a distinction between environmental and social factors, and efforts to reduce infant mortality. He noted the favourable climate, and the comparatively high standard of living when compared with that of contemporary America. Woodbury argued that these factors alone could not explain the decline in infant mortality.

Woodbury concluded that the steady decline in infant mortality in New Zealand between 1875 and 1905 had been due to a combination of factors:

These influences include the gradual increase in medical knowledge of the best methods of disease prevention, the raising of the level of training in the medical profession, the improvements in public sanitation, the gradual education of the public-health work in the Dominion as shown by the increase of the powers and improvements in methods of administration of the health department, and the gradual education of the public in methods of preventing disease and of maintaining health.

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67 Ibid., p.31.
68 Ibid.
69 A status that was often remarked upon in contemporary Year-Books, but that was at least partially due to the exclusion of Maori infants in the national figures, see above.
72 Ibid., p.158.
73 Ibid., p.164.
74 Ibid., p.166.
75 Ibid., p.177.
Woodbury saw three public health measures as being particularly important in the accelerated decline of infant mortality in New Zealand from 1905:

These are the laws regulating midwives and nurses and establishing state maternity hospitals, the protection of children boarded out apart from their mothers afforded by the Infant Life Protection Act, and the infant welfare work of the Royal New Zealand Society for the Health of Women and Children.\(^76\)

In Woodbury’s opinion the system of registration of nurses and midwives had helped raise the standards of maternity care. The St Helen’s Hospitals provided inexpensive care for the mothers whose household income was less than £4 a week.\(^77\) By 1925 one in ten of all births in New Zealand either took place in a St Helen’s Hospital or was attended by a St Helen’s nurse.\(^78\) In the cities where the hospitals were located (including Dunedin), the figure was one in four. Woodbury noted that the urban figures also included births to mothers who live outside the urban areas. “Many mothers come in from the surrounding districts to avail themselves of the hospital accommodation of the cities.”\(^79\) Because of these measures a high standard of maternity care was available to a comparatively high proportion of New Zealand mothers.

Woodbury also examined the effect of the Infant Life Protection Act 1896. A lack of birth data meant that Woodbury could only calculate the infant mortality rate for illegitimate children from 1921. The average rate between 1921 and 1924 was 70.5 per thousand live births.\(^80\) The equivalent rate for legitimate infants was 42.0 per thousand live births.\(^81\) A similar programme of ‘infant life protection’ also operated in New South Wales from 1891. Woodbury credited this scheme as being partially responsible for the reduction of the mortality rate amongst illegitimate infants in New South Wales from 276 per thousand births between 1895-1899 to 108 per thousand births in 1919.\(^82\)

Woodbury was an enthusiastic supporter of the work of Truby King and the Plunket society, seeing it as, “the most important influence in the reduction of the infant mortality rate.”\(^83\) Plunket nurses saw over one quarter of all New Zealand infants by 1919. In the four main centres

\(^{76}\) Ibid., p.166.  
\(^{77}\) Ibid., p.168.  
\(^{78}\) Ibid., p.169.  
\(^{79}\) Ibid.  
\(^{80}\) Ibid., p.171.  
\(^{81}\) See Chapter Four for an examination of the Dunedin figures between 1900 and 1920.  
\(^{82}\) Woodbury, p.171.  
\(^{83}\) Ibid., p.179.
the figure was 43.4 per cent.\textsuperscript{84} The figure for Dunedin may well have been over one half, as Dunedin was the centre of the Plunket network. Woodbury also noted that the society also influenced the care of other infants through its newspaper articles and pamphlets.\textsuperscript{85} Woodbury saw the emphasis upon infant feeding as reducing mortality from gastric and intestinal diseases and the general instruction in infant hygiene as reducing mortality from respiratory diseases.\textsuperscript{86}

**Conclusion**

An investigation of the patterns in infant mortality pattern in Dunedin between 1900 and 1920 provides a valuable addition to what we know about infant mortality decline in New Zealand in the early-twentieth century. While the general trends in national infant mortality have been studied, localised studies of infant mortality in New Zealand do not exist. Dunedin was the centre of the Plunket society, and the influence of the Plunket programme can reasonably supposed to have been felt earlier in Dunedin than in the other main centres. By investigating the stories behind each little person’s death in Dunedin we may find out why less infants died as time went on. The next chapter outlines how the study of infant mortality was conducted, some of the problems encountered, and the measures taken to deal with those problems.

\textsuperscript{86} *Ibid.*
II.

Methodology of the Study

Medical science has improved dramatically over the eighty years since the end of the study period. The technology, training and resources available to the doctors of the first twenty years of the twentieth century would seem terrifyingly crude to the present-day counterparts. The most difficult problem this presents for our purposes the change over time of diagnoses and nomenclature. McKeown found the vagueness and inaccuracies of diagnoses and the changes and nomenclature were such as to make even differentiating between infectious and non-infectious diseases difficult when studying mortality statistics from nineteenth century England and Wales.¹ Although this study focuses on a more recent time period similar problems had to be addressed.

This chapter outlines the difficulties encountered in the course of the study and the methods adopted to solve (or at least live with) them. The results of this process are contained in the next chapter.

History of the ICD and its incorporation into New Zealand

The Tenth Revision of the International Statistical Classification of Diseases and Related Health Problems, ("ICD-10"), represents the culmination of over a century’s efforts to provide an internationally applicable, uniform classification of causes of death and diseases. The first general attempt to classify diseases is usually credited to François Bossier de Lacroix (1706-1777), also known as Sauvages.² However a century earlier John Graunt had subjected the London Bills of Mortality to statistical analysis.³ Graunt endeavoured to estimate the proportion of live-born children who died before the age of six years. As no records of age at death were available Graunt took all deaths attributed to what he regarded as childhood diseases and added half of the deaths attributed to smallpox, swinepox, measles and ‘worms without convulsions’. His resulting

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³ Ibid.
estimate of a 36 per cent mortality rate before the age of six is still regarded as reasonably accurate.4

At the beginning of the nineteenth century the most widely used classification of disease was William Cullen's *Synopsis nosologiae methodicae*, first published in 1785.5 William Farr, the first medical statistician of the Register Office of England and Wales, (established in 1837), found Cullen's classification unsatisfactory and called for its replacement.6 Farr’s call for a uniform system of classification led to international recognition, and in 1853 the first International Statistical Congress requested Farr and Marc d’Espine to prepare such a system. At the next Congress, held in Paris two years later Farr and d’Espine presented two separate lists for approval.7 Farr’s list was arranged under five groups: epidemic diseases, constitutional (general) diseases, local diseases arranged according to anatomical site, developmental diseases and diseases that are the direct result of violence. D’Espine arranged his list along very different lines, looking instead to the ‘nature’ of the disease. The compromise list adopted by the Congress underwent revisions in 1864, 1874, 1880 and 1886. Although never universally adopted the World Health Organisation acknowledges the influence of Farr’s general arrangement.8

Farr and d’Espine’s work was superseded by the International List of Causes of Death in 1891. This system of classification was the direct ancestor of the present international system. The List was largely the work of the French statistician Jacques Bertillon (1851-1922). Bertillon’s influence was so great that the List was commonly called the ‘Bertillon Classification’.9 In the course of decennial revisions the Bertillon classification eventually evolved into the present ICD-10. In 1908 the Bertillon system was adopted in New Zealand.10 Before 1908 New Zealand statistics were presented under a system that divided causes of death according to their causative agent. This division reflected pre-germ theory ideas about disease. Thus diseases were grouped according to their ‘miasmatic’ or ‘zymotic’ natures. Miasmatic diseases were believed to be the result of exposure to noxious or infectious vapours. Zymotic diseases were the result of infection by outside agents.

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4 Ibid.
5 Ibid.
6 Ibid., p.140.
7 Ibid.
8 Ibid.
9 Ibid.
Chapter Two: Methodology

The slow acceptance of germ theory of disease noted by Rice is indicated by the late adoption of the Bertillon system. Older doctors, who as late as 1900, “still clung to miasmatic theories of infection”, may only have been reflecting the official system of classification still in use at the time. 11 The differences between the two systems were such as to make “comparisons of certain causes of mortality between years prior and subsequent to 1908... impossible.”12 Due to the incompatibility between the older classification systems and the ICD-10 it was necessary for my study to start from scratch and re-code all the original records in line with the ICD-10 system.

Under the ICD-10 system different types of disease are organised into chapters. Within each chapter each condition receives a three-character code. For example ‘atelectasis’ falls under the code “P28” which is described as “Other respiratory conditions originating in the perinatal period”. In addition some conditions may be further sub-divided by the addition of a fourth or fifth character. For example infants born prematurely after less than 28 weeks gestation are described by the code “P07.3”, whereas all other premature infants are coded “P07.4”. Both of these examples are codes from Chapter XVI, (“Diseases occurring in the perinatal period”). Two other chapters which figure heavily in the study are Chapter I (“Certain infectious and parasitic diseases”) and Chapter XVIII (“Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified”).

The Sources

In order to gain a full and complete picture of the trends in infant mortality and causes of death during the years between 1900 and 1920 it was necessary to build up a comprehensive record of the deaths during the period. Although official statistics on infant mortality were published throughout the period these do not provide a complete breakdown of infant deaths in Dunedin. The official statistics were compiled under a three-step process, involving registration, certification, and then coding in a form suitable for publication. Each of these steps could conceivably result in error.

As only registered deaths enter the official statistics their reliability depends upon all births and deaths being recorded. Until 1913 the registration of Maori births and deaths was optional. The Births, Deaths and Marriages Registration Act empowered the Governor-General in Council to promulgate regulations requiring Maori births and deaths to be registered. Such regulations were gazetted on the 27 March 1913.\textsuperscript{13} After 1913 Maori births and deaths had to be registered on a separate roll.\textsuperscript{14} If not all deaths are being registered throughout the period trends in the data could be the result of changes in the percentage of registered to total deaths.\textsuperscript{15} Throughout the period non-Maori deaths had to be registered within three days of burial, or if there was an inquest, within three days of that inquest.\textsuperscript{16} Because registration is a statutory duty, only those who do not have funerals are likely to be missed out.\textsuperscript{17} It is highly unlikely that false deaths were registered.

In 1950 New Zealand adopted the form of death certificate recommended by the World Health Organisation.\textsuperscript{18} Under this certificate the causes of death are entered in a more structured manner than the certificates previously in use. This gave rise to some difficulties when the causes were coded as described below. Because the causes were coded from the original entries the problems involved with transferring statistics from one coding system to another were avoided.

The records examined were the Death Certificate registers of the Dunedin area held by the Dunedin office of the Department of Internal Affairs. These are the official records from which official statistics are compiled. A thorough study of the primary records allows the accuracy of these official compilations to be tested. Previously published official compilations of causes of death were not relied upon, largely because of the differences in classification systems. Total birth numbers were obtained from the \textit{Statistics of New Zealand} series for the relevant years.

