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## An Aberrant Trend

With population ageing becoming an issue of major importance for societies in the developed countries, in both the scientific and policy communities there is widespread interest in the determinants of these structural changes (eg. Tuljapurkar, Li and Boe 2000). The focus has been on declines in fertility, arguably the major causal factor, but increasingly analyses are turning to the other major determinant, improving survivorship (Calot and Sardon 1999). This paper relates to this aspect of ageing but not to fertility *per se*.[1]

Conventionally, it had been argued that improvements in survivorship were important as determinants of the probability of reaching old age, say 65 years and over, but that within older age-groups few radical increases in expectation could be expected. It was posited that there were strict limits to human longevity and that even the most epidemiologically advanced societies had reached, or would shortly reach, these limits (reviewed Oeppen and Vaupel 2002: esp. Figure 1).

This has provoked debates around two themes. The first is whether there really is an upper limit to human longevity. The second relates to what might be the durations between when (i) premature death would occur among members in any cohort, (ii) that and the cohort "average" age at death, and (iii) ages at death of those dying at a "grand old age" (Robine 2001; for New Zealand see Cheung 1999 and 2001), and to the dynamics of morbidity related to this. In this regard three different explanatory paradigms have been put forward (compression, extension and dynamic equilibrium, demonstrated respectively, for example, by the work of Fries (1980), Olshansky (1993) and Manton (1982). Linked to the duration question are also arguments about linkages between mortality, morbidity and good health, an issue outside the scope of this paper (see Crimmins *et al.* 1989; Robine and Romieu 1998 )

Those arguing that there may be no limits to human longevity, or at least that it may

reach significantly higher levels than those observed, point to the fact that some life expectancies recorded recently have exceeded the levels that various experts have posited as upper limits. This gap between what might be expected and what is observed is not just a contemporary phenomenon. Oeppen and Vaupel (2002) have used data on Pakeha (European origin) New Zealanders to show that earlier in the 20th century this population had already achieved levels of longevity above what the pundits of that period were projecting.

Indeed, in an analysis of Pakeha demography, the American demographer Campbell Gibson had established New Zealand's early advantaged situation and thus set the argument to be pursued in this paper:

> From perhaps as early as the middle of the 19th century until the 1930s, New Zealand had the lowest mortality of any country on record. Comparison with mortality in England and Wales indicates that New Zealand's lower mortality was broadly based among various age-groups and causes of death. Most striking perhaps is New Zealand's record in the reduction of infant mortality (1972:173).

With these points in mind the central aims of this paper are to review New Zealand longevity prior to World War II. Firstly it evaluates whether or not New Zealand life-expectancies were really the highest on record at that time, or at least as elevated as the data suggest. As the quotation above shows, there is no doubt that levels were high, and thus a second aim is to analyse why this might have been the case. This second objective raises questions that are of wider interest for researchers on human longevity.

In this paper three hypotheses relevant to the general objectives are addressed:

1. That the favourable position of New Zealand was really artefactual, a function of data quality (inadequate data at infancy prior to 1900), the index selected to illustrate longevity ($e_0$ vs $e_x$) and the restriction of analyses to Pakeha alone (the exclusion of the indigenous Maori from early "national tables).
2. That rapid improvements in ${}_nP_x$ prior to 1900 produced momentum effects in

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1 Nor to migration, a third dimension, nor to age-structural momentum effects.

cohort survivorship that favoured Pakeha well into the 20th century.

3. That a mix of causally more remote determinants – a fertility decline, advantageous social and economic conditions, and the introduction of welfare state measures in the 1890s – placed Pakeha in a favourable situation as early as the 1880s, and that this was reinforced in the decades either side of the dawn of the 20th century.

