

# Differential Trends in the Compression of Mortality: Assessing the Antecedents to Current Gaps in Health Expectancy in New Zealand

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## Abstract

Health Expectancies (HEs) for New Zealand show significant differentials between Maori and non-Maori, but also by gender and period. These differentials correlate with findings from both generation and synthetic life-tables relating to New Zealand's epidemiologic transition. At the beginning of that transition quartile 1 ( $Q(1)$ ), and Median ( $Med$ )  $d(x)$  values were close and centred at young ages; during the transition the gap became very wide; at the transition's end the gap again narrowed. Cohort and synthetic trends in  $d(x)$ ,  $l(x)$ ,  $M$ ,  $Qs$  and  $Meds$  are reviewed and linked to recent HEs. Data point to epidemic polarisation. Cohort analysis allows the evaluation of the role of past experiences on the recent HEs, and thus point to possible strategies for reducing gaps in both  $d(x)$ , and HEs.

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## Introduction: Past and Current Gaps in Health Status in New Zealand

In assessing population health many governments and researchers use state-of-the-art methods that are now *de rigueur* in the European Union and for the World Health Organisation (WHO) (Tobias *et al* 2008). These are variants of ‘Sullivan methods’, a form of life-table termed Health Expectancies (HEs) (see Johnstone *et al.*, 1998). They combine two dimensions of health: health status, as measured by functionality (achieving of tasks of daily living) and survivorship. The series of HEs for New Zealand (and most Western Developed Countries, WDCs)<sup>1</sup> are very recent, running only from the mid-1990s (Tobias *et al.*, 2009a). That said, they confirm that this population’s health trends fit with those seen in other WDCs. There are clear improvements in health status even over such a short period, a result confirmed in another study with an independent data set (Pool *et al.*, 2009).

New Zealand’s population is multi-cultural, with almost a third of the population having origins other than European. This overall picture obscures major ethnic gaps in HEs, and all other measures of morbidity and mortality. The reduction of gaps between different ethnic groups has been a long-standing issue for health planning in New Zealand.

To maintain consistency over time most of this analysis is on Maori and non-Maori<sup>2</sup>, even though we recognise that increasingly this dichotomy is confounded by three factors: the non-Maori population has become more culturally diverse; there is segmentation within the Maori population; and there have always been high levels of intermarriage between different ethnic groups.

The ethnic differentials shown in recent health status measures appear to correlate with findings on the compression of mortality derived from both generation and synthetic life-tables relating to New Zealand’s epidemiologic transition. Compression occurs when the range of ages at which people die is becoming narrower and narrower – happening at older ages in today’s society.

Compression has been a normal phenomenon throughout much of history, but at younger ages as against the older ages. A shift in the force of mortality from younger to older ages occurred over each population’s

epidemiologic transition. For example, early in the Maori transition (1890s), quartile 1 ( $Q1$ ) and median  $d(x)$  values were close and centred at young ages; during the Maori transition that gap, median minus  $Q1$ , became very wide; but in the transition's latest phases the gap is again narrowing. Today, compression shows up only at older ages, producing a uni-modal 'normal' distribution of deaths (Cheung, S. et al., 2005, p. 246)<sup>3</sup>, whereas at the start of the epidemiologic transition there was a bi-modal distribution, with the force of mortality occurring both at childhood and at older ages. The non-Maori trend for the period from 1876 is less marked. There was a wide gap between  $Q1$  and the median, but also showing marked bi-modality, and narrowing to show classical forms of compression today (Pool, 1994; see also Pool & Cheung, J., 2003).<sup>4</sup>

The health of an individual or of a cohort is a product of two historical trends – the experiences of the cohort itself, and the passage through an epidemiologic transition of the population to which that generation belongs. HEs measure health for cohorts that, in a country such as New Zealand, live in a period when the epidemiologic transition has run much of its course. In a pluralistic society not only does each ethnic group go through a different epidemiologic transition, but each has a different mix of social, economic and health experiences accumulated during their life-spans, experiences which may play a significant role in determining their health statuses at older ages.

## Aims of this Paper

This paper identifies and analyses the long-term differentials in health status in New Zealand, by focusing on the compression of mortality as measured from survival functions drawn from both cohort and synthetic life-tables, going back to the 19th century. It then compares these results with recent HE data showing that the same gap persists for health status. The analysis allows us to address three interlocking issues:

1. *There is an empirical question of concern primarily to New Zealand health policy-makers and service providers.* As in other WDCs, New Zealand's high risk populations are now mainly at older ages. This paper attempts to assess whether the historical shift to compression at these ages and the present health gaps are linked to ethnic differences in cohort patterns of survival, and thus to differential

risks, or are a function more of period effects, over time and at present, such as socio-economic disparity and differential access to health care.

2. *There is a more theoretical question of wider interest:* The role of cohort effects, especially as seen in measures derived from the  $l(x)$  function of the life-table, may be rather powerful, whereby changes at any age may have momentum effects which structure patterns and trends subsequently at much older ages. The non-Maori population benefited from rapid decreases in infant and childhood mortality in the late 19th century, in a period prior to when the New Zealand public health system or bio-medical factors could have made any significant impact on health status. These gains for the values  $l(0)$  to  $l(15)$  produced momentum effects that continued to have an impact on older cohort  $l(x)$  values, and thus on expectancies throughout much of the inter-war period (Pool & Cheung, J., 2005).

Our paper reviews cohort and synthetic trends in  $d(x)$ ,  $l(x)$ , modes, quartiles and medians, analysing all ages as well as adult ages, and links them to recent HEs and related data (e.g. a Sullivan's observed prevalence method of Hospitalisation Utilisation Expectancies (HUEs), (Cheung, J. et al., 2001)), which show compression of both mortality and morbidity at older ages as measured by bed-use combined with life expectancy. We will also explore dynamics of the oldest-old (Robine & Cheung, 2008: Discussion). Cohort analyses of  $l(x)$  and  $d(x)$  allow us also to evaluate the role of past experiences of older cohorts on the recent HEs and thus on current polarisation, and thus to point to possible strategies for reducing gaps in both  $d(x)$ , and in HEs.

## **Health Expectancy, Compression of Mortality and Related Trends**

That longevity is increasing is incontrovertible; what is disputed is how far out longevity might be extended, and what are the implications for human populations and health systems (e.g. Oeppen & Vaupel, 2002; Tuljapurkar et al., 2000). Up until the early 1990s various protagonists put forward different scenarios about mortality itself, notably whether or not the survival curve was "rectangularising" (summarised in Levy, 1998). In a

recent paper (Cheung, S. et al., 2005), this simple geometry of survivorship curves has been shown to be rather more complex.

Along with this were debates about what would happen to morbidity: would older people live longer but suffer disability or illness for many of the later years, or would compression also be seen for sickness (classical papers include Fries, 1980; Manton, 1982; Olshansky et al., 1993). The construction of measures that looked at health in terms of functionalities proceeded at pace, and HEs have become increasingly accepted as conventional tools for health status research and policy analysis (Tobias et al., 2008).