The death certificate registers are entered on pre-printed forms. Five certificates are entered down each double page spread. The entries contain information on the deceased person under several headings Reading from left to right the first column contains the death certificate number. Certificates are numbered in order of registration throughout the year. The interval

\textsuperscript{13} \textit{New Zealand Gazette}, 1913, vol.1., p.946.
\textsuperscript{14} \textit{New Zealand Official Year-Book}, Wellington, 1920, p.115.
\textsuperscript{15} Ford and Pearce, "Research Note", p.79.
\textsuperscript{16} \textit{Ibid.}, p.80.
\textsuperscript{17} \textit{Ibid.}, p.81.
between death and registration can vary between a day or two and several weeks. For this reason deaths occurring in the last days of one year appear in the next year’s list. In addition certificates that have been altered are required by statute to be cancelled and replaced by a new certificate. Two of the certificates used in the study were officially altered in this way. In both situations the father’s name and occupation had been deleted and replaced as ‘illegitimate’.

The second column from the left contains the date and location of death. Occasionally the date of death is given as an approximation. This is particularly evident in cases of abandonment. The location of death can give rise to certain complications as discussed below. In earlier entries the location is usually given as a suburb, sometimes with a street name. Later certificates become more specific and by 1920 the inclusion of street numbers has become standard. People who died in hospitals have the name of the hospital entered on their entry. Occasionally there may be a pencilled reference to their normal residence (‘from Dunedin’, ‘from 126 Castle St’, and so forth). However this practice is sporadic.

The next column gives the full name of the deceased and their occupation. For infants and children the occupation field is left blank for obvious reasons. Then the sex and age of the deceased is given. The ages of infants are given in fairly precise terms. These entries may range from ‘a few moments’ to ‘11 months’. The next column, probably the most important for our purposes, contains the causes of death (and the length of time that the deceased suffered from them), the name of the certifying person and the date when they last saw the deceased alive. The cause entries range from laconic, one-word entries, (‘marasmus’, ‘prematurity’) to the more extended, (‘Gastroenteritis 2 month Marasmus 4 months, malnutrition, head injury from difficult instrumental labour’). Where the deceased was not attended to by a doctor or other health professional before death the coroner’s verdict or the verdict of a jury is entered.

The next column contains the names of the deceased’s father and mother and the father’s occupation. Children of unmarried couples usually have the entry ‘illegitimate’ in place of the father’s name and the occupation space left blank. In some entries of this type the father’s name and occupation is given, but these are exceptional. The mother’s previous name(s) are given. However the ages of the parents do not appear, nor any indication of the deceased’s number of siblings. The final column on the left-hand page gives the location and date of the deceased’s resting-place.

18 Ibid., p.81.
The first column from the left of the right hand page gives the name and denomination of the Minister conducting the funeral service, or if there is no service the names of two witnesses to the burial. The next column gives the deceased's place of birth and the length of time spent in New Zealand. Some conscientious registrars entered 'life' in this column for infants born in New Zealand. The next column redundant for our purposes, as it gives the name of the deceased's spouse, length of time married, and the age and sex of any surviving children. The last two columns give the name of the informant, (the person supplying the information to the registrar), and the name of the registrar and the date of registration.

**Data Collection**

The initial data collection process involved the collection of information on every death of an infant in the Dunedin area between 1900 and 1920. The information recorded on the first six columns on the left-hand page of each entry and the first two of the right hand page was collected for every entry for an infant whose death was registered in the Dunedin district. The annual death registers were systematically examined and the data entered onto a laptop computer. This initial collection was deliberately over-inclusive for two reasons. No attempt was made to exclude infants from outside the study area. This was to avoid any chance of inadvertently excluding infants who should have been included in the study. Children whose age was recorded as '12 months or '1 year' were also initially included. This was due to fears that the official compilations may have also included such children. These fears ultimately proved to be unfounded. The resulting database contained 2203 entries. The initial set of records is deposited with the Caversham Project at the History Department of the University of Otago. This group of records was then reduced by excluding infants who did not meet the criteria outlined below.

**Locality**

The first criterion on which infants were excluded was the locality where the infants ordinarily resided. With improvements in health care and transportation the tendency for parents in rural districts to come to urban centres to give birth, or to bring their children to town to receive the best available medical care no doubt increases. When an infant born in Waitati is transported to Dunedin hospital and dies there the death will be recorded in the Dunedin register. Arguably the inclusion of such infants will tend to overestimate the infant mortality rate for urban centres, with a corresponding underestimation for rural areas. Strictly speaking such children are not within the urban population. This problem was recognised by the compilers of the official
statistics in the following manner: “Deaths occurring at hospitals have been omitted, except
where deceased had previously resided in one or other of the above boroughs, in which case the
death is counted against the borough of residence.”19 This study proceeds along the same lines.
Thus only the deaths of infants who had previously resided in the study area are included in the
final results. Children born outside Dunedin were therefore excluded.

As the number of births was taken from the Statistics of New Zealand series it was logical
to define the study area in a manner consistent with that series. This is complicated by the way in
which data for the Dunedin metropolitan area was presented over the period. In 1900 the table
entitled “Chief Towns-Births and Deaths” lists figures for. “Dunedin, Caversham, Maori Hill,
Mornington, North-east Valley, Roslyn, St Kilda, South Dunedin, West Harbour.”20 These
divisions were adhered to until the 1905 issue, where Caversham and South Dunedin no longer
appear. Comparing the population figures given for these suburbs and ‘Dunedin’ in 1904 and
1905 reveal that in 1904 the combined estimated population of ‘Dunedin’, ‘Caversham’ and
‘South Dunedin’ was 37 952.21 In 1905 the population of ‘Dunedin’ was given as 38 366,
indicating that Caversham and South Dunedin were incorporated in the figures for ‘Dunedin’.22
Similarly the figures for Northeast Valley are reported as part of Dunedin from 1910, as is Roslyn
from 1913. From 1911 figures for Green Island are reported. By 1916 the list of suburbs has
shrunk to “Dunedin, West Harbour, St Kilda and Green Island”, and from 1917 the table for
“Metropolitan Areas-Births and Deaths” gives details for “Dunedin City and Remainder of area”.

Despite the considerable variation in the subdivision of the Dunedin area the total area
remained stable. For the purposes of this study therefore the ‘Dunedin metropolitan area’
comprises of the area consisting of the suburbs described as Dunedin, Maori Hill, Northeast
Valley, Ravensbourne/West Harbour, South Dunedin, Anderson’s Bay, St Kilda, Caversham,
Mornington, Roslyn, and Green Island/Abbotsford. This area is somewhat smaller than the
“Dunedin registration district” for which figures were also given in the Statistics of New Zealand
series throughout this period.23 Records were excluded from the study where the deceased’s place

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19 Statistics of New Zealand series, tables headed “Chief Towns-Births and Deaths” and “Metropolitan
Areas-Births and Deaths”, 1900-1920.
20 Statistics of the Colony of New Zealand 1900: With Statistics of local government bodies for the year
ended 31st March 1901, Wellington, 1901, p.69.
21 Statistics of the Colony of New Zealand 1904: With Statistics of local government bodies for the year
22 Statistics of the Colony of New Zealand 1905: With Statistics of local government bodies for the year
23 The implications of this difference are explored in Chapter Four.
of residence fell outside of Greater Dunedin. Infants who died in Dunedin hospitals from Waitati, Port Chalmers, Portobello, Mosgiel and so forth were excluded. Place of residence was determined from the marginal notes of ‘from X’ (if available), or from the infant’s place of birth.

One cause of concern is the seemingly intractable problem of infants who were originally born elsewhere but who then resided in Dunedin before dying in hospitals. There may also have been cases of infants born in Dunedin but who died outside the Dunedin area. Unfortunately these problems could not be resolved, and thus represent an unquantifiable source of error in the study. This represents one of the problems inherent in attempting to study characteristics of a mobile population in a defined area. The approach selected is admittedly arbitrary but no superior method could be found.

Age

Another potential source of error is the reported age. ‘Infant’ is defined as a child who has not yet reached the age of 1 year. Children who die aged 350 days and above will almost certainly be registered as having been 1 year old. The inclusion of children aged over twelve months or the exclusion of children aged between eleven months and one year could result in an inaccurate mortality rate. For this reason data was collected on children whose age was given as “1 year”. By comparing the total number of such children with those whose age is given as “10 months” and “11 months” one might assume that some impression may be gained of the tendency (or lack of it) for informants to round up ages of infants. However, given that ages could also be rounded down to ‘1 year’, such a comparison is impossible. The group of children whose age is given as ‘1 year’ thus comprises of those who have almost lived for twelve months, those who have lived for twelve months and not more than thirteen, and presumably some proportion of children who have lived for rather more than twelve months.

Once again the adoption of arbitrary divisions results in intractable problems. Absolute precision in this matter is impossible. As mentioned above it is impossible to gauge how many children had their ages rounded up to ‘1 year’. Even if the proportion of such children could be ascertained there would still exist the problem of deciding which children in the ‘1 year’ class should be counted as infants. 24 This casts doubt upon all official infant mortality rates. Compilers

24 There is one sure way around this problem. By going back to the birth certificates of every child whose age is given as ‘1 year’ and thus obtaining their date of birth it would be possible to precisely determine the
who exclude all children whose age is given as "1 year" will tend to underestimate infant mortality rates, while those who include them (or some proportion of them) run the risk of overestimating such infant mortality rates.

In this study the age of children as entered in the register has been taken as decisive. All children whose age is given as ‘1 year’ are excluded. This may result in some exclusion of children who were ‘in fact’ infants. The gap between the age as represented and the ‘actual’ age is, as with place of residence, unavoidable. The ages at death were coded into three groups, in accordance with the ICD-10 guidelines. Infants who died before seven completed days of life were coded as dying in the ‘early neonatal’ period. Infants who died after seven completed days but before 27 completed days were coded as ‘late neonatal’ deaths. Together these two groups make up the ‘neonatal period’. Infants who lived at least 28 completed days but less than 365 completed days were coded as dying in the ‘post-neonatal’ period.

**Cause Coding**

The causes of death as recorded on the death certificates were coded in accordance with the ICD-10 system. The Twentieth World Health Assembly (1967) defined ‘cause of death’ as:

> All those diseases, morbid conditions or injuries which either resulted in or contributed to death and the circumstances of the accident or violence which produced any such injuries.  

Symptoms and modes of dying (such as ‘heart failure’) are not included in this definition. Where only one cause of death is recorded then that cause is coded. Where there is more than one cause of death then the ‘underlying cause of death’ is coded.

Underlying cause of death is defined as:

(a) the disease or injury which initiated the train of morbid events leading directly to death, or (b) the circumstance of the accident or violence which produced the fatal injury.

For most of the individuals studied only one cause of death is given. These causes were coded under the appropriate three-character code in ICD-10. Where multiple causes are recorded an underlying antecedent cause must be selected, and possibly modified, in accordance with the

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25 The perinatal period also ends seven completed days after birth, but begins at 22 completed weeks of gestation.

guidelines set out in ICD-10. As far as was possible multiple causes of death were coded in accordance with the ICD-10 guidelines.

The World Health Organisation recommends that causes of death be recorded in a form that places the various conditions contributing to death in reverse chronological order (Part I of the certificate) with any other morbid conditions recorded in Part II. The causes of death as recorded in the sources used do not follow this form. However where multiple causes are recorded some of approximation of this type of recording can be reached, due to the practice of recording the duration of illness. The rules for selection and modification of multiple causes are set out below. In the ICD-10 selection rules a 'sequence' consists of, “two or more conditions, each being an acceptable cause of the one above it.”