## The New Zealand Context

### *Studying Historical New Zealand Mortality Patterns*

An interest in New Zealand life expectancies, and particularly the advantages the country seemed to have over similar societies has been very long standing, at least in the national literature and even internationally (United Nations 1953:65). In Europe Victorian officials and intellectuals were fascinated by natural sciences and by "moral" statistics, and this held true for this class of New Zealand settler, albeit that the colony seemed so remote from the mainstreams of scientific thought. Thus, as early as 1882, when the first life-tables became available, a physician, Dr Alfred Newman, published a paper "in conjunction with my friend Mr Frankland [whose] great mathematical powers and his long and thorough acquaintance with the vital statistics of the Colony are an absolute guarantee of their correctness" (1882). This article, based on a presentation to the Wellington Philosophical Society, was published by the Colony's fledgling scientific academy, the *Transactions and Proceedings of the New Zealand Institute* (later to become the Royal Society of New Zealand), and was entitled *Is New Zealand a Healthy Country?* It concluded "Mr Frankland's statistics show that New Zealand possesses the lowest death rate of any country in the world; and that the conditions favourable to life and common to all ages."

Newman not only presented comparative data, but also mused on the advantages, especially climatic, that New Zealand had over Australia and Canada. One would like to view his paper as constituting an exercise in pure scientific curiosity, but like many

of his late Victorian peers Newman was also a social Darwinist who saw Pakeha as a superior race supplanting an inferior indigenous people, the Maori. At that time they were perceived to be on the verge of extinction by most observers, whether they were sympathetic, or, like Newman, seeing this as historical inevitability. In his words "... the disappearance of the race is scarcely subject for much regret. They are dying out in a quick, easy way and are being supplanted by a superior race" (1881: 477).

By the 1890s Maori were indeed only about seven per cent of the total population (from 50 per cent around 1860 and almost 100 per cent in 1840). They then dipped further below this level, but had edged back up to six per cent in 1945 (Papps 1985: Table 9), to reach about 15 per cent today. The rapid percentage–point decrease in relative contribution to the national total, 1860-90, was primarily a function of the rapid Pakeha population increases. Over that period Maori numeric decreases were certainly rapid but more modest (from about 60,000 to 44,000), than was true for the shift in their share of the national population.

Very large inflows, particularly around 1870, of non-Maori settlers, mainly British but some Scandinavian and German in origin, came as a result of policies intended to "swamp" Maori (Belich 1996). In 1860, Pakeha had also numbered around 60,000, but by 1891 when numerically Maori were at their nadir (44,000), the Pakeha population had grown tenfold, and went on to reach one million in 1911. For Maori, however, the 1890s did see a "renaissance" set in, led by a Maori Cabinet Minister (Sir James Carroll), and supported by Maori doctors, (eg. Sir Peter Buck, Sir Maui Pomare), parliamentarians and others (Pool 1991: *passim)*.

By 1901, the mortality and census data available for Pakeha were of high quality, at least by the standards of that day. In contrast, until about World War II Maori data were still very inadequate (Pool 1985: 213-218), and for the "oldest of the old" may be imperfect even today (Kannisto 1994: 14-16). However, there are questions about the quality of Pakeha infant mortality data in the period prior to 1900 (Sceats and Pool

1985a: Table 141). The present paper updates that analysis as a part of an investigation of its first hypothesis.

*Economic and Social Context*

In general, therefore, this paper deals with the Pakeha population simply because they are close culturally and ethnically to other societies in Western developed countries. That said, Maori data will be included at two later points to give a "national" picture.