The emerging evidence suggests that increasing longevity has been associated with two trends: a narrowing band of ages at which the majority of people die (compression), and paralleling this, a narrowing range of causes from which most people die. This shift in mortality has been brought about by changes in morbidity, also entailing compression by age and cause. These patterns have been reported widely overseas, and also for New Zealand (Pool, 1994; Cheung, J., 1999; and 2001, a paper cited internationally in Cheung, S. et al., 2005, p. 243).

Robine & Cheung (2008) argue that these trends support the Fries hypothesis. Nevertheless, they strongly qualify this by citing the rapid growth in the number of persons at oldest-old ages, especially centenarians, and emerging evidence of derectangularisation (a shift of the survival curve to the right) as indicative of extension of longevity rather than compression.

Paralleling these trends are the relationships between mortality and morbidity. This is reflected in the growing body of data on HEs and related measures, and decreases in their reciprocal - "life expectancy with severe disability" (Cai & Lubitz, 2007), both overseas and for New Zealand (Ministry of Health, 1999a: Chapter 7; Tobias et al., 2008, 2009a; Pool et al., in press).

## Measuring Compression

Siu Cheung et al. (2005) review theories about 'normal' longevity, as proposed by Wilhelm Lexis (1837-1914). Using data on Hong Kong, they then make empirical observations on longevity, compression of mortality and related topics. They elaborate on Lexis' (1878) and Kannisto's (2001) work to build a framework, the parameters of which are determined by the function "four standard deviations  $[+/-]$  from  $[\text{the Modal age at death, M,$

in this case adult deaths only]” (p. 246). The authors delineate and statistically define three dimensions identified by earlier theorists:

- The degree of horizontalisation, which is an incremental plateauing of  $l(x)$  values over longer and longer periods of the life-span as “infant and premature deaths are reduced”. Clearly, this is a process that is related to, and drives the onset of verticalisation.
- The degree of verticalisation, “the steepness of the survival curve in the region of M. This steepness depends on the concentration of the ages at death around M [i.e. this is a measure of compression]”.
- Longevity extension, which “corresponds to changes in the right-hand tail of the survival curve and describes how far the highest normal life durations can exceed the modal age at death” (Cheung, S. et al., 2005, p. 248).

These parameters are determined, as noted already, by computations of standard deviations around the mode. These calculations are far from easy to perform; indeed, Cheung, S. et al. remark, somewhat obliquely, “one must be able to carry out the indicated operations” (2005: 254). Our data are not sufficiently refined to be able to do this. For earlier cohorts the source data for both our Maori and non-Maori, period and cohort life-tables are abridged, and end at 80-100 years, depending on the date. Although the recent official period ones are full-tables, they also close off at 100, while a cohort analysis carried out by Statistics New Zealand (2006) for the total population (Maori and non-Maori) gives single-year values, again with closure at  $l(100)$ .<sup>5</sup>

But we have another concern. For older populations, even in a country like New Zealand, one must question whether age reporting is of sufficient accuracy to carry such refined analyses, especially for the 19th and early 20th century. For example, Kannisto (1994) gave a less than flattering assessment about age-reporting in his review of the data available to study the ‘oldest-old’ in WDCs. The situation is known to be far more severe for Maori. Complete birth registration for Maori was not finally achieved until 1947/48 (Pool, 1977, p. 64), and neonatal death registration finally became complete after World War II (Sceats & Pool, 1985, pp 244-46).<sup>6</sup> In this context it must be remembered that life-table computations require accurate reporting both for the denominator (self-reporting by a census respondent)

and the numerator (a third party who may not have exact details of the deceased's date of birth). We would also worry whether some single-year age-distributions are the products of actuarial smoothing to eradicate age-heaping and other data concerns. A tendency to exaggerate self-reported ages is also common among the very elderly (Shryock & Siegel, 1976, p. 128, who see elderly as 80+ years).<sup>7</sup>

Thus we have not used the more exact and powerful statistical techniques prescribed by Cheung, S. et al., but, following some other authors, we have used modes, based on quinquennial age-groups, and arbitrarily selected medians and other percentile-based statistics applied to  $l(x)$  and  $d(x)$  life-table functions (see Cheung, S. et al., 2005, Table 1). Our rationale for this is that, as the properties of percentiles and modes computed from grouped data are well known and simple, they adequately serve an exploratory comparative study of the sort we are working on. One can also appeal to the old statistical principle that to reduce perturbations, such as those due to less than perfect age-reporting, one should cumulate. For example, as the authors argue, "Intuitively, the degree of horizontalisation can be measured by the age reached by some high percentile of survivors in a life table (i.e. the age reached by 90%, 95% or 99% of the survivors)...". They then qualify this by adding "...but this approach is limited to a situation in which infant mortality is low and is undermined by the arbitrary nature of the percentiles". We must thus accept that we are in breach of a general principle enunciated by Kannisto, whose experience with these data is probably still unsurpassed: that "indicators should be free from any fixed age or percentile determinations..." (pp. 245-46). Our findings are thus indicative rather than definitive.

This is nowhere more problematic than at the oldest ages and for longevity extension, an area according to Cheung, S. et al. that has received limited attention. Moreover, as they stress, the measures often used, such as the age reached by some small defined minority (e.g. one per 10,000) are affected by population size, a problem faced by all researchers of all demographic phenomena relating to older New Zealanders – even today our population total is only just over four million. This becomes a more urgent issue because "drectanglearization of the survival curve is emerging". Moreover, they point to "a significant growth in the number of centenarians in Europe and Japan, findings that are more in favor of an acceleration in the increase in longevity than a slowing down" (p. 244).

We will not use data on centenarians, as our preliminary investigations suggest that a growth in their numbers as observed today, may be, at least in part, a function of the size of the cohort at birth and inter-cohort decreases in childhood and premature mortality occurring many years ago. But even if we dampen this effect by applying life-table values to birth cohort sizes, there are still problems in making inter-cohort comparisons.<sup>8</sup>

This analysis focuses on the importance of dynamics at earlier phases of the life-span, and for this we can compare New Zealand's two major populations, as defined above. As we will show below, there are also some emerging indications of drectangularisation.

Finally, this is an exploratory study only. For that reason we do not look at gender differentials and will use males only, except in the first substantive section where we compare Maori and non-Maori females. There are significant gender differences that have been discussed fairly fully elsewhere (see Pool, 1982, 1994; summarised also in Pool & Cheung, 2003).

## **New Zealand's Populations and Health Trends**

New Zealand has a higher proportion of its population from outside of Europe than any other WDC.<sup>9</sup> About 15 percent of New Zealanders belong to the indigenous ethnic group, Maori, with 8 percent in Pacific ethnicities, 9 percent in Asian and around one percent African and other non-European ethnicities. It should be noted that a growing proportion of the population identify with more than one ethnicity, for example at the 2006 Census roughly half of Maori also identified with at least one other ethnic group.

The Asian population is composed of two very different groupings, each diverse, from East Asia and South Asia, and is a roughly similar proportion of the total population compared to the Australian, Canadian and United States populations. But it is the higher proportion belonging to the indigenous ethnic group which sets New Zealand apart from other WDCs, plus the inflows of large numbers of Pasifika, typically from eastern tropical Oceania.