**The General Principle:**
Where more than one condition is recorded, the condition selected alone in the lowest used line of Part I [i.e. the first occurring condition] should be selected only if it could have given rise to all the conditions entered above it.

**Rule 1:**
If the General Principle does not apply and there is a reported sequence terminating in the condition first entered on the certificate, select the originating cause of this sequence. If there is more than one sequence terminating in the condition mentioned first, select the originating cause of the first mentioned sequence.

**Rule 2:**
If there is no reported sequence terminating in the condition first entered on the certificate, select this first mentioned condition.

**Rule 3:**
If the condition selected by the General Principle or by Rule 1 or Rule 2 is obviously a direct consequence of another reported condition, whether in Part I or Part II select this primary condition.

As an example of the application of the selection rules consider the following cause of death entry from 1904:

"Influenza 10 days Pneumonia, Congestion 7 days Meningitis 3 days."

Rearranged into the ICD-10 format the entry would be in this form:

Meningitis 3 days
Congestion 7 days
Influenza 10 days, Pneumonia

Pneumonia may be assumed to be a complication of any disease. So there is a reported sequence that reads: "Influenza leading to pneumonia, leading to congestion". If this were all that was recorded then Rule 1 would apply and influenza would be selected as the originating cause. However as this sequence does not terminate in the most recent disease (that is the one that would be entered in the top line of the ICD-10 certificate) then Rule 2 applies and therefore meningitis should be selected.

Often the certifying physician reports the causes of death in sequential order, as this example shows:

"Congenital angiomatous tumour Haemorrhage Exhaustion"

In these instances the first mentioned cause coincides with the cause that would be selected under the ICD-10 guidelines. As the ICD-10 manual reminds us, "it should be borne in mind that the medical certifier's statement reflects an informed opinion about the conditions leading to death and about their interrelationships and should not be disregarded lightly."

The Modification Rules

However coding multiple causes does not end with the selection of the originating cause of death. The modification rules are, "intended to improve the usefulness and precision of the mortality data and should be applied after selection of the original antecedent cause". The modification rules are as follows:

**Rule A. Senility and other ill-defined conditions**

Where the selected cause is classifiable to Chapter XVIII (Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified) except for R95 (Sudden infant death syndrome), and a condition classified elsewhere than to R00-94 or R96-R99 is reported on the certificate, reselect the cause of death as if the condition classified to Chapter XVIII had not been reported, except to take account of that condition if it modifies the coding.

**Rule B. Trivial conditions**

Where the selected cause is a trivial condition unlikely to cause death and a more serious condition is reported, reselect the underlying cause as if the trivial condition had not been reported. If the death was the result of an adverse reaction to treatment of the trivial condition, select the adverse reaction.

**Rule C. Linkage**
Where the selected cause is linked by a provision in the classification or in the notes for use in underlying cause mortality coding with one or more of the other conditions on the certificate, code the combination.

Where the linkage provision is only for the combination of one condition specified as due to another, code the combination only when the correct causal relationship is stated or can be inferred from the application of the selection rules.

Where a conflict in linkages occurs, link with the condition that would have been selected if the cause initially selected had not been reported. Make any further linkage that is applicable.

**Rule D. Specificity**

Where the selected cause describes a condition in general terms and a term that provides more precise information about the site or nature of this condition is reported on the certificate, prefer the more informative term. This rule will often apply when the general term becomes an adjective, qualifying the more precise term.

**Rule E. Early and late stages of disease**

Where the selected cause is an early stage of a disease and a more advanced stage of the same disease is reported on the certificate, code to the more advanced stage. This rule does not apply to a “chronic” form reported as due to an “acute” form unless the classification gives special instructions to that effect.

**Rule F. Sequelae**

Where the selected cause is an early form of a condition for which the classification provides a separate “Sequelae of...” category, and there is evidence that death occurred from residual effects of this condition rather than from those of its active phase, code to the appropriate “Sequelae of...” category.33

Consider this example from 1913:

Contracted legs since birth. Collapse after operation for a bone 24 hours

If the operation was intended to correct the first mentioned condition the operation could be considered a direct consequence of the condition.34 The sequence would thus read:

Contracted legs at birth, leading to an operation, leading to collapse

Rule 1 would then lead to the selection of ‘contracted legs at birth’. However modification rules A and B suggest that the operation should be selected instead.

The vast majority of the cases report only a single cause or sequence. These examples represent some of the more difficult causes to classify. Every attempt was made to code the data in accordance with the guidelines given in ICD-10. Inevitably in some instances a single, ill-

defined condition is reported, leading to rather more classifications chapter XVIII than those who
drafted Rule A prescribe. After coding each cause was represented by one three-character code.

**Infant Mortality and Class**

William Farr, the founder of medical statistics commented upon the relationship between
health and socio-economic status in 1851.

Wealth gives the command of the necessaries of life in food, clothing and dwelling; it implies personal purity, and also secures prompt and skilful medical treatment. Poverty represents the sad reverse. Hence the poor as a general rule suffer more than the rich.  

In order to investigate the effects of socio-economic class on mortality figures the information in the father’s occupation was coded along the same scheme as used by the Caversham project. The codes are based upon the description of the father’s occupation and range from ‘1’ for employers and ‘9’ for unskilled workers. Infants whose fathers’ occupation were not recorded (mainly illegitimate infants) were coded ‘10’. As there exists no set of birth numbers by occupational class mortality rates for the various occupational classes could not be produced. The number of illegitimate births for each year were recorded in the Statistics of New Zealand series. This allowed mortality rates to be calculated for illegitimate and non-illegitimate children. The results of these calculations are set out in chapter four.

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35 Quoted in Lado Ruzicka and Penny Kane, “Health transition, the course of mortality and morbidity”, in John Caldwell, Sally Findley, Pat Caldwell, Gigi Santow, Wendy Cosford, Jennifer Braid and Daphne Broers-Freeman, eds., *What we know about health transition: The cultural, social and behavioural determinants of health*, vol. 1., Canberra, 1990, p.9.
Conclusion

The lack of any birth weight data meant that weight specific rates were unobtainable. Instead the study produced a set of infant mortality rates for the period. Every rate is expressed as the number of infant deaths per thousand registered births. Reference is made to the ‘significance’ of some statistics. Significance is a measure of the probability that the observed figure is the result of random fluctuations in the data. The common practice is to say that there is ‘some evidence’ if the test statistic is significant at the 5 per cent level, ‘strong evidence at the one per cent level, and ‘very strong evidence’ at the 0.1 per cent level. If the probability that the observed result could be produced by random fluctuations than there is no evidence that the result is significant. As data was collected on the entire population no estimation of sampling error is given.

III.

Results of the Study

In order to assess which of the three commonly advanced explanations for the decline in infant mortality has the most validity it is necessary to examine the details of that decline. The McKeown thesis, that rising living standards contributed to a decline in mortality from infectious diseases, suggests that the rate of mortality from infectious diseases declined over time. Szreter argues that improvements in sanitation were to a large extent the cause behind the decline in mortality from the mid-nineteenth century onwards. The third explanation stresses the importance of 'personal health care practices' in reducing mortality. This kind of explanation has often been advanced in the New Zealand context, as discussed in chapter one.

McKeown’s thesis would suggest that any decline in overall infant mortality would largely be the result of a decline in mortality from infectious diseases. Szreter’s argument suggests that the decline would be more noticeable in the types of diseases associated with poor sanitation. The ‘improving personal health care practices’ explanation suggests that declines in mortality will first be experienced by the better educated. The main aim of the study of the Dunedin rates of infant mortality was to investigate whether the trends displayed over the period supported one or other of these three hypotheses. In this chapter the results of the study are presented.

The results of this study should be viewed as a part of a longer trend. In effect this study focuses on the trends in infant mortality in one city, Dunedin, over a twenty-year period roughly in the middle of the period represented in figure 1.1. This poses problems when seeking to evaluate the three hypotheses suggested above. It may well be that the trends in infant mortality over the period between 1900 and 1920 were atypical of the longer period.

Infant Mortality Rates

The first step, after collecting all the data and excluding those infants who did not fit the criteria laid out in chapter two, was to calculate the infant mortality rate for the Dunedin area. After excluding those children who were over age and those who were from outside the study area 2003 infants remained. The column headed “Infant Deaths”, in table 3.1 shows the number
of infant deaths in the Dunedin area, as included in the study, for each year between 1900 and 1920. The year with the greatest number of infant deaths, and the highest infant mortality rate, was 1907, where 143 infants died. The year with the lowest number of infant deaths was 1919, where 52 infant deaths were recorded.

Table 3.1. Infant mortality rates in the Dunedin metropolitan area, 1900-1920.

<table>
<thead>
<tr>
<th>Year</th>
<th>Infant deaths (number)</th>
<th>Births (number)</th>
<th>Infant mortality rate</th>
<th>Infant deaths (number)</th>
<th>Births (number)</th>
<th>Infant mortality rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1900</td>
<td>76</td>
<td>1120</td>
<td>67.86</td>
<td>1911</td>
<td>77</td>
<td>1658</td>
</tr>
<tr>
<td>1901</td>
<td>106</td>
<td>1280</td>
<td>82.81</td>
<td>1912</td>
<td>76</td>
<td>1732</td>
</tr>
<tr>
<td>1902</td>
<td>112</td>
<td>1235</td>
<td>90.69</td>
<td>1913</td>
<td>113</td>
<td>1471</td>
</tr>
<tr>
<td>1903</td>
<td>83</td>
<td>1268</td>
<td>93.02</td>
<td>1914</td>
<td>82</td>
<td>1437</td>
</tr>
<tr>
<td>1904</td>
<td>128</td>
<td>1376</td>
<td>93.02</td>
<td>1915</td>
<td>105</td>
<td>1370</td>
</tr>
<tr>
<td>1905</td>
<td>100</td>
<td>1430</td>
<td>69.93</td>
<td>1916</td>
<td>83</td>
<td>1498</td>
</tr>
<tr>
<td>1906</td>
<td>117</td>
<td>1529</td>
<td>76.52</td>
<td>1917</td>
<td>64</td>
<td>1544</td>
</tr>
<tr>
<td>1907</td>
<td>143</td>
<td>1489</td>
<td>96.04</td>
<td>1918</td>
<td>61</td>
<td>1284</td>
</tr>
<tr>
<td>1908</td>
<td>114</td>
<td>1489</td>
<td>76.56</td>
<td>1919</td>
<td>52</td>
<td>1233</td>
</tr>
<tr>
<td>1909</td>
<td>83</td>
<td>1626</td>
<td>51.05</td>
<td>1920</td>
<td>90</td>
<td>1602</td>
</tr>
<tr>
<td>1910</td>
<td>138</td>
<td>1606</td>
<td>85.93</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: 'Deaths' are from the results of the study. 'Births', gives the total number of registered births in the Dunedin area, as recorded in the Statistics of New Zealand series. 'Infant mortality rate' gives the number of deaths per thousand registered births.

Unfortunately the figures for the numbers of infant deaths given here differ somewhat from the official figures recorded in the Statistics of New Zealand series. These differences can be seen in table 3.2. These differences are attributable to the use of different criteria in including or excluding infants from the Dunedin area. As can be seen from table 3.2 for most years the official compilations tend to exclude somewhat more of the infant deaths registered in Dunedin than does my study. This was to some extent expected, as the criteria chosen when excluding infants tended to be over-inclusive. It is possible that the compilers of the official returns had access to additional information about the ordinary place of residence of the infants that was not recorded on the death certificates.