The economic and social historical context of this paper starts in the late 1860s. By then, the "New Zealand Wars" between some Maori and the Crown were almost over, and immigrants were being recruited in large numbers primarily to settle land confiscated from tribes that had fought the Crown. As the area available proved far from sufficient, new legislation individualised Maori land titles and set up judicial processes, the Native Land Courts,[2] essentially to provide a legitimation to the wholesale wresting from Maori of the land they still held. This had been largely achieved by the early 1900s, and had provided the means by which huge inflows of Pakeha could settle and make New Zealand a "better Britain" (Belich 2001: passim). This most effective of population policies, entitled *The Immigration and Public Works Act* (1870), initially favoured the development of sheep (for wool) and grain farming. But refrigeration of exports (1882) and genuine land reform in the 1890s, saw New Zealand set up a society of small business-people (owner-operated dairy and sheepmeat farms), and a very efficient and productive, rural, export, pastoral economy that was to persevere in this form until the United Kingdom entered the Common Market. Thus New Zealand over the entire period 1890 to 1970 was virtually "a town supply district of London" (Belich 2001:30). At the same time the primary sector labour force was highly efficient and, proportionately small, so that New Zealand became "urban" at a very early stage (Pool and Bedford 1997).

In common with some American and Canadian Prairie states/provinces and the

2 Called the "Land-Taking Court, te Kooti tango whenua" by Maori, Williams (1999).

Australian colonies, New Zealand introduced welfare measures at an early stage (eg. free, compulsory, secular education for both Maori and Pakeha, in 1877, fully implemented by the 1890s). These legislative initiatives were most radical in the 1890s and early 1900s under Richard Seddon's populist Liberal Party. This embryonic Social Democratic government introduced "state experiments" that excited the attention of Fabians and other European socialists, for these were measures dreamt of but not implemented in Europe. They included universal franchise (men and women, Maori[3] and Pakeha) old age pensions and industrial conciliation and arbitration,[4] and initiatives that substantially increased the role of the state (Belich 2001: 38-46; see also Sinclair 1959: Part 2, Chapt. II).

The early 1900s saw the end of the basic pioneer phase and the country settled into a situation of equilibrium despite enthusiastically joining in two World Wars,[5] and suffering severely in the depression of the 1930s. The equilibrium was based around a pastoral economy exporting overwhelmingly to the "mother country", and, in turn, importing almost exclusively British manufactured items. Linked to this was a low level of population redistribution (Pool 2002). Except in depressions income levels were high, and at the turn of the 20th century New Zealand enjoyed both a favourable balance of trade and one of the highest levels of Gross Domestic Product of any country at that time (Hawke 1985: 73, 77). There was, however, polarisation between Pakeha and Maori whose economy was semi-subsistence and who lived in isolated regions in the north and east of the country. Paradoxically, interactions at an individual level also occurred as did very high levels of intermarriage, while Maori

3 All Maori men had already received the vote in 1867 before all Pakeha men.

4 This Act, 1894, went far beyond labour relations "in that it provided the structural framework for income redistribution" (Michael Law, Sociology and Social Policy, Waikato University, Personal Communication).

5 Of a population just over one million, 100,000 men went overseas in World War I, mainly to the Western Front and Gallipoli, but also Samoa, Mesopotamia and Palestine. Of these some 17,000 perished. Unfortunately, no detailed data were available to allow us to estimate the effects of these deaths on synthetic and cohort tables.

were represented in parliament (and in Cabinet), and there was never *de jure* segregation in institutions such as education (Pool 1977: Chapt.2).[6] The election of a Labour government in 1935 saw the introduction of a Scandinavian style welfare system, to which both Maori and Pakeha had equal access.

**Trends in Life Expectation and Mortality**

Table 1 presents data on life expectation and survivorship across broad age-ranges ($_nP_x$) for both Maori and Pakeha. Maori data come from analyses carried out by Ian Pool, Pakeha from the work of Jit Cheung. Pakeha tables follow actuarial conventions, whereas Maori life tables are constructed using best estimates taken from a range of non-conventional, indirect estimation techniques, mainly involving fitting distribution to Coale-Demeny model tables. They are included merely as indicators of trends.

Data on causes of death for Pakeha became available as early as the 1870s. While certification standards were low and the categories used would not meet modern criteria, the results do have some utility for the later part of this section of the paper. There are, however, two sets of causes that have meaning today: accidental death and phthisis (tuberculosis). Presumably most accidents and violence could be diagnosed and did not need clinical reporting. Donovan (1969) has argued that tuberculosis is one of the few major diseases that was, historically, reported adequately enough to allow relatively meaningful time series analysis.