New Zealand became a colony in 1840. By 1859, the settler population (mainly of British Isles-European origin) outnumbered Maori. This was part of a longer-term decline in numbers of Maori from about 80,000 to 100,000 in 1769 to a nadir of 40,000 around 1890. This decrease was driven mainly by the introduction of diseases to which Maori had no immunity. It was a

catastrophic loss, but was arguably less severe than the fate suffered by Hawaiians, Tahitians and other Pasifika. In part, and this is important for the analysis that follows, this was because New Zealand was not hit by the great apocalyptic diseases such as smallpox, but instead succumbed to the prevalent diseases of Europe, typically the childhood and other communicable disorders to which they had had no previous exposure such as measles, tuberculosis and influenza.<sup>10</sup> Malaria and most other 'tropical diseases' were neither endemic nor have they been epidemic in New Zealand. Representing only about six percent of the total in 1901 the proportion of the population who identify as Maori has grown to 15 percent by 2006. This came about despite a rapid decline in fertility in the 1970s, and large migration flows to Australia in particular, a characteristic shared with non-Maori New Zealanders.

By the time the first life-tables were constructed in the 1870s, non-Maori (mainly Pakeha for much of the period covered in this paper) had achieved significantly higher levels of life-expectation at birth than levels recorded from the British Isles' populations from where they were drawn - even the English. Life expectancy for non-Maori women reached 55 years in 1876, and 60 years in 1901 - seemingly the first national population to attain these levels. Indeed they appear to have had higher levels than seen in Dr. William Farr's 'Healthy Districts'.<sup>11</sup> This summary statistic obscures an important qualification seen by comparing Pakeha with Norway and Sweden: relatively speaking,  $e(0)$  and  $(15)p(0)$  values were high, but  $e(x)$ s at older ages fell below those for Norway. The reason may have been due to migration - at the end of the 19th century, many and even the majority of Pakeha were British-born, but children were almost all New Zealand-born. The migrants had carried with them their past cohort health experiences, whereas the non-Maori children benefited from conditions in the colony that we will describe below.

Throughout the colonial and post-colonial history of New Zealand, there has never been formal segregation. Even in the 19th century social policy measures applied, in principle, to Maori (e.g. free, compulsory, secular education introduced in 1877). More importantly, critical steps were taken in the early 20th century to reduce health gaps between Maori and non-Maori. The Public Health Act of 1900 set up a Department of Public Health (1901), and a Division of Maori Hygiene, in which the head and most practitioners were Maori medical graduates. These physicians played a

significant role in achieving an improvement in life-expectation at birth from about 25 years in the 1890s to 35 years by 1911. This ensured 'the survival of the Maori race', something that had not been certain two decades earlier.

The introduction of a Nordic-style welfare State in 1938 went a step further by making a particular effort to ensure that Maori and non-Maori gained equal access to policy measures and health services. In the 1930s and 1940s concerns were being expressed about Maori health, notably tuberculosis death rates. Between 1940 and the 1960s, a wide range of reforms were introduced to all populations, and were to have a marked beneficial effect, particularly on Maori health. Maori were targeted in screening programmes and interventions, particularly for communicable disorders: in 1945 well over 50 percent of Maori died from this category of disease; by 1976 the level had dropped to 16 percent (Pool, 1991, Table 6.7). Campaigns such as that against tuberculosis were facilitated by the fact that Maori were geographically clustered in the rural northern and eastern North Island of New Zealand. The new chemotherapeutics, available from the 1940s, and improved biomedical and public health services could be accessed by everybody. But the very rapid urbanisation of Maori at this time, a process assisted by Government, accelerated this and also meant that the Maori workforce went quickly through an industrial labour force transformation that had flow on effects for housing, income and general wellbeing. The nesting of health policy into social policy, especially in the campaigns against tuberculosis, was an emblematic feature of these very successful reforms, and this was reflected in rapid advances in Maori survival. As these changes and their health impacts have been reported elsewhere there is no need to go into detail here (Pool, 1991; Pool & Cheung, 2003).

In the 1980s and 1990s there was radical economic restructuring, and associated cohort deterioration. This generated a great deal of concern, expressed in a number of reports and papers published in New Zealand and overseas, about marked social and ethnic differences in health status (esp. Ajwani et al., 2003; Blakely et al., 2005, 2008; Tobias et al., 2009a), and the links between social wellbeing and health - in particular about cardiovascular mortality and its proximate determinants such as diabetes (Smith et al., under editorial review). This was followed more recently by

attempts to integrate primary and secondary health care. This will be picked up later when we review mortality in relation to morbidity trends.

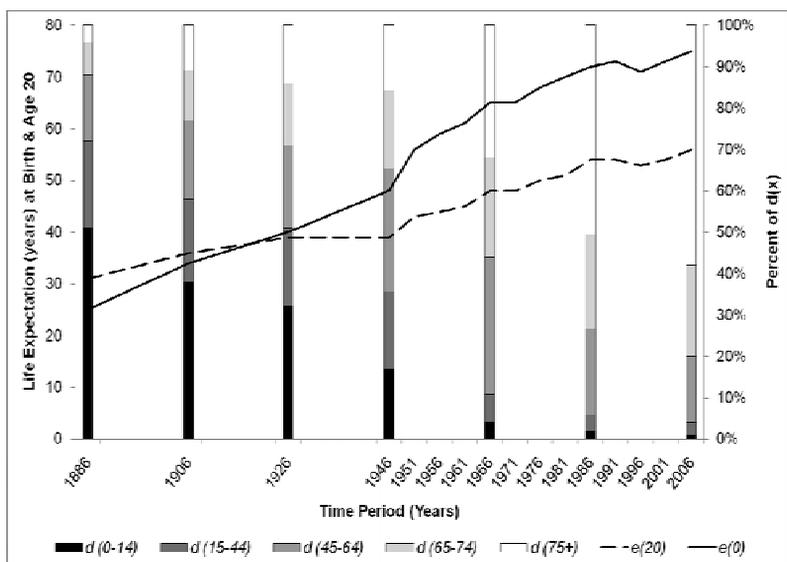
In sum, a continuing policy objective, for more than a century, has been to effect a convergence between Maori and Pakeha levels of health, while continuing to maintain and improve the high expectancies experienced by Pakeha from the time of early colonisation.

Figures 1 and 2 provide a big picture overview for the Maori and non-Maori epidemiologic transitions to be discussed in subsequent sections of the paper. They graph the values for female  $e(0)$  plus the proportion of the period  $d(x)$  falling into broad age-groups. Essentially they show how the force of mortality has moved up from childhood to centre at the oldest ages, in this case 75+ years. The Maori transition is much more marked than the non-Maori, with the shift-share by age becoming entrenched from about 1940 onward.

In high mortality populations  $e(0)$  is normally lower than  $e(x)$  values at age 1, and even up to adult ages. Thus the crossover for Maori, when  $e(0)$  started to exceed  $e(20)$ , is particularly interesting and has useful analytical properties in disaggregating premature mortality from later mortality.<sup>12</sup> In the 1880s, a Maori woman reaching her early 30s still had a longer life-expectation ahead of her than she had at birth. As recently as 1976, the non-Maori  $e(1) > e(0)$ .

The remainder of this paper analyses in more detail at horizontalisation and verticalisation, implicit in these graphs, comparing and contrasting Maori and non-Maori. It also looks for evidence of drectangularisation. Finally it links these results to HEs.