Of more concern is the fact that for certain years the official deaths exceeded the numbers recorded in the study. This inconsistency is particularly disconcerting and perhaps demonstrates the problems that can arise when undertaking studies of this kind. Although the collection of data and the exclusion of infants was done carefully, the resulting figures differ from those obtained by the official compilers, who no doubt worked equally carefully.
Fig. 3.1. Infant mortality rates in the Dunedin metropolitan area, 1900 to 1920

Source: Births and 'official' deaths in the Dunedin area obtained from the *Statistics of New Zealand* series for the years 1900 to 1920 inclusive.
The largest discrepancies occur in the years 1900 and 1912, where the results obtained in both years differ by some 17 per cent from those recorded in the official figures.\(^1\) Taking over the entire period these discrepancies constitute five per cent of the total number of deaths included in the study.\(^2\)

**Table 3.2. Infant deaths in the Dunedin metropolitan area, official and study death numbers. 1900-1920.**

<table>
<thead>
<tr>
<th>Year</th>
<th>Official deaths (number)</th>
<th>Difference</th>
<th>Official deaths (number)</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1900</td>
<td>76</td>
<td>13</td>
<td>77</td>
<td>7</td>
</tr>
<tr>
<td>1901</td>
<td>106</td>
<td>5</td>
<td>76</td>
<td>43</td>
</tr>
<tr>
<td>1902</td>
<td>112</td>
<td>2</td>
<td>113</td>
<td>6</td>
</tr>
<tr>
<td>1903</td>
<td>83</td>
<td>4</td>
<td>82</td>
<td>4</td>
</tr>
<tr>
<td>1904</td>
<td>128</td>
<td>0</td>
<td>105</td>
<td>2</td>
</tr>
<tr>
<td>1905</td>
<td>100</td>
<td>4</td>
<td>82</td>
<td>2</td>
</tr>
<tr>
<td>1906</td>
<td>117</td>
<td>6</td>
<td>64</td>
<td>2</td>
</tr>
<tr>
<td>1907</td>
<td>143</td>
<td>1</td>
<td>61</td>
<td>2</td>
</tr>
<tr>
<td>1908</td>
<td>114</td>
<td>3</td>
<td>52</td>
<td>-4</td>
</tr>
<tr>
<td>1909</td>
<td>83</td>
<td>-2</td>
<td>90</td>
<td>-5</td>
</tr>
<tr>
<td>1910</td>
<td>138</td>
<td>11</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Sources: ‘Official deaths’ gives the number of infant deaths in the Dunedin metropolitan area from *Statistics of New Zealand* series. ‘Deaths’ gives the number of infant deaths from the Dunedin mortality study.

The total numbers of registered births in the Dunedin area were obtained from the *Statistics New Zealand* series. As was mentioned in chapter two, the lack of any birth-weight data means that only the crude infant mortality rates can be derived. Infant mortality rates are expressed as a rate per thousand births. The resulting rates are shown in figure 3.1 The infant mortality rate derived from the official compilations is also shown. As can be seen from the graph in figure 3.1 the two rates follow each other fairly closely. In general the rate derived from the study is usually somewhat greater than the official rate for Dunedin. However at either end of the series the study results are somewhat lower. This is the result of the discrepancies between the official numbers of deaths and the results obtained in the study as noted in the previous section. The infant mortality rates range between a highest of 96.04 per thousand in 1907 (95.37 in the official figures) and a lowest of 41.45 in 1917 (officially 40.16, although the official rate for 1912 was 36.37 somewhat lower than my figure of 43.88). Although generally speaking the rates for

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1 Percentage calculated using the study results as the denominator.
2 Obtained by taking the sum of the absolute values of the discrepancies (100) and dividing by the total number of deaths included in the study (2003).
Fig. 3.2. Infant mortality rates in New Zealand and the Dunedin metropolitan area, 1900 to 1920

the end of the period are somewhat lower than the beginning there does not seem to be any obvious straight-line decrease from year to year.

The national and Dunedin infant mortality rates are shown in figure 3.2. When compared with the national figures the Dunedin infant mortality rate seems to lack the obvious decline that occurred nationally throughout the period. The Pearson's correlation coefficient for these two sets of figures is 0.7857. This indicates that there is very strong evidence that there is a positive linear correlation between the Dunedin infant mortality rate and the national infant mortality rate. This is to be expected, as the Dunedin rate is a subset of the national rate. When the Dunedin figures are looked at in five-year intervals, rather than annually, a decline in infant mortality becomes more readily apparent. The results can be seen in Table 3.3 below.

### Table 3.3. Five-year infant mortality rates in the Dunedin metropolitan area, 1900-1904 to 1915-1919.

<table>
<thead>
<tr>
<th>Period</th>
<th>Registered births (number)</th>
<th>Total deaths (number)</th>
<th>Infant mortality rate (Dunedin)</th>
<th>Infant mortality rate (National)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1900-1904</td>
<td>6279</td>
<td>505</td>
<td>80.43</td>
<td>77.47</td>
</tr>
<tr>
<td>1905-1909</td>
<td>7563</td>
<td>557</td>
<td>73.65</td>
<td>70.22</td>
</tr>
<tr>
<td>1910-1914</td>
<td>7904</td>
<td>486</td>
<td>61.49</td>
<td>56.67</td>
</tr>
<tr>
<td>1915-1919</td>
<td>6929</td>
<td>365</td>
<td>52.68</td>
<td>48.98</td>
</tr>
</tbody>
</table>

Sources: ‘Total births’ gives the sum of the registered births in the Dunedin metropolitan area over each five-year period as recorded in the *Statistics of New Zealand* series.

‘Total deaths’ gives the total number of infant deaths over each five-year period from the Dunedin mortality study.

‘Infant mortality rate (Dunedin)’ gives the rate of infant death per thousand registered births over each five-year period.

‘Infant mortality rate (National)’ gives the average national infant mortality rate over the five-year period.

The Dunedin infant mortality rate over the five-year period between 1915 and 1919 was 34.5 per cent less than the equivalent rate for the period between 1900 and 1904. The decline in the infant mortality rates over time is perhaps better appreciated by taking a wider perspective. It should also be noted that the Dunedin infant mortality rate was consistently higher than the national rate for each five-year period. Overall then it seems that the infant mortality rate in Dunedin did decline over the period to some extent. However the fluctuations from year to year show that this decline may not have taken place at an even rate.

**Deaths in the Neonatal and Post-Neonatal Periods**

The neonatal period commences at birth and ends 28 completed days after birth. The post-neonatal period begins 28 completed days after birth and ends 1 completed year after birth. The mortality rates per thousand live births appear in figure 3.3. This chart also shows the linear
Fig 3.3. Neonatal and post-neonatal mortality rates in the Dunedin metropolitan area, 1900 to 1920

Source: Dunedin mortality study.
trend lines of both sets of rates. As a general rule of thumb deaths during the post-neonatal are more commonly the result of infectious diseases than deaths during the neonatal period. As such post-neonatal deaths can be said to be “more preventable” than neonatal deaths.

As figure 3.3 shows the trend line for the mortality rate during the neonatal period is much flatter than that for the mortality rate during the post-neonatal period. This suggests that the post-neonatal mortality rate was decreasing at a faster rate than the neonatal mortality rate over the period. Such a result would be predicted by all three explanations for the decline in infant mortality rates. Table 3.4 shows the neonatal and post-neonatal mortality rates in Dunedin between 1900-1904 and 1915-1919.

Table 3.4. Neonatal and post-neonatal mortality rates in the Dunedin metropolitan area, 1900-1904 to 1915-1919.

<table>
<thead>
<tr>
<th>Year</th>
<th>Registered births (number)</th>
<th>Neonatal deaths (number)</th>
<th>Neonatal mortality rate</th>
<th>Post-neonatal deaths (number)</th>
<th>Post-neonatal mortality rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1900-1904</td>
<td>6279</td>
<td>218</td>
<td>34.72</td>
<td>287</td>
<td>45.71</td>
</tr>
<tr>
<td>1905-1909</td>
<td>7563</td>
<td>256</td>
<td>33.85</td>
<td>301</td>
<td>39.80</td>
</tr>
<tr>
<td>1910-1914</td>
<td>7904</td>
<td>285</td>
<td>33.53</td>
<td>221</td>
<td>27.96</td>
</tr>
<tr>
<td>1915-1919</td>
<td>6929</td>
<td>212</td>
<td>30.60</td>
<td>153</td>
<td>22.08</td>
</tr>
</tbody>
</table>

Rates per thousand registered births.
Sources: ‘Registered births’ from Statistics of New Zealand series.
‘Neonatal deaths’ and ‘Post-neonatal deaths’ from Dunedin mortality study.

The neonatal mortality rate showed a slight decline over the period. The post-neonatal rate declined dramatically. This trend is in line with the findings of similar studies overseas. In Australia the neonatal mortality rate was 33 and the post-neonatal mortality rate was 63 per thousand live births in 1901-1905.\(^3\) By 1915-1920 these rates were 33 and 32 respectively.\(^4\) Improvements in infant survival in general were largely the result of the reduction of the post-neonatal mortality rate. This indicates that whatever factors contributed to infant mortality decline in this period had a greater effect on post-neonatal deaths than neonatal deaths.

**Causes of Death**

‘Mortality tabulation list 3’, for infant and child mortality in groups the individual cause codes into fifteen categories.\(^5\) Once all the individual entries had been assigned their three

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\(^4\) Ibid.

character codes they were also coded into the codes in the condensed list and into these fourteen groups. Throughout this chapter the following definitions shall be used. ‘Cause code’ refers to the three or four character codes assigned to each condition in the ICD-10 system. ‘Condensed code’ refers to the codes given in the condensed list for tabulating infant and child mortality. ‘Cause group’ refers to the groups of condensed codes set out in that list. The groups generally follow the chapter structure of the ICD-10 coding system.

For example, cases of marasmus receive the cause code ‘E41’ under the ICD-10 system. In the condensed list for infant and child mortality, marasmus receives the condensed code ‘3-024’, described as “Malnutrition and other nutritional deficiencies”. As such marasmus is part of the “Endocrine, nutritional and metabolic diseases” cause group. The fifteen cause groups are described below.

Certain infectious and parasitic diseases
This group contains all the conditions coded A00 to B99 under the ICD-10 system. As such it contains such diseases as tuberculosis, measles, whooping cough and tetanus. It also includes diarrhoea and gastroenteritis of presumed infectious origin. In all 363 of the infants included in the study died of diseases within this group, making it the second largest cause group.

Neoplasms
This group consists of all those conditions coded C00 to D48. It is primarily composed of cancers and similar conditions. Only seven infants died of such conditions in the study.

Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism.
Conditions coded D50 to D89 are coded in this group. Three infants died of disorders included in this group, two of which suffered from haemophilia.

Endocrine, nutritional and metabolic diseases.
This group contains codes E00-E88. The most frequent cause in this group is marasmus. In all 132 infants died of conditions from within this group.

Diseases of the nervous system.
This category covers codes G00-G98, and includes meningitis, which was responsible for the great majority of the 33 deaths that fell under this category.
Diseases of the ear and mastoid process.  
No infants died from any of the causes included in this group (H60-H93).

Diseases of the circulatory system.  
Five children died from diseases coded 100-199. Four of them suffered from morbis cordis.

Diseases of the respiratory system.  
Pneumonia is included within this group, which includes all conditions coded between J00 and J98. Bronchitis is also part of this cause group. This group was responsible for 229 infant deaths in the Dunedin Metropolitan area between 1900 and 1920.

Diseases of the digestive system.  
This group covers codes K00 to K92 and includes gastritis and teething. Fifty-four infants died of diseases from within this group.

Diseases of the genitourinary system.  
This group is comprised of all conditions coded N00-N98. Nine children died from such causes.

Certain conditions originating in the perinatal period.  
This group includes all those infants where premature birth was the underlying cause of death. In all 601 infants died from this cause alone. In addition this group also includes those children who suffered from birth injuries and the vague descriptors ‘debility’ or ‘insufficient vitality’. Made up of codes P00 to P98 this group of conditions was responsible for a total of 784 infant deaths over the period, somewhat over one third of the total number of deaths.

Congenital malformations, deformations and chromosomal abnormalities.  
This group includes all conditions coded Q00 to Q99. Congenital heart defects were the biggest single cause in this group, which also includes spina bifida and hydrocephalus. 176 deaths were attributable to the conditions included in this group.

Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified.  
This is the ‘ill-defined’ category, and as such should only be used when no other code is available. Due to the imprecision of many of the causes of death recorded, the codes in this group had to be used to describe the cause description entered in the register for 156 infants. This was particularly so where the cause of death was entered by the coroner or a jury. Descriptions such as “natural causes” or “convulsions” formed the majority of these cases. Cot death (also known as

---

Fig. 3.4. Infant mortality rates from the six major cause groups in the Dunedin metropolitan area, 1900 to 1920

Source: Dunedin mortality study.
sudden infant death syndrome) falls into this category under the ICD-10 system, but was not a recognised condition until the ninth revision of the ICD-10 system in 1975.7

**All other causes.**
This catch-all category consists of all conditions coded F01-F99, H00-H59, L00-L98 and M00-M99. Ten infants died of conditions coded in this group.

**External causes of morbidity and mortality.**
This category includes all conditions coded V01-Y89. 42 children died from causes within this category. Accidental asphyxiation (including being overlain in bed), lack of sufficient medical care at birth and assault are all causes that fall into this category.

The nine least frequent cause groups were responsible for a total of 163 deaths, some 8.1 per cent of the total. The remaining 92 per cent died of conditions coded in six groups. These six groups (1, 4, 8, 11,12, and 13) are considered individually in turn.

**Mortality rates for the six largest cause groups.**

Figure 3.4 shows the mortality rates from the six largest cause groups. The rates for these categories, as well as the total infant mortality rates for Dunedin and New Zealand are reproduced in Appendix 1. It should be noted that the mortality rates presented in figure 3.4 refer to the number of deaths from each cause group per thousand births. They are no indication to the incidence of such conditions in the population as a whole, nor do they reveal the virulence of each condition. In 1907, for example, the above table reveals that for every thousand children born in Dunedin, (1489 children were born that year), 96 died before the age of one year. The rate of death from the conditions grouped in the ‘certain infectious diseases’ group was 32 per thousand live births, the mortality rate from ‘metabolic disorders’ was 10 per thousand registered births; and so on.

**Mortality from ‘certain infectious diseases.’**

This was the second most frequent group of causes. The rate of mortality from this group of diseases fluctuated between a high of 30.89 deaths from this group of diseases per thousand live births in 1907 and the far lower 1.26 per thousand in 1919. The last time the national infant mortality rate from all causes was as high as 30 per thousand was 1949.8 The rates in 1904 and

---

7 Ford and Pearce, “Infant Mortality in New Zealand”, p.22.
Figure 3.5. Infant mortality rates from diarrhoea, whooping cough and the certain infectious diseases cause group in the Dunedin metropolitan area, 1900 to 1920.

Source: Dunedin mortality study
1910 are almost as high, being 26.16 and 29.89 respectively. Out of every hundred children born in 1907 three would die from this group of causes alone. The total infant mortality rate for that year was 96.04, the highest during the period of the study.

The leading cause of mortality in this group was code A09, ‘Diarrhoea and gastroenteritis of presumed infectious origin’. This was the dreaded ‘summer diarrhoea’. Mein Smith estimated that one half of all Australian infant deaths were caused by this condition. But Mein Smith arrived at this figure by including deaths registered under other conditions, which has not been done here. Every attempt was made to code the causes of death under the ICD-10 guidelines. Some of the infants who ‘in fact’ died of diarrhoea during this period may have been miscoded as a result. But the only ‘facts’ we have are those entered in the cause column of the death certificates. In any event the opinion of the certifying physician represents a professional assessment by someone who actually saw the infant concerned. The cause codes in this study were not arrived at by ‘second guessing’ the entries on the certificate.

In 1907 the rate of mortality from this single cause alone was 24.8 per thousand births. By way of comparison the last time the national infant mortality rate from all causes exceeded this level was in 1953. Figure 3.5 displays the mortality rates from diarrhoea, whooping cough and the rest of the diseases in the certain infectious diseases group. The highest rate recorded by the ‘other group 1’ category was in 1915, at 16.06 per thousand births. By this time the mortality rate from diarrhoea was less than 2 per thousand births, and did not exceed this level for the rest of the period. It would appear then that the decrease in deaths from diarrhoea after around 1910 played a major role in the decline in the mortality rate from this category of diseases. Indeed Woodbury noted that between 1922, 1923 and 1924 no infant deaths were reported in Dunedin from ‘gastric and intestinal diseases’.

Whooping cough is the only other cause in the ‘certain infectious diseases’ group to be responsible for more than 25 deaths over the period. The rate of mortality from whooping cough explains the large figure for the ‘certain infectious diseases’ rate in 1915 as seen in figure 3.5. 1910 and 1915 are the only years in which the number of deaths attributable to this disease

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Chapter Three: Results

The mortality rate from whooping cough exceeded 10. The mortality rate from whooping cough can be seen in figure 3.5. The extremely uneven incidence of this disease indicates its epidemic character.

The highest mortality rates from 'certain infectious diseases' for each five-year period between 1900-1904 to 1915-1919 occurred in 1904, 1907, 1910 and 1915. For ease of reference the rates for whooping cough, diarrhoea, 'certain infectious diseases and the total infant mortality rates for these four years are set out in table 3.5 below.

Table 3.5. Infant mortality rates from whooping cough and diarrhoea in the Dunedin metropolitan area: 1904, 1907, 1910 and 1915.

<table>
<thead>
<tr>
<th>Year</th>
<th>Infant mortality rate from whooping cough</th>
<th>Infant mortality rate from diarrhoea</th>
<th>Infant mortality rate from 'certain infectious diseases'</th>
<th>Infant mortality rate from all causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1904</td>
<td>0.73</td>
<td>20.35</td>
<td>26.16</td>
<td>93.02</td>
</tr>
<tr>
<td>1907</td>
<td>4.07</td>
<td>24.18</td>
<td>30.89</td>
<td>95.37</td>
</tr>
<tr>
<td>1910</td>
<td>11.83</td>
<td>16.81</td>
<td>29.89</td>
<td>79.08</td>
</tr>
<tr>
<td>1915</td>
<td>12.41</td>
<td>1.46</td>
<td>17.52</td>
<td>72.26</td>
</tr>
</tbody>
</table>

Rates per thousand registered births. Source: Dunedin mortality study.

In 1904 there was only one infant death from whooping cough recorded. The high rate of mortality from 'certain infectious diseases' was entirely due to the high mortality rate from diarrhoea. The mortality rate from certain infectious diseases in 1907 was the highest in any single year during the period. As can be seen from the table above it was also directly attributable to a high mortality rate from diarrhoea. In 1910 the high mortality rates from both whooping cough and diarrhoea combined were largely responsible for the second highest mortality rate from certain infectious diseases. By 1915, the last year in which mortality from 'certain infectious diseases' exceeded 15 per thousand births, mortality from diarrhoea had declined to such an extent that only two deaths were attributable to it in that year. In fact, 1915 is the only year where a peak in the overall infant mortality rate is not accompanied by a corresponding peak in the mortality rate from diarrhoea.

Why did the mortality rate from diarrhoea cease to be so closely reflective of the overall infant mortality rate? Some explanation can be found in the type of cause code that A09 is. Code A09 is entitled "Diarrhoea and gastroenteritis of presumed infectious origin". Conditions described as catarrh, enteritis or diarrhoea are included in this code. It does not include conditions where the infectious agent is specified (codes A00-A08), or non-infective diarrhoea (K52.9), or
Fig. 3.6. Infant mortality rates from premature birth in the Dunedin Metropolitan area, 1900 to 1920.

Source: Dunedin mortality study.
non-infective diarrhoea in the perinatal period (P78.3). When the causes of death were coded diarrhoea and enteritis without further qualifications were coded A09 unless the child was less than 1 month old, in which case the code P78.3 was used. The sudden decrease in the rate of deaths from diarrhoea after around 1912 could partially be due to increased specificity in the recording of causes of death by doctors.

There are two other possible explanations for the decline in the mortality rate per thousand births for any specific cause. The first is that fewer infants suffered from this cause as a whole. In relation to diarrhoea, it could have been that fewer infants suffered from the types of conditions that are included in this cause code. The second possible explanation is that while the number or rate of the incidence of such diseases remained similar, the rate of death from the cause declined. More effective treatment or better overall health could have contributed to a lower rate of mortality from each cause. These two trends could also have operated in combination with one another. It is impossible to make any evaluation of these possibilities from mortality statistics alone. Mortality statistics do not reveal the incidence of diseases as a whole. This limitation affects the study as a whole. However in view of the rates presented above it seems clear that by 1920 the conditions coded as diarrhoea were no longer a major cause of infant death in Dunedin. Whatever the factors behind the decline it seems apparent that they were more effective in reducing deaths from diarrhoea after about 1912 than whooping cough.

*Deaths from ‘certain conditions originating in the perinatal period’*

More deaths were recorded from the conditions in this cause group than any other between 1900 and 1920. 601 of them being the result of prematurity. A further 72 were due to causes such as ‘insufficient vitality’ and ‘debility’. The mortality rate from premature birth over the period is shown in figure 3.6. The rate of mortality from the conditions in this cause group was largely static, indicating that it contributed little to the decline in infant mortality over the period.

The general consensus is that health care services of the time could do little to prevent the deaths of premature causes. Woodbury reported that systematic prenatal supervision had halved the neonatal mortality rate in New York City. Woodbury believed that the St Helen’s hospitals had secured a slight decline in neonatal mortality in New Zealand, despite placing little emphasis

on prenatal care. Improvements in nutrition, as suggested by McKeown’s thesis, could have also played a part, by improving the health of mothers. No definitive explanation can be derived without further data, particularly as improved sanitation could also have reduced the mortality rate from premature birth by reducing the risk of maternal infection.