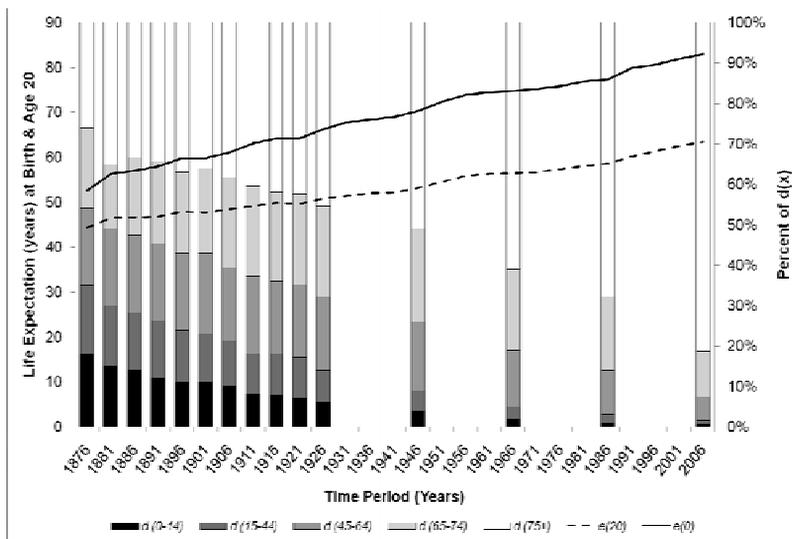
**Figure 1: Maori female life expectancy (years) at birth and at age 20 years, and percent of all deaths occurring at ages 0-14, 15-44, 45-64, 65-74, and 75+ (Period life-tables)**



Sources: Computed directly by Ian Pool and Jit Cheung from vital data and censuses, 1945 on are from official tables. Prior to that adjusted official data (1926-1941), or indirect estimates described in Pool (1991: pass).

Notes: Maori data are highly unreliable until about 1936, becoming satisfactory only from 1945 on. These are based on life table deaths,  $d(x)$ : the number of any cohort dying at a given age group,  $x$ . The percent of  $d(x)$  at ages below 45 years are so few (<5%) in recent decades that they are difficult to discern on this graph.

**Figure 2: Non-Maori female life expectancy (years) at birth and at age 20 years, and percent of all deaths occurring at ages 0-14, 15-44, 45-64, 65-74, and 75+ (Period life-tables)**



Sources: Computed directly by Ian Pool and Jit Cheung from vital and census data, or drawn from official series.

Notes: These are based on life table deaths,  $d(x)$ : the number of any cohort dying at a given age group,  $x$ . The percent of  $d(x)$  at ages below 45 years are so few (<5%) in recent decades that they are difficult to discern on this graph. The data for  $d(1876)$  are the average of tables computed for census dates 1874 and 1878, Thereafter, with the exception of 1931, 1941 (when no census was taken) and 1946 (when the census had been taken six months earlier in 1945), all censuses are carried out in March of years ending in digits 1 and 6.

## Horizontalisation

Tables 1 and 2 (below) show that horizontalisation may be achieved very rapidly and then the trend plateaus out.<sup>13</sup> For non-Maori, this had occurred for the cohorts born around 1930; for Maori it was delayed until those born after the 1950s. Nevertheless, there has been some convergence: for the cohort of 1921-26 the gap was 30.2 years, and this had extended to 47.9 years for the cohorts of 1941-46, but had dropped back to 17.8 in 1996 (adjusted data), only to increase again in 2006. This seems to have been because the non-Maori value increased a little more rapidly than did the Maori. This was despite the Maori value actually decreasing between the synthetic table of 1986 and that of 1996.

**Table 1: Horizontalisation: Age (years) by which  $l(x)$  has declined to the 90th percentile, Maori and non-Maori males, cohort life tables**

Cohorts born	Maori	Non-Maori
1871-76	--	0.9
1881-86	--	1.0
1991-96	0.3	2.6
1901-06	0.4	4.5
1911-16	0.4	17.8
1921-26	0.5	30.7
1931-36	0.8	42.6
1941-46	1.0	48.9
1951-56	4.5	<u>54.1</u>
1961-66	32.7	--

Notes: .. = No Data.

— = Based fully/partially on projections. Projections for non-Maori are more reliable than those for Maori, so are not reported for Maori.

The non-Maori figure for 1951/56 is based in part on projections for the period 2001-06. Synthetic data (see next table) exaggerate the level of percentiles at older ages, as the younger cohorts included in such tables have markedly better survival rates than true cohorts had had when they were younger.

The Statistics New Zealand (2006) full cohort tables for the average of the birth cohorts of 1901 and 1906 yield a lower value (1.4) than that shown here, but they are for the total population and thus include Maori (0.4). At  $l(5)$  the full total population tables are close to the non-Maori used here, 87,793 vs 89,727 (non-Maori) and 65,945. Reweighting the Maori and non-Maori  $l(5)$  proportional to population yields 88,300, a difference of only 0.6%.

**Table 2: Horizontalisation: Age (years) by which  $l(x)$  has declined to the 90th percentile, Maori and non-Maori males, period life tables**

Cohorts born	Maori	Non-Maori
1976	41.0	50.0
1986	46.7	52.6
1996	46.2 (32.7)	58.1 (50.5)
2006	49.5 (35.0)	62.2 (54.1)

Note: Figures in brackets are adjusted results. For each population, the cohort figure is used to adjust the synthetic data to the cohort, where a reference year allows such an approximation (Maori = Cohort 1961-66/Synthetic 1996; non-Maori = cohort 1951-56/ synthetic 2006). The adjustment was carried only as far as an adjacent date, as the underlying assumptions about distributions of mortality would become difficult to sustain before and after that.

The cohort data already presented, but using data for quinquennia rather than decades, allow some interesting but speculative comments to be made about the timing and slopes of the two curves. This is shown in Table 3.

What is surprising in this table are the similar values reached by Maori and non-Maori in these periods of most rapid improvement. However, the Maori improvements in survival occurred more rapidly. The cohort of 1951-56 had already fallen off to its 90th percentile by age 4.5 years, but for those born in the 1960s this was 32.7 years after birth. These changes were implemented in a 40 year span running from circa 1958 (4.5 years after the average year of birth of this cohort) to circa 1998. For non-Maori the shift was slower, about 45 years from circa 1908 to circa 1953. This difference in velocity is to be expected as the non-Maori changes had come about primarily through social and economic change, whereas the Maori shifts came through a mix of improvements in socio-economic wellbeing, and social and health policy interventions, including access to modern chemotherapeutics and other bio-medical technology.

**Table 3: Age (years) by which  $l(x)$  has declined to the 90th percentile, Maori and non-Maori male, cohort life tables, periods of steepest change**

Maori, cohorts of 1951-56 to 1961-66				
4.5 (1951-56)	17.0 (1956-61)	32.7 (1961-66)		
Non-Maori, cohorts of 1901-06 to 1921-26				
4.5 (1901-06)	9.3 (1906-11)	17.8 (1911-16)	22.7 (1916-21)	30.7 (1921-26)

Nevertheless, the change within non-Maori had occurred at a relatively rapid speed, for an era in which shifts in health were due more to social and economic factors than bio-medical. Certainly, non-Maori benefited more than Maori from the public health reforms of the early 20th century, which were less dependent on chemotherapeutic and other bio-medical technologies than those that came after World War II and more on regulatory measures relating to hygiene and sanitation for water supplies and sewage, food processing and hospitals. But these shifts were also driven by a more latent factor. Between 1876 and 1901 male non-Maori  $(15)d(0)$  had decreased very significantly, from 19,636 to 12,655. Public health and medical technology accounted for little if anything in this shift. Instead it

occurred because of social and economic changes that ran the whole gamut from the effects of rapid fertility decline<sup>14</sup>, to improvements in material wellbeing. This change produced a momentum effect in the  $l(x)$  column of life-tables that moved up the age-ranges affecting increasingly older age-groups through the inter-war period (Pool & Cheung, J., 2005).

But these advances were not always sustained. A detailed analysis of the cohort tables for the 1990s, for example, showed evidence of cohort deterioration – slight for non-Maori, more marked for Maori. A first hypothesis was that this could have been an artefact of definitional shifts in the ethnicity question and in coding in that period, but a review of the data showed that these changes probably instead statistically dampened the effects of any deterioration. An alternative hypothesis relates to the radical economic neo-liberal restructuring of the period, which *inter alia* pushed disproportionately more Maori than non-Maori into casual employment and other forms of marginalisation.<sup>15</sup> It was concluded that the negative effects of marketisation, the stripping of the welfare state and the elimination of many of the jobs in which Maori were clustered (e.g. manufacturing). In turn, it was argued, “the deterioration in [the 1990s], especially for Maori male cohorts, was a residual effect of cycles of cohort gain and deterioration reinforced by period effects coming from restructuring”. For female Maori cohorts, who had had very high levels of fertility followed by a rapid decrease, but retaining early child-bearing (TFR. 6.1, 1961; 5.0, 1973; 2.8 1978) and who were over-represented in occupations affected by the shift to casualisation, the case was made that “the negative effects of restructuring on Maori reinforced the residual cohort effects coming from a history of high fertility” (Pool & Cheung, 2003, pp. 122-23).

The most radical shifts in the process of horizontalisation, and the factor that sets the trajectory, come at infancy and early childhood. It is possible in this regard to use a mix of cohort and synthetic data without the results being affected in any significant way. This is done in Table 4, which presents values for the entire period. Three key results are shown by these data. First,  $(5)d(0)$  is high at the start of the transition; very high – almost half the total  $d(x)$  – in the case of Maori. Secondly, the levels drop very rapidly triggering horizontalisation. Thirdly, by the end of the transition ethnic differentials have all but disappeared.

**Table 4:** *(5)d(0)* (thousands), cohort and synthetic, Maori and non-Maori males, selected years

Cohorts born	Maori	Non-Maori
1876	--	16.6
1896	46.8	11.4
1916	30.9	9.0
1936	18.1	4.8
1956	10.3	2.8
1976	<i>2.4</i>	<i>2.0</i>
1996	<i>1.3</i>	<i>0.7</i>
2006	<i>1.0</i>	<i>0.6</i>

Note: Figures in italics are taken from period tables

Tables 5, 6 and 7 below turn to indicators of compression itself. Although the focus in these tables is on aspects of verticalisation, they also throw further light on dimensions of horizontalisation ( $Q_1$ ,  $LTM$ ,  $d(x)$  ( $LTM$ )). Above all, at an early stage in the epidemiologic transition there is a markedly bi-modal distribution, with the larger mode at 0–4 years of age. Later the mode shifts to the older adult ages, at which the secondary mode had been seen earlier in the transition. This is accompanied by increments in  $Q_1$  and for Maori, increases in the median, whereas for non-Maori the median remains relatively stable, only increasing at a later stage in the transition, and after an initial growth the  $Q_1$  for non-Maori horizontalises more and more. This produces an increasing narrowing gap (years of age) between  $Q_1$  and the median, a squeeze playing a major role in the process of horizontalisation.

## Verticalisation

The data in Tables 5 and 6 allow us to identify the major factors in verticalisation and thus compression, *per se*. To reiterate, these are crude rather than refined indices, but they still provide interesting insights about the dynamics and structures of compression. In this regard, the mode may be a more realistic measure of compression, or at least of concentration, than we might tend to think – we are habituated to the seeming exactitude of mean based statistics, and thus intuitively reject such an imprecise statistic. But perhaps such a rejection is unwarranted.

In fact, in making a detailed review of the differences in  $d(x)$ s between those at the quinquennial adult modal age (see below) and those in the adjunct age-groups, it became clear that these were very small, but often with quite sharp verticalisation up to and after the modal spread itself. The

impression that comes from that review is that the mode, rather than being a spike, as it were, is fitted by a broad bell-shaped curve. This is merely a methodological observation at this point, yet the question must be raised whether or not it has wider theoretical, substantive and even policy implications. This becomes a more pressing issue when synthetic life-table data are employed (see Table 7).

Turning to the results in Tables 5 and 6, a number of points stand out.

- Medians for non-Maori are relatively stable across cohorts, while those for Maori increase. This may mean be an indicator of the steps towards horizontalisation (Maori), and an indication (non-Maori) of the achievement of verticalisation.
- Q1 increases rapidly for Maori, but after such a surge then plateaus increasingly at what historically would have been regarded as a geriatric age. The ranges for the Q1 are huge: Q1 for the most recent non-Maori cohorts is 100+ times the age of that for the earliest Maori cohort.
- In contrast, Q3 is high, stable and not very different for both populations.
- As a result, the gap between the median and Q1 decreases, a clear result of horizontalisation, and then shows the effects of verticalisation.
- The gap between Q3 and the median remains stable for non-Maori, but decreases for Maori. By the cohort of 1921-26 the ethnic differences are relatively limited. For non-Maori the IQR also declines rapidly at first and then more slowly, but converging towards the Q3 – Median range. This ethnic difference is clearly a function of the timing differences for the two transitions.
- In Table 7 synthetic life-table data are presented on Qs and Medians, with all the caveats that we noted earlier. But they do suggest that there has been a convergence with Maori ages for Qs and the median converging on the non-Maori, which still remain higher Q-Median and IQ ranges for Maori are now smaller than for non-Maori, but this will be in part a function of censoring biases, so is a far from definitive finding.

These two tables also present a number of findings on modes. In interpreting these it is necessary to recognise the methodological point we

made earlier – that the modes are uneven mesa-like shapes rather than spikes. The table provides separate data on both life-time (0+ years), and, following Cheung, S. et al. (2005), adult modes (15+ years).

- At earlier dates, for both populations the life-time mode is centred at ages 0-4 years; but for non-Maori in more recent years the life-time mode is the adult mode.
- For both populations, the age-groups represented in, and the size of the  $d(x)$ , for adult modes, and particularly for the spread around the modes, remain remarkably stable, especially for non-Maori, but with a suggestion of compression for non-Maori for younger cohorts. Larger  $d(x)$  values are also starting to show up for both populations in the age-groups immediately above the spread around the mode (Table 8), 8-84 years for Maori and 90-94 for non-Maori. This might be interpreted as an early sign of derectangularisation.
- There is, however, a persisting gap between Maori and non-Maori for adult modes, and the two populations seem to move almost in tandem (as indicated in Table 8). This raises the question whether these are purely an artefact of the different stages reached by each population, or whether the patterns of a ‘normal’ lifespan vary between groups.