**Deaths from respiratory diseases, congenital malformations, metabolic disorders and ill-defined conditions.**

Due to the small numbers of deaths from these four cause groups annual mortality rates are inappropriate. Instead the total number of deaths from each cause group over five-year periods was divided by the total number of births for the same five-year period to obtain the five-year mortality rate from each cause group. The resulting figures are displayed in table 3.6 below.

<table>
<thead>
<tr>
<th>Year</th>
<th>Infant mortality rate from respiratory diseases</th>
<th>Infant mortality rate from congenital malformations</th>
<th>Infant mortality rate from metabolic disorders</th>
<th>Infant mortality rate from ill-defined conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1900-1904</td>
<td>11.47</td>
<td>4.78</td>
<td>6.21</td>
<td>5.73</td>
</tr>
<tr>
<td>1905-1909</td>
<td>8.59</td>
<td>3.83</td>
<td>6.21</td>
<td>5.82</td>
</tr>
<tr>
<td>1910-1914</td>
<td>6.96</td>
<td>5.69</td>
<td>4.68</td>
<td>2.91</td>
</tr>
<tr>
<td>1915-1919</td>
<td>4.33</td>
<td>7.94</td>
<td>4.04</td>
<td>3.61</td>
</tr>
</tbody>
</table>

Source: Dunedin mortality study

The decline in mortality from diseases of the respiratory system is readily apparent from table 3.6. The most significant causes of death within this group were pneumonia and bronchitis. Of the 229 deaths attributable to this group 130 were due to various forms of pneumonia and 71 were due to bronchitis. This decline is no doubt attributable to similar factors to the decline in certain infectious diseases.

Increased specificity of recording was no doubt behind the decline in the rate of mortality from ill-defined conditions. ‘Congenital malformations’ was probably the cause group that was most effected by the increased specificity. The apparent increase in mortality from congenital malformations may have been due to a change in the cause of death reporting. Infants who died of ‘heart failure’ within the neonatal period are coded in this cause group. If doctors became more specific in attributing cause of death in neonatal infants then more would be placed into this

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Ibid.
group than under ‘ill-defined conditions’. The decline in the number of deaths attributed to ‘convulsions’ and the like may have been due to such changes in diagnoses.

Woodbury provides another explanation for the apparent rise in the national mortality rate from congenital malformations. The Births and Deaths Registration (Amendment) Act 1912 required the registration of stillbirths. "It seems probable therefore, that the increase in the rate from malformations was influenced by the more accurate distinction between births and stillbirths." 14 Woodbury believed that an improvement in the registration of deaths at very early ages also lay behind the rise in mortality from ‘conditions peculiar to early infancy’ between 1872-1874 and 1900-1904. 15

The vast majority of deaths in the metabolic disorders cause group were due to one cause code, E41, which records incidences of ‘marasmus’. There are only two condensed codes in this group. Proponents of the Plunket Society’s efforts would no doubt see the decline in mortality from this cause group as being attributable to the improved methods of infant feeding promoted by Truby King. On the other hand this decline could also be explained by reference to improving water and milk supplies (the public works/ Szreter explanation) or by a general improvement in living standards (after McKeown). Once again without further data it is impossible to decide between the various explanations.

Conclusions

The infant mortality rate amongst infants in the Dunedin metropolitan area did decline between 1900 and 1920. The neonatal mortality rate for these infants declined less than the rate for post neonatal infants. This indicates that whatever factors lay behind the decline in the total infant mortality rate were affecting infants older than one month more than young infants. The relatively static neonatal rate is largely the result of the almost constant mortality rate from deaths due to prematurity.

Most of the decline in the total infant mortality rate over the period occurred in the post-neonatal period. The decline in the certain infectious diseases group is particularly important in this respect. The relative rates of diarrhoea and whooping cough indicate that whatever factors were involved in reducing the mortality rate from the conditions in this period were more

14 Ibid., p. 161.
15 Ibid., p. 160.
effective in reducing deaths from diarrhoea than whooping cough. Woodbury noted the virtual elimination in infant deaths from diarrhoea in Dunedin from 1922.\textsuperscript{16} Deaths from infant diarrhoea are now considered to be a product of deprivation and poor hygiene. As such mortality rates from diarrhoea are more susceptible to 'simple' improvements than mortality rates from whooping cough.

However as all three general hypotheses predict such a result the changes in the mortality rates from the various causes outlined above do not in themselves provide any evidence for selecting between them. Improved nutrition, improved public health infrastructure and improvements in personal health care practices could all have been responsible for the trends outlined above.

All three factors seem to have been at work in Dunedin between 1900 and 1920. The 1912 Cost of Living Commission reported a general improvement in the standards of living New Zealanders in the early twentieth century, indicating an improvement in nutrition. Woodbury also noted that wages had increased faster than inflation between 1900 and 1918, a development that he linked with the system of compulsory arbitration introduced in 1893.\textsuperscript{17} The foundation of the St Helen's hospital in Dunedin in 1906 provided medical assistance to mothers who could not previously have afforded it before and the Dunedin water and sewerage system was largely rebuilt by the Dunedin Drainage and Sewerage Board between 1903 and 1904.\textsuperscript{18} The establishment of Plunket in 1907 and the promotion of Truby King's system of infant care thereafter has traditionally been credited with the decline in the post-neonatal mortality rate by improving personal healthcare practices. Mortality data alone cannot reveal the relative impact of these developments, but it does tell a story of substantial improvements in the chances of survival of infants born in Dunedin, as the risk of dying in the post-neonatal period in particular was reduced.

\textsuperscript{16} Ibid.
\textsuperscript{17} Ibid., p. 165.
For every thousand children born in Dunedin between 1900 and 1904 thirty-five infants had died before reaching the age of one month and a further forty-six had died between the ages of one moth and one year. By 1915 to 1919 the proportion dying in the neonatal period had declined somewhat to thirty-one per thousand births, but the post-neonatal mortality rate had halved to twenty-two per thousand births.
IV.

Socio-economic and Seasonal Differentials

In this chapter the effects of socio-economic and seasonal differentials on infant mortality are considered. The impact of declining fertility on infant mortality rates has often been noted. Unfortunately, only the crude birth rates for each year in the Dunedin metropolitan area could be obtained. Due to this limitation the effects of changes in the fertility rate on infant mortality in Dunedin could not be investigated. The national fertility rate had been declining since at least 1880. Between 1880-2 and 1900-2 the marital fertility rate had declined from 322.1 legitimate births per thousand married women aged 15 to 45 to 243.2 legitimate births per thousand married women in this age group.¹ The average completed family size (the mean number of children that each married woman could expect to bear during their life) of New Zealand women born in the 1870s had been over six, for women born around 1900 this figure had declined to 2.4.² These changes in fertility must have had an effect on the infant mortality rate, but without more specific data such effects can not be investigated here.³

The data collected in the course of the study is capable of sustaining some investigations into the effect of socio-economic differentials and changes in the ‘seasonality’ of infant mortality in Dunedin over this period.

Deaths of Illegitimate children

A ‘rate of risk’ is defined as the rate of morbidity or mortality in a population. An infant mortality rate is an example of a rate of risk; namely the rate that infants face the risk of dying before they reach one year old. By comparing the rate of risk in one population with that of another the ‘comparative risk’ can be established. The comparative risk is simply the proportion between the two rates. Epidemiologists commonly use rates of risk and comparative risk to express the differential between one population and another.

¹ 1913 New Zealand Official Year-Book, Wellington, 1914, p.144.
Ford and Pearce compared the Maori and Non-Maori mortality rates between 1950 and 1991 to estimate the effect of socio-economic differentials upon mortality rates. They found a substantial differential between these two rates, and concluded that socio-economic factors influenced infant mortality rates. As outlined in chapter two no data was available for Maori infants before 1912, and after 1912 the death register did not differentiate between Maori and non-Maori infants. This lack of data meant that Maori and non-Maori rates could not be calculated.

No birth number data is available by occupational class. However, the Statistics of New Zealand series does give the total number of illegitimate births in each registration district for each year. It seems reasonable to assume that illegitimate infants were more likely to come from a less privileged background than their legitimate peers. Many illegitimate infants were separated from their mothers', who often found it difficult to provide for both themselves and their children. The charitable institutions of the time often recommended such separation, as this was believed to reduce the needs of the mother for relief. Whether with their mothers or in foster homes or institutions illegitimate children faced a hard life. Comparisons between illegitimate and legitimate infant mortality rates would seem to be as valid a measure of socio-economic mortality differentials as comparisons between Maori and Non-Maori infant mortality rates. Ford and Pearce noted the limitations of using the latter comparison as a measure of socio-economic differentials, but were also constrained by the lack of more significant data.

Unfortunately, the registration district for Dunedin is larger than the metropolitan area used in the study. In order to estimate the total number of illegitimate births in the study area it was necessary to assume that the rate of illegitimate births in the study area would be in the same proportion to the total number of births in the registration district. The number of illegitimate births in the study area was therefore estimated by multiplying the number of illegitimate births in the registration district by the proportion of total births in the Dunedin metropolitan area for each year. Over the 20 years between 1900 and 1919 (inclusive) the number of births in the Dunedin

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5 Throughout this section the terms 'illegitimate' and 'legitimate' shall be used, in line with the contemporary usage. For the effects of the Legitimation Act see below.
7 Ibid., p.85.
metropolitan area was on average 92 per cent of the total number of births in the Dunedin registration district (the figure varied between 84 and 96 per cent for each year). Once this figure is known the rate of infant mortality for illegitimate infants can be established. The number of deaths of illegitimate children was obtained from the study.

The assumption that the proportion of illegitimate births to total births in the Dunedin metropolitan area was the same as the Dunedin registration district may be an unwarranted one. It may have been that the urban area had a higher proportion of illegitimate births than the remainder of the registration district. For this reason table A2.2 presents the same calculations, this time assuming that all the illegitimate births occurred in the Dunedin metropolitan area. This has the effect of reducing the figure for the mortality rate amongst illegitimate infants, (by increasing the birth number denominator), and comparative risk, (due to a lower illegitimate mortality rate) by an average of around ten percent.

The proportion of illegitimate births to total births in the study area over the time is shown in Table 4.1. Figures are given for both the proportional and the maximal estimates of the number of illegitimate births in the Dunedin metropolitan area.

| Table 4.1. Estimates of legitimate and illegitimate births in the Dunedin Metropolitan area, 1900-1919, (i.) proportional estimate and (ii.) maximal estimate. |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
|                                 | 1900-1904       | 1905-1909       | 1910-1914       | 1915-1919       |
| Total births (Dunedin metropolitan area) | 6279           | 7563           | 7904           | 6929           |
| (i.) Proportional estimate     |                 |                 |                 |                 |
| Illegitimate births (number)    | 408            | 502            | 498            | 394            |
| Legitimate births (number)      | 5871           | 7061           | 7406           | 6635           |
| Proportion of illegitimate births to total births (percentage) | 6.50          | 6.64          | 6.30          | 5.69          |
| (ii.) Maximal estimate         |                 |                 |                 |                 |
| Illegitimate births (number)    | 445            | 538            | 548            | 439            |
| Legitimate births (number)      | 5834           | 7025           | 7356           | 6490           |
| Proportion of illegitimate births to total births (percentage) | 7.09          | 7.11          | 6.93          | 6.34          |


9 For ease of reference the figures so derived shall be called the “proportional estimate” of illegitimate births.
10 As this is the highest possible number of illegitimate births that could have occurred in the metropolitan area this estimate shall be termed the “maximal estimate”.
11 The actual figures are 9.4 per cent and 10.3 for the illegitimate mortality rate and the comparative risk respectively.
The figures for the mortality rate amongst legitimate infants are less affected (0.7 per cent lower) because of the larger numbers involved. The infant mortality rate figures obtained under the proportional estimate are shown in Table 4.2 below. Once again the mortality rates were calculated over five year periods because of the relatively low number of infants, (the full figures are reproduced in Appendix 2.).