**Table 5: Maori male cohort life-table verticalisation**

	Cohorts born				
	1891-96	1901-06	1911-16	1921-26	1931-36
<b>Years</b>					
Median	13.8	40.2	47.9	49.4	62.6
Quartile 1	0.7	0.9	1.0	4.6	22.6
Quartile 3	60.3	67.1	68.7	72.8	..
Gap Median – Q 1	13.1	39.3	46.9	44.8	40.0
Gap Q 3 – Median	46.5	26.9	20.8	23.4	..
Inter-Quartile Range	59.6	66.2	67.6	68.2	..
Lifetime Modal Quinquennial					
Age at Death ( <i>LTM</i> )	0-4	0-4	0.4	0.4	..
$d(x)$ ( <i>LTM</i> )(000)	46.8	34.1	30.9	25.5	..
Modal Adult Age at Death ( <i>AM</i> )	65-69	60-64	65-69	65-69	
$d(x)$ ( <i>AM</i> )(000)	5.8	7.3	7.7	8.2	
$d(SAMx)/d(15+)$	33%	34%	34%	33%	

Notes: Figures relate to Years, except  $d(x)$  values, which are in 000s or %s.

$$d(SAMx) = d(AMx) + (dAMx+5) + (dMx-5), \text{ where } x = 5 \text{ year age-group.}$$

**Table 6: Non-Maori male cohort life-table verticalisation**

Years	Cohorts born						
	1871-76	1881-86	1891-96	1901-06	1911-16	1921-26	1931-36
Median	67.5	68.0	68.5	70.3	71.6	74.8	76.5
Quartile 1	32.3	44.1	54.9	55.7	57.9	60.7	65.1
Quartile 3	77.9	78.6	78.9	79.7	80.7	84.7	<u>87.3</u>
Gap Median – Q 1	35.2	23.9	13.6	14.6	13.7	14.1	11.4
Gap Q 3 – Median	10.4	10.6	10.4	9.4	9.1	9.9	10.8
Inter-Quartile Range (IQR)	45.6	34.5	24.0	24.0	22.8	<u>24.0</u>	<u>22.2</u>
Lifetime Modal Quinquennial Age at Death ( <i>LTM</i> )	0-4	0-4	75-79	70-74	70-74	80-84	85-89
$d(x)$ ( <i>LTM</i> )(000)	16.6	13.2	13.7	13.4	12.6	13.4	14.8
Modal Adult Age at Death ( <i>AM</i> )	75-79	75-79	75-79	70-74	70-74	80-84	85-89
$d(x)$ ( <i>AM</i> )(000)	11.7	12.8	13.7	13.4	12.6	13.4	14.8
$d(SAMx)/d(15+)$	41%	42%	43%	43%	40%	40%	44%

Notes: Figures relate to years, except  $d(x)$  values, which are in 000s or %s.

$d(SAMx) = d(AMx) + (dAMx+5) + (dMx-5)$ , where  $x = 5$  year age-group.

— = Based fully/partially on projections. Projections for non-Maori are more reliable than those for Maori, so are not reported for Maori.

Tables 7 and 8 draw on recent period data. It is not clear whether differences with cohort changes are real or merely a methodological artefact of the effects of using synthetic results rather than cohort ones. They suggest, however, that a gap in survival still exists. The Maori adult modal spread is compressing to become a higher and higher percent of the adult  $d(x)$ , but the changes are almost in parallel with non-Maori so the gap is not closing. Two-fifths of the Maori adult  $d(x)$  and more than half of all non-Maori  $d(15+)$  occur over only about 17 percent of the adult life-span (15-105 years).

Finally, the data on Independent Life Expectancy (ILE, free from disability requiring assistance) from recent HE tables point to a similar direction. Today, both Maori and non-Maori are spending an increasing part of their old age – at the ages around which compression of  $d(x)$  is also occurring – also free from disability-based dependence. Moreover, Maori levels seem to be converging on non-Maori. But ILEs are also expanding; durations free from dependence are longer.

**Table 7: Recent (2005–07) Official Period Life-Table data Q1, median and Q3 values, Maori and non-Maori males**

	Maori	Non-Maori	Gap: Maori – Non-Maori
Quartile 1	62.7	73.6	10.9
Median	73.5	82.2	8.7
Quartile 3	82.0	88.4	6.4
Median-Quartile 1	10.8	8.6	-2.2
Quartile 3-Median	8.5	6.2	-2.3
Inter-Quartile Range (IQR)	19.3	14.8	-4.5

**Table 8: Recent Maori and non-Maori period data from life tables and health expectancies**

	Maori			Non-Maori		
	1996	2001	2006	1996	2001	2006
$l(75)$	34,037	40,082	45,898	61,068	66,633	71,793
$D(SAMx)/d(15+)%$	41	43	43	50	52	53
$AM$	65-79	65-79	65-79	75-89	75-89	75-89
ILE(65)*	6.8	8.0	10.3	9.9	9.7	12.0

\* Independent Life Expectancy (ILE, free from disability requiring assistance, Yrs)

Notes:  $d(SAMx) = d(AMx) + (dAMx+5) + (dMx-5)$ , where  $x = 5$  year age-group.

ILE data are from unpublished series, Ministry of Health. Because of risk of sampling and other statistical errors, Maori data for ILEs are not accepted as highly reliable.

There is another possible aspect to this. Without resorting to Social Darwinism, we must remember that many older persons living without disability, whatever level, represent the hardy survivors of past epidemics and other life experiences that affect health and disability. This will be truer for Maori than for non-Maori as Maori cohorts have gone through the processes of horizontalisation and verticalisation after non-Maori. A clue to this is a measure computed for the 2006 HLEs – a so-called “survival curve”, combining the life-table  $L(x)$  with ILEs, and defined as person years lived without Level 2 disability. The Maori figure for age-group 70–79 years is 28,303 and the non-Maori is 41,302. This shows that cohort effects play an important role in fashioning disability and survival at older ages.

This raises two issues. First, as cohorts more widely representative of health experiences in the past, perhaps including in their number persons who survive because of advances in bio-medical technology, rather than dying, more and more could face ILE-level dependency or worse. For example, the interaction between having diabetes and survival on renal dialysis, and surviving but without independence, might be an example.

Secondly, health planners and policy makers in New Zealand, and elsewhere, must be aware of the fact that the experiences of cohorts of different sub-populations will vary. This means that there must be targeted surveillance and intervention, not just by socio-economic group but by cohort (see Discussion).

## Discussion

This paper has identified the very different trajectories for the epidemiologic transitions of two New Zealand sub-populations. The results show that epidemiological polarisation has been evident, markedly in the 19th and early part of the 20th centuries, but over the long term there is a degree of convergence. The data presented above show that convergence is not necessarily assured, but may require strategic interventions, and, as a consequence of this, the rates of closing-in can vary over time. The interventions must not only counteract period differences in wellbeing, access to health services and health status, but also must respond to cohort factors - for persons at middle and older ages, these are the differing cohort experiences, social and health, to which they have been exposed earlier in their life-spans. To add to this, the way the health system is fiscally organised - either through private insurance, or mainly publicly-funded - can make a difference, as a recent United States-Canada comparison shows (Huguet et al., 2008). New Zealand's system is similar to the Canadian, so, in principle, hospital care and much of primary and pharmaceutical care is free or subsidised. Both the public health campaigns of the early 20th century, and the post-war programmes, which were responsible for a major step forward in closing gaps between Maori and non-Maori, were publicly funded.