Table 4.2. Illegitimate and legitimate mortality rates in Dunedin, 1900-1919.

<table>
<thead>
<tr>
<th>Year</th>
<th>Illegitimate Mortality Rate</th>
<th>Legitimate Mortality Rate</th>
<th>Comparative Rate of Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>1900-1904</td>
<td>191.24</td>
<td>72.73</td>
<td>2.63</td>
</tr>
<tr>
<td>1905-1909</td>
<td>177.23</td>
<td>66.28</td>
<td>2.67</td>
</tr>
<tr>
<td>1910-1915</td>
<td>136.61</td>
<td>56.44</td>
<td>2.42</td>
</tr>
<tr>
<td>1914-1919</td>
<td>104.06</td>
<td>49.58</td>
<td>2.1</td>
</tr>
</tbody>
</table>

Sources: See Table A2.1 in Appendix 2.

Table 4.2 indicates that after 1910 the mortality rate amongst illegitimate infants was decreasing at a faster rate than amongst their legitimate peers. The change in the relative rate of risk shows that this decline was not merely a function of the initial high rate of mortality amongst illegitimate children. Between 1900-1904 and 1905-1909 the mortality rate amongst illegitimate infants declined from 191.24 to 177.23. However, the comparative risk between illegitimate and legitimate infants increased slightly. After 1910 the comparative risk declines, indicating that the illegitimate mortality rate was improving relative to the legitimate mortality rate. Ewbank and Preston found that the gap between the 'best-educated' classes and the average rate in America widened between 1900 and 1930. The figures in table 4.2 suggest that the gap between illegitimate infants and legitimate infants in Dunedin narrowed after 1910.

The Legitimation Act 1894 could have lain behind the decrease in the proportion of illegitimate births. Under this Act children born out of wedlock whose parents married within twelve months of their births could be deemed legitimate by application. A total of 78 illegitimate births...
children were 'legitimised in Dunedin between 1900 and 1910. However only 11 children were 'legitimised' in the Dunedin area within one year of birth between 1890 and 1910. The Legitimation Act may have operated to reduce the number of illegitimate births. As such the Act may be partially behind the reduction in the number and proportion of illegitimate births seen in table 4.2.

However, it seems reasonable to assume the Act’s effect on the mortality rate amongst illegitimate infants, if any, would be to increase the mortality rate. Infants who died before the age of one were unlikely to be legitimised. Thus the numerator for the mortality rate amongst illegitimate children would be unaffected. Legitimisation would have reduced the number of illegitimate births recorded, and a reduction in that number is apparent. This would reduce the denominator for the mortality rate amongst illegitimate children and thus increase the rate accordingly. There were certainly no 'post mortem' 'legitimisations' of infants in the Dunedin register over this period. To do so would require the cancelling of the original death certificate and the recording of a new one. In fact the only infant death certificate cancelled in the Dunedin register over the period operated in the other direction. The infant concerned was from outside the study area so was excluded after the initial records had been compiled. While originally recorded as a legitimate infant, with the father’s details, the original certificate was cancelled and replaced with a new entry classing the child as illegitimate.

If the reduction in the mortality rate amongst illegitimate infants shown in table 4.2 cannot have been the result of the Legitimation Act some other factor must have been at work. By identifying the cause group(s) that contributed most to the decline in the mortality rate amongst illegitimate infants we may be better able to evaluate which of the explanations for infant mortality decline is at work. Cause groups rather than cause codes will be analysed here because of the relatively small numbers involved.

More than 30 illegitimate infants died from the conditions in three cause groups between 1900 and 1919. These cause groups were: certain infectious diseases (the group containing infant diarrhoea), conditions arising in the perinatal period (the group containing prematurity), and metabolic disorders. Altogether 182 illegitimate infants died from the conditions in these three

14 Ibid., p.106.
15 For obvious reasons this child can not be identified.
groups, representing nearly two thirds (65.9 per cent) of the total number of illegitimate infant deaths (276).

Again the mortality rate for all other infants is shown for each cause group and the comparative risk between the two populations. The figures given are based upon the proportional estimate for illegitimate births only. It should be remembered that this estimate tends to increase the differential between the illegitimate and the legitimate populations when compared with the maximal estimate figures. Table 4.3 below shows the mortality rates amongst illegitimate and legitimate infants from the conditions in the certain infectious diseases cause group.

Table 4.3. Mortality rates from certain infectious diseases amongst illegitimate and legitimate infants in the Dunedin metropolitan area, 1900-1904 to 1915-1919.

<table>
<thead>
<tr>
<th></th>
<th>1900-1904</th>
<th>1905-1909</th>
<th>1910-1914</th>
<th>1915-1919*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality rate amongst illegitimate infants (illegitimate deaths per thousand illegitimate births) n=55</td>
<td>36.76</td>
<td>29.88</td>
<td>42.17</td>
<td>10.15</td>
</tr>
<tr>
<td>Mortality rate amongst legitimate infants (legitimate deaths per thousand legitimate births) n=298</td>
<td>13.97</td>
<td>14.30</td>
<td>9.72</td>
<td>6.58</td>
</tr>
<tr>
<td>Comparative rate of risk (illegitimate mortality rate/legitimate mortality rate)</td>
<td>2.63</td>
<td>2.09</td>
<td>4.34</td>
<td>1.54</td>
</tr>
</tbody>
</table>

Source: Dunedin mortality study
*The total number of illegitimate infant deaths from certain infectious diseases for this period was 4 (the number of legitimate deaths for the same period was 43).

The mortality rate from certain infectious diseases amongst legitimate infants declines at a relatively steady rate. They rate amongst illegitimate infants shows more variation, almost certainly because of the smaller numbers involved. The low figure for mortality from certain infectious diseases amongst illegitimate infants between 1915-1919 suggests that this group benefited to a disproportionate extent from the elimination of diarrhoea as a major cause of death amongst Dunedin infants. The reduction in the mortality rate for illegitimate causes from this cause group made up almost all of the decline in overall infant mortality amongst illegitimate infants between 1910-1914 and 1915-1919.

The decline in mortality from the certain infectious diseases cause group can be contrasted with the position in relation to conditions arising in the perinatal period. Once again the figures are based on the proportional estimate of illegitimate births.
Table 4.4. Mortality rates from conditions arising in the perinatal period amongst illegitimate and legitimate infants in the Dunedin metropolitan area, 1900-1904 to 1915-1919.

<table>
<thead>
<tr>
<th></th>
<th>1900-1904</th>
<th>1905-1909</th>
<th>1910-1914</th>
<th>1915-1919</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality rate amongst illegitimate infants</td>
<td>53.92</td>
<td>57.77</td>
<td>54.22</td>
<td>43.15</td>
</tr>
<tr>
<td>(illegitimate deaths per thousand illegitimate births) n=95</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality rate amongst legitimate infants</td>
<td>27.59</td>
<td>25.35</td>
<td>23.36</td>
<td>20.81</td>
</tr>
<tr>
<td>(legitimate deaths per thousand legitimate births) n=650</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Comparative rate of risk</td>
<td>1.95</td>
<td>2.28</td>
<td>2.32</td>
<td>2.07</td>
</tr>
</tbody>
</table>

Table 4.4 reveals that the mortality rate from conditions arising in the perinatal period was consistently higher amongst illegitimate children. While the disparity between these two groups in certain infectious diseases declined the illegitimate population maintained a consistently high mortality rate from perinatal conditions. Once again whatever factors contributed to the decline in mortality had little effect on this group of conditions.

Woodbury suggested that the Infant Life Protection Acts were responsible for a decline in illegitimate mortality from 1921. This legislation regulated the placement of children to foster homes, requiring the registration of foster parents and, from 1906, the inspection of foster homes by trained nurses.16 As the first Infant Life Protection Act had been passed in 1893 it seems reasonable to assume that their effect had been operating before this time. Woodbury certainly believed so, and pointed to figures from New South Wales to support this conclusion.17 The mortality rate amongst infants in foster homes declined nationally from 143 per thousand in 1908 to 21 per thousand in 1918.18 A decline of this magnitude must have effected the rate of mortality amongst illegitimate infants in Dunedin.

An improvement in the care of infants in foster homes therefore seems to have been the main cause behind the reduction in the differential between the mortality rates between illegitimate and legitimate infants. Although the gap between these two populations reduced by some 20 per cent there remained a huge disparity between them. An infant born to an unmarried mother in Dunedin between 1915-1919 still faced more than twice the risk of dying than their legitimate peers. Although the mortality rate amongst illegitimate infants had declined by more

16 Woodbury, p.170.
17 Ibid., p.171.
18 Ibid., p.170.
than 45 per cent, it was still higher than 100 per thousand births. It seems reasonable to conclude that a similar differential existed between 'rich' and 'poor' infants.

**Seasonal Fluctuations**

One effect of the virtual elimination of diarrhoea as a major cause of death was to alter the 'seasonality' of infant mortality in Dunedin. This section follows the approach taken by Naomi Williams in her study of infant mortality in Sheffield.\(^{19}\) The indexed figures show the amount that the actual seasonal figures differ from what one would expect to find if the mortality rate was constant across all seasons. The expected figure for each season is derived by taking the total number of deaths that were recorded for the period, divided by the total number of days over the period (1826) and then multiplying this figure by the total number of days in each three month interval.\(^{20}\)

Between 1900 and 1904 a total of 505 infants died in the Dunedin metropolitan area. If the deaths had been distributed at an even rate across the seasons we could expect 125 infants to have died between January and March, 126 to have died between April and June, and 127 to have died between both July and September and October and December. These numbers vary because the total number of days over five years in each seasonal interval ranges between 451 between January and March and 460 between July and September and October and December. In fact the actual number of deaths in each season was 133, 124, 129 and 119. The resulting index figures can be seen in table 4.5.

The Dunedin figures show far less seasonality than Williams's Sheffield ones for 1870-1871. In Williams's figures the summer (July-September for the Northern Hemisphere) index for all causes is around 150. This was due to diarrhoea indices of over 300. Williams gives the Sheffield indices by age group. Due to small numbers this is inappropriate for my figures. However by giving the indices over time we can see how the seasonality changes over time in Dunedin.

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\(^{19}\) Naomi Williams, "Death in its Season: Class, Environment and the Mortality of Infants in Nineteenth-Century Sheffield", *Social History of Medicine*, 5(2) (1992), pp. 71-94.

\(^{20}\) Because 1900 was not a leap year each five-year period between 1900-1904 and 1915-1919 had the same number of days.
Table 4.5. The seasonality of infant deaths in Dunedin by five-year period for: (i) all causes, (ii) diarrhoea of presumed infectious origin, and (iii) premature birth. (Index = 100; i.e. total deaths distributed by quarter in proportion to the number of days in each quarter.)