Early non-Maori health status benefited from high per capita incomes (arguably the highest anywhere in the late 19th century), and associated factors such as diet. A bonus, as it were, came from a rapid decline in fertility. From the beginning of the 20th century, both public health and biomedical factors gradually became more and more significant in maintaining high levels of survival, although the cohort momenta affected the  $l(x)$  function of the life-table - a latent effect produced by the radical declines in infant and childhood mortality in the late 19th century. In the post-war period, the non-Maori  $e(0)$  slipped gradually in rank among WDCs, but the range of expectancies had become narrow among members of this privileged

club – a sort of a ‘rich list’ of health status, where ranks change but the overall differences are minor on a global scale.

The differences between non-Maori and other populations on the ‘rich list’, whether in 1890, 1906, 1990 or 2006, or any other year, were far less than the epidemiological polarisation apparent within New Zealand itself. We deal with only one such dimension here – between Maori and non-Maori.<sup>16</sup> Over the long term there was incomplete convergence, but this has tended to go in starts and stops, with the particular phase dependent to a large degree on the policy environment and thus the service programmes being delivered at any one time.

Historically, there were long periods in which, more by neglect than design, decreases in the gap between Maori and non-Maori were gradual. This was not because separate and unequal services were available, but because Maori were mainly living in isolated areas, away from health facilities, and were dependent on a semi-subsistence income or casual work. The descriptions of life on the East Coast of the North Island – a region with high concentrations of Maori – in a very competent social-epidemiological study of tuberculosis in the 1930s show this (Turbott, 1935). At this juncture the non-Maori  $e(0)$  at 65 years for males and 68 years for females, was among the highest anywhere, and contrasted markedly with Maori at 46.3 years for males and 46.0 for females (Pool 1985: Tables 116 and 124).<sup>17</sup> The key to reducing gaps, therefore, was being more assertive in getting services out to Maori.

Between these phases when little happened to accelerate convergence, there were interventionist phases which had major positive benefits in closing gaps. Earlier in this paper two were highlighted: the WHO ‘Alma Ata-like’ programmes of public and community health driven by Maori physicians working in the Department of Health in the first decade of the 20th century; and the wide-ranging, comprehensive and effective programmes introduced in the 1940s after New Zealand had established a comprehensive welfare state (1938).

From the late 1960s these concerns seemed to be less marked, except for a focus on infant mortality, resulting in the attainment of almost no differential in neo-natal rates by about 1981 and decreases in the post-neonatal gap. By 2006, rates had decreased for both groups, but more rapidly among non-Maori, so a gap still existed. As a result, the absolute difference was greater at the neonatal age than it had been in 1981, but not

at the post-neonatal. At both ages, though, relative differences had actually extended. Nevertheless, both were low by world standards, so that most of the risks at infancy had been eliminated.<sup>18</sup> These rates - the post-neonatal rate in particular - reflect social and economic conditions, including the impacts of economic restructuring on Maori families, as well as health factors.

There was a period of socio-economic crisis provoked by neo-liberal restructuring during the late 1980s and early 1990s, in which many of the social welfare state props “were effectively eliminated in New Zealand” (Esping-Andersen, 1999, p. 89). This seems to have been associated with the cohort deterioration in survival discussed earlier. In the early 21st century however, there was a return to targeting sub-populations with greater needs. This was achieved by integrating primary and secondary services - for example, screening more effectively for diabetes (Smith et al., under editorial review), and by more systematic referral of more advanced cases to secondary or tertiary facilities. This meant that hospitalisations increased, and, because people (most commonly Maori or Pasifika) were presenting late, longer and more complex procedures had to be carried out. (For example, a spike in hospitalisations of Maori males around 2004, especially of older men diagnosed with cardiovascular causes appears to have come from improved screening and diagnosis at a primary health care level, coupled with increased referral into the hospital system.) Hospitalisation utilisation expectancies<sup>19</sup> for Maori men aged 70 years increased, by comparison with years before and after that date, but, interestingly  $d(x)$ , the ultimate measure of the success or failure of the system, were lower (Pool et al., forthcoming).

These three interventionist periods directed at gaps in health status could be summarised as follows:

- Period 1 (1900-1910): was one of community health approaches addressing major issues of sanitation and housing;
- Period 2 (1945-1961): saw the reduction of communicable disease mortality, both the common acute infectious diseases and tuberculosis;
- Period 3 (early 2000s): complex campaigns against chronic non-communicable diseases and their co-morbidities.

## Conclusion

Horizontalisation and verticalisation have occurred for both parts of a dichotomous split of the New Zealand population but much later, yet faster, for Maori than for non-Maori. A gap in health status between Maori and non-Maori has always existed and still exists, yet there is an indication of long-term convergence. Over three periods, the first decade of the 20th century, post-World War II and during the first decade of the 21st century convergence was accelerated by policies that aimed at closing gaps, and involved direct intervention at a community and individual level.

The first comprehensive intervention showed how much can be achieved by simple community health measures. The second, after World War II, shows that a great deal can be achieved by public health and bio-medical technologies appropriate for campaigns directed primarily at communicable disorders. But these data also show that gains achieved in this way, without a systematic and continuing underpinning of this by improved social and economic wellbeing, and the careful monitoring of higher risk populations, may be difficult to sustain. In New Zealand this was compounded by the negative impacts of economic and social policy, introduced for economic restructuring purposes rather than health development goals, using neo-liberal strategies, and without recognising the differential cohort health vulnerabilities in some ethnic groups. Under these circumstances, as in New Zealand, improvements in survival may decelerate or even show signs of cohort deterioration.

There is an irony to add to this: because of the success of earlier health programmes, there has been a shift-share in ages at which increased morbidity and  $d(x)$  cluster. Again, because of successful earlier disease control programmes, there has been a parallel shift-share in the mix of causes of morbidity and mortality -- from communicable causes, which are more likely to respond to simpler interventions, to non-communicable diseases that require more complex responses. Moreover, these are exactly the causes and ages at which co-morbidities confound the effects of interventions. The response that came in the 2000s, necessarily involved more complex forms of intervention than those which had achieved the post-war health gains for Maori. The prognosis must be that to achieve further closing of gaps (and also those between Pasifika and the Pakeha population)

must involve increasingly complex, multi-tiered programmes of screening, primary care, referral and secondary or tertiary procedures.

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## Notes

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- 1 We refer here to the northern, western and peninsular-Mediterranean countries and micro-states of continental Europe, as far east as Austria (excluding the states in transition – the Baltic, central European and Balkan states that were formerly a part of the ‘Soviet Bloc’), the Russian Federation and Turkey; plus Europe’s offshore island-nations - Malta, Iceland, Ireland and the United Kingdom; and, outside Europe, Australia, Canada, Japan, New Zealand and the United States. For Europe, this could be seen as countries to the west of the Hajnal line.
- 2 The use of the term ‘non-Maori’ here refers to people who at the time of recording information did not state that they were of Maori ethnicity. It does not purport to be in any way regarded as an exclusive ethnic group and is used with the awareness that this traditional term is fraught. Thus non-Maori is a diverse residual.
- 3 Citing Lexis, whose ideas on the concept of ‘normal life durations’ built on the work of both Quetelet and Laplace to give statistical power to his notions on “...common human longevity... [T]he concept of an average man is not an arithmetic mean, but a typical, central value along a normal curve that expresses the deep nature of things. The modal age at death...” (Cheung, S. et al., 2005, p. 246).
- 4 Unfortunately we cannot at present take the non-Maori back much further, but ongoing work by Professor Ian Pool, as part of a broader analysis of New Zealand’s demographic history, will attempt to make estimates for earlier periods to see whether non-Maori tables at around 1860 showed compression at young ages.
- 5 The co-authors of the present paper, Boddington, Cheung and Didham, played instrumental roles in the construction of these tables, along with the lead researcher Kim Dunstan.
- 6 This is despite the fact that New Zealand had instituted free, compulsory education for Maori and non-Maori as early as 1877, an undertaking fully implemented by late in the 19th century. Moreover, it had a welfare state dating from the 1890s but then greatly extended by the 1938 Social Security

- Act, which covered Maori and non-Maori equally, and the administration of which was dependent on the reporting of age and other vital details.
- 7 For Maori this tendency is far more marked. Even as recently as 1956, there were 13 Maori and 22 non-Maori centenarians, yet the non-Maori population was 15 times the size of Maori at that date.
  - 8 The centenarians of today were born at the beginning of the 20th century and their comparators for studies of improvements in survival were born between the 1870s and 1900. Among non-Maori the quinquennial male birth cohort size between 1876-81 and 1901-06 rose from 43,350 to 57,375. Then, after allowing for changes in the survival regimes over their life spans to age 99 years experienced by these birth cohorts between these birth dates and when they became centenarians, the number of centenarians would be expected to have tripled between the 1970s to around 2001-06. A major inter-cohort improvement in the survival of these cohorts came before they had reached age 35 years, a decrease of 6,701, or 29% at  $(35)d(0)$ , compensated by a small increase of 3,088, or a rise of 7%, for  $(40)d(35)$ , and a further increment of 2,799, or 8%, for  $(20)d(75)$ , the ages at which compression is being felt. There was also a significant percentage change at 95+ years, 131%, but only a numerical increase of 814. Standardising for duration, per year of age there were 191 fewer deaths at 0-34 years, but 77 more per year at 35-74, 139 more at 75-94, and an extension in the force of mortality of 81 life-table deaths per year at age 95 years and over. Taking absolute values, the gains in survival at age group 0-34 years accounted for 50% of this shift-share, and the increases in  $d(x)$  at 35-74 for 23%, at 75-94 for 21% and at 95+ for 6%; or weighted for differing durations, 39% at 0-34, 16% at 35-74, 29% at 75-94 and 16% at 95+. These data allow us to infer that longevity extensions have occurred, but the evidence on the oldest-old is weak in part because of data and measurement problems.
  - 9 If all Hispanic-Americans are counted as 'non-European', then New Zealand and the United States share similar proportions of 'non-European', but many Hispanics are classified as 'white'.
  - 10 A ship with smallpox aboard berthed in Wellington in the 1840s but passengers were not allowed to land. A small-scale smallpox epidemic did occur in 1913 in a region with many Maori, but was quickly contained by a team led by (Dr) Sir Peter Buck (Te Rangihiroa), who was a Member of Parliament and a medical practitioner, and who later became a distinguished Professor of Anthropology at Yale and the leading Pacific ethnographer of the 1920s. He also wrote what is probably New Zealand's first piece of modern epidemiology on this epidemic (Te Rangihiroa, 1914), a study which, inter alia, compared fatality rates between the vaccinated and non-vaccinated.
  - 11 A separate analysis underway by Professor Ian Pool is comparing mortality in the British Isles, especially inter-country regional differences for England with those in New Zealand. There was massive immigration from the British Isles into New Zealand in the 1860s and 1870s, after which flows dropped off radically, almost to nothing. A further analysis, looking at these migration trends, will review  $(15)p(0)$  prior to when the main inflows to New Zealand occurred, for English regions from which major emigrant flows to New Zealand were drawn, and analysed by Robert Woods (2000) in his seminal book, to

- compare with the  $p(x)$  values at older ages in New Zealand around 1876-1881. The analysis is partly to test for selective migration, and partly to respond to a comment by Jim Oeppen about the low correlation between socio-economic status and mortality in 'healthy districts' in the "Home Counties", and James Vaupel about Sweden, oral presentation of Pool and Cheung 2005 (reported in 1) at the Max Planck Institute, Rostock. A parallel test for the effects of selective migration on Pakeha fertility, which was very high in the 1870s (TFR 7.0) showed no relationship between New Zealand fertility levels and British region of origin (Pool et al., 2007).
- 12 The Italian demographers Gabriella Caselli and Viviana Egidi wrote a series of instructive papers on Italy and Europe highlighting geographical differences between  $e(0)$  and adult  $e(x)$ , which raise important questions about the varying paths of the epidemiologic transition (Caselli & Egidi, 1980; also 1981).
  - 13 Only one measure, when the population drops below the 90th percentile, is used here. First cohort data are drawn on, and then recent period tables are analysed.
  - 14 The TFR moved from 7.0 in 1876 (a marital TFR of almost 9.0) to 3.5 by 1901, a change resulting from what Dutch demographers Engelen and Kok describe as closing down the 'nuptiality valve' (cited Pool et al., 2007). This had major impacts on families, including decreases in child accident death rates as supervision of younger siblings shifted to parents from older siblings; all the expected benefits from improvements in housing, disposable income/ family member and thus things like nutrition, and declines in risks of cross-sibling infection and overcrowding. Add to this declines in maternal mortality, and, more importantly, decreases in female death rates at reproductive and post-reproductive ages due to a wide range of causes, notably tuberculosis, determined at least in part by the 'physiological burden of child-bearing' (Pool and Cheung, 2005, citing American bio-demographer Ingrid Waldron).
  - 15 The Pasifika minority in the non-Maori population were as adversely affected as Maori.
  - 16 Clearly there are others, including social class and geographical differences. Because of the social demography of New Zealand, these tend to be confounded by the ethnic differential on which we have focused.
  - 17 It is worth recalling that in 1940, Japan's  $e(0)$ s were only 48 years (m) and 51 (f); Australia's were 64 (m), 68 (f), England and Wales 59(m), 64 (f), Sweden 65 (m), (68 (f).
  - 18 1981: Neonatal (month 0), Maori 6.3, non-Maori 5.4; Post-neonatal, Maori 9.1, non-Maori 5.7, (Sceats & Pool, 1985, Table 147); 2006: Neonatal, Maori 4.6, non-Maori 2.1; post-neonatal, Maori 4.6 and non-Maori, 1.5. The post-neonatal rates reflect social and economic conditions as well as health factors.
  - 19 Life-tables constructed using a Sullivan's observed prevalence method, giving expectancies (days) for admission to hospital while still surviving (Cheung, J. et al., 2001).

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