<table>
<thead>
<tr>
<th></th>
<th>Total deaths (number)</th>
<th>Jan-Mar (index)</th>
<th>Apr-Jun (index)</th>
<th>Jul-Sep (index)</th>
<th>Oct-Dec (index)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(i). all causes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1900-1904</td>
<td>505</td>
<td>107</td>
<td>99</td>
<td>101</td>
<td>94</td>
</tr>
<tr>
<td>1905-1909*</td>
<td>557</td>
<td>122</td>
<td>85</td>
<td>108</td>
<td>85</td>
</tr>
<tr>
<td>1910-1914</td>
<td>348</td>
<td>109</td>
<td>99</td>
<td>100</td>
<td>91</td>
</tr>
<tr>
<td>1915-1919</td>
<td>365</td>
<td>97</td>
<td>90</td>
<td>122</td>
<td>91</td>
</tr>
<tr>
<td>(ii). diarrhoea</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1900-1904**</td>
<td>64</td>
<td>196</td>
<td>94</td>
<td>37</td>
<td>74</td>
</tr>
<tr>
<td>1905-1909**</td>
<td>80</td>
<td>253</td>
<td>75</td>
<td>55</td>
<td>20</td>
</tr>
<tr>
<td>1910-1914**</td>
<td>59</td>
<td>254</td>
<td>61</td>
<td>27</td>
<td>61</td>
</tr>
<tr>
<td>1915-1919†</td>
<td>11</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>(iii). premature birth</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1900-1904*</td>
<td>135</td>
<td>84</td>
<td>92</td>
<td>100</td>
<td>123</td>
</tr>
<tr>
<td>1905-1909</td>
<td>159</td>
<td>87</td>
<td>93</td>
<td>102</td>
<td>117</td>
</tr>
<tr>
<td>1910-1914**</td>
<td>148</td>
<td>88</td>
<td>152</td>
<td>94</td>
<td>67</td>
</tr>
<tr>
<td>1915-1919</td>
<td>109</td>
<td>67</td>
<td>103</td>
<td>127</td>
<td>102</td>
</tr>
</tbody>
</table>

*Indicates significant variation at the five per cent level (using Chi-squared test).
**Indicates significant variation at the one per cent level (using Chi-squared test).
† The small number of deaths from diarrhoea in this period makes index figures inappropriate.

In the deaths from all causes section only one period exhibits significant variation from the expected figures. This coincides with the greatest number of deaths from diarrhoea. There is strong evidence for seasonal variation in the diarrhoea figures throughout the period. Infant diarrhoea was recognised as a condition that predominately occurred in summer by contemporaries. Premature birth is not so seasonal in its distribution. In four sets of figures the premature birth index peaks in three different seasons. Unlike Sheffield in 1870 infant mortality in Dunedin did not have a marked seasonal pattern between 1900 and 1920.

**Areas for further study**

Because of a lack of birth data by occupational class no other comparison of rates of risk can be made at this time. One obvious area of interest would be further studies of socio-economic factors relating to mortality decline. The mortality data already collected provides details of the occupation of the father of each infant (unless the infant is illegitimate). By collecting data from

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the birth certificates in Dunedin over this between 1900 and 1920 (some 30,227 infants) a set of
the annual numbers of births in each occupational class could be derived.

Another question that could only be investigated by the collection of further data is the
role of the Plunket society in the decline of infant mortality. To do so we need to establish which
infants who died were 'Plunket babies' and the total number of infants in Dunedin who were
visited for by Plunket nurses each year. The remainder of infants would constitute the group
against which the rate of risk would be compared. This comparison should be made between post-
neonatal mortality rates, as the Plunket programme would have had little effect on neonatal
mortality. By linking the records from all three sources the effect of socio-economic differentials
could be controlled when making comparisons between Plunket and non-Plunket infants. Such an
exercise would allow us to see whether attendance by a Plunket nurse had any significant effect
on infant survival.
Conclusion

'Anyone can have big ideas,' said Concepcion. 'I have some big ideas, and most of them I thought of for myself, and then I found out that others have thought the same, and then I found out that other people have big ideas that are exactly the opposite. And when I think about it even more, I decide that only small ideas can be true, and the big ideas are too big to fit inside anybody's mind, so there is no point in trying to have them.'


This dissertation has come up against the problem identified by Concepcion. For each individual little person the death records give us what the registrar and the certifying physician (or coroner or jury) recorded as the 'truth'. In a sense each of these individual records gives an interpretation of the cause of death. Even in these individual records some element of doubt may arise. Chapter two surveyed some of the problems involved in trying to ascertain who exactly an infant, where they came from, and how to interpret the cause of death given. The data at an individual level obtained in the study cannot be said to be 'perfect', in any 'absolute' sense. Aggregating the data under arbitrary categories, so that generalisations about the data can be made, introduces new sources of doubt. The shift from small ideas to big ones is not an easy one.

Most of the explanations advanced to explain infant mortality decline have tended to downplay the role of individual agency in the decline of infant mortality. Mein Smith is no doubt correct to say that mothers deserve more credit for improved infant survival. At the individual level the choices that mothers made, the autonomy that they exercised in deciding how to care for their babies, was the most important factor in each individual baby’s experience. In a very real sense the choices made by individual mothers constituted an ‘invisible hand’ promoting infant survival throughout society as a whole.

But while mothers exercised some autonomy in their choices as to how to care for their infants, this autonomy was necessarily constrained. Each mother had to deal with the exigencies of their own situation. No doubt every mother wished to live up to some constructed ideal (whether constructed individually, through family tradition or by some expert in ‘mothercraft’) of the perfect mother. It would be perverse to suggest otherwise. But each mother had to deal with the constraints imposed by time, environment and financial circumstances. During this period unmarried mothers felt these constraints the most. At a time when illegitimacy was seen as a
problem, and the goal of welfare was to reduce dependency, unmarried mothers were faced with near insurmountable odds. To transcend them required superhuman effort, or at least comfortable independent means. The high rate of mortality amongst illegitimate infants was not the result of some ‘moral degeneracy’, but a reflection of the constraints imposed upon unmarried mothers by the society of the time. Seven mothers in Dunedin over this period were so pressured (for whatever reason) that they felt the only way out was to abandon their babies.

The traditional explanations of infant mortality, if viewed from this perspective, are no longer ‘causes in themselves’. Improvements in sanitation, or living standards, or access to healthcare services, did not reduce infant mortality directly. Instead they provided more mothers with more opportunity to take advantage of them when caring for their babies. The teachings of organisations like the Plunket society meant nothing in terms of infant survival unless mothers chose (or could be persuaded) to carry them into practice. Even then the various explanations must have acted concurrently. It would be well nigh impossible to carry out the Plunket programme without clean water, adequate time and money, and effective plumbing.

This study does not conclude with a general explanation of the causation of the decline in infant mortality rates in Dunedin as a whole. However, the results of this study can be built upon to measure at least one of the major explanations commonly ascribed to infant mortality decline in New Zealand, as outlined in chapter four. A study on the differentials in post-neonatal mortality rates between ‘Plunket babies’ and other babies over different occupational classes and over time would be an important contribution to our understanding of infant mortality decline in New Zealand. Did it make a difference in terms of infant survival if mothers, whose personal situations were in all other respects (fairly) equal, chose to take their baby to Plunket or not? The small ideas about these little people in this dissertation may thus provide a base for examining this larger question.
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*Articles and theses:*


## Appendix 1

### Mortality rates from the six main cause groups

**Table A1.1. Annual infant mortality rates from the six major ICD-10 cause groups, in the Dunedin metropolitan area, 1900 to 1920.**

<table>
<thead>
<tr>
<th>Year</th>
<th>Total infant mortality rate from all causes</th>
<th>Infant mortality rate from 'certain infectious diseases'</th>
<th>Infant mortality rate from 'metabolic disorders'</th>
<th>Infant mortality rate from 'respiratory conditions'</th>
<th>Infant mortality rate from 'certain conditions arising in the perinatal period'</th>
<th>Infant mortality rate from 'congenital malformations'</th>
<th>Infant mortality rate from 'ill-defined conditions'</th>
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<tbody>
<tr>
<td>1900</td>
<td>67.86</td>
<td>10.71</td>
<td>3.57</td>
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<td>22.32</td>
<td>9.82</td>
<td>5.36</td>
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<td>1901</td>
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<td>7.03</td>
<td>14.84</td>
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<td>10.16</td>
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<td>31.47</td>
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<td>5.18</td>
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<td>4.37</td>
<td>24.34</td>
<td>10.61</td>
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</table>

Rates per thousand registered births.

Source: Birth figures used to calculate these rates from *Statistics of New Zealand* series.

Death numbers from Dunedin infant mortality study.

NB. The 'total rate' figure is **not** the sum of the other six columns, as it contains deaths from the nine other cause groups in the ICD-10 scheme.
## Appendix 2. Illegitimate and Legitimate Mortality Rates

### Table A2.1. Illegitimate and legitimate infant mortality rates in the Dunedin metropolitan area, 1900-1904 to 1915-1919 (proportional estimate).

<table>
<thead>
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<th>1910-1914</th>
<th>1915-1919</th>
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<tbody>
<tr>
<td>Illegitimate deaths (number)</td>
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<td>Illegitimate births (number)</td>
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<td>Illegitimate mortality rate (rate per thousand illegitimate births)</td>
<td>191.24</td>
<td>177.23</td>
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<td>104.06</td>
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<td>Legitimate deaths (number)</td>
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<td>468</td>
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<td>324</td>
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<td>Legitimate births (number)</td>
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<td>Legitimate mortality rate (rate per thousand legitimate births)</td>
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<td>Comparative risk (illegitimate rate/legitimate rate)</td>
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<td>2.67</td>
<td>2.42</td>
<td>2.10</td>
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Sources: 'Illegitimate births' derived under the proportional estimate from the Dunedin registration area figures in the *Statistics of New Zealand* series. 'Legitimate births' derived from the total number of births in the Dunedin metropolitan area given in the *Statistics of New Zealand* series, less the estimated number of illegitimate births. 'Illegitimate deaths' and 'Legitimate deaths' from the Dunedin mortality study.

### Table A2.2. Illegitimate and legitimate infant mortality rates for the Dunedin metropolitan area, 1900-1904 to 1915-1919 (maximal estimate).

<table>
<thead>
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<td>548</td>
<td>439</td>
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<td>165.43</td>
<td>124.09</td>
<td>93.39</td>
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<tr>
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<td>Legitimate births (number)</td>
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<td>7025</td>
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<td>6490</td>
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<td>Legitimate mortality rate (rate per thousand legitimate births)</td>
<td>73.19</td>
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<td>2.39</td>
<td>2.48</td>
<td>2.18</td>
<td>1.87</td>
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Sources: 'Illegitimate births' derived using the maximal estimate by taking the total figure for the Dunedin registration district from the *Statistics of New Zealand* series. 'Legitimate births' derived from the total number of births in the Dunedin metropolitan area given in the *Statistics of New Zealand* series, less the total number of illegitimate births for the Dunedin registration district. 'Illegitimate deaths' and 'Legitimate deaths' from the Dunedin mortality study.