Three major trends are evident in Table 1. Firstly, Pakeha levels of expectation and survivorship were not only high but far exceeded those for Maori. By 1901 Pakeha women were reaching 60 years expectation at birth.

Secondly, Pakeha infant and childhood survivorship improved rapidly in the late 19th century, but this gain then slowed thereafter. This shows up better in data for 1876,

6 In districts where Maori were concentrated “Native Schools” existed, but Pakeha could attend these, and Maori could attend general schools.

1896 and 1916 in Table 2 on survivorship to age 10 years. The year 1906 is also included there to show that the deceleration had occurred by the turn of the century. This is also clear in cause-specific data discussed below Table 3. The importance here of the period 1876-1896 will be returned to when the third hypothesis is investigated.

**Table 1: Values for $e_x$ (years)and broad $_{15}p_x$ selected years, Pakeha [and Maori] New Zealand, 1881-1931**

| Gender | | 1881 | 1891 | 1901 | 1911 | 1921 | 1931 |
|---|---|---|---|---|---|---|---|
| Males | $e_0$ | 52.9 | 55.2 (25) | 57.6 | 60.5 | 61.9 | 65.3 |
| | $e_{20}$ | 43.9 | 45.0 | 46.3 | 47.4 | 48.1 | 49.7 |
| | $e_{40}$ | 28.6 | 29.2 | 30.0 | 30.5 | 31.1 | 32.1 |
| | $e_{60}$ | 15.3 | 14.8 | 15.2 | 15.4 | 15.8 | 16.3 |
| Females | $e_0$ | 56.4 | 58.1 (23) | 59.9 | 63.2 | 64.3 | 67.8 |
| | $e_{20}$ | 46.5 | 46.8 | 47.8 | 49.2 | 49.7 | 51.4 |
| | $e_{40}$ | 31.5 | 31.4 | 31.6 | 32.5 | 32.8 | 33.8 |
| | $e_{60}$ | 17.1 | 16.4 | 16.5 | 16.6 | 17.0 | 17.4 |
| Males | $_{15}p_0$ | 0.834 | 0.855 | 0.873 | 0.901 | 0.913 | 0.940 |
| | $_{15}p_{15}$ | 0.926 | 0.931 | 0.946 | 0.958 | 0.958 | 0.965 |
| | $_{15}p_{30}$ | 0.883 | ***0.904*** | 0.912 | 0.921 | 0.924 | 0.942 |
| | $_{15}p_{45}$ | 0.759 | 0.789 | 0.807 | 0.819 | 0.834 | 0.852 |
| Females | $_{15}p_0$ | 0.854 | 0.877 | 0.889 | 0.919 | 0.928 | 0.952 |
| | $_{15}p_{15}$ | 0.930 | 0.934 | 0.945 | 0.956 | 0.956 | 0.969 |
| | $_{15}p_{30}$ | 0.887 | ***0.897*** | 0.914 | 0.928 | 0.931 | 0.948 |
| | $_{15}p_{45}$ | 0.819 | 0.830 | 0.831 | 0.856 | 0.860 | 0.877 |

Note: Figures in bold italics are where female $_np_x$ are lower than male. Figures in parentheses are estimates of Maori life expectation in 1891 (Pool 1994: Table 1.4). The Maori gender pattern is estimated by reference to the Maori 1945 table, directly calculated from data of a reasonable quality (Pool 1994: Table 7.6). Moreover, age-sex structures used to fit to model tables had relatively high levels of masculinity, a pattern that persisted until well after World War II, and that is not explained by gender biases of the sort seen, say, in Asia. In 1945 male expectation at birth was 48.8 years, and female 48.0. Official Maori tables became available only in 1951.

**Table 2: Pakeha, probabilities of surviving to age 10 years, 1876, 1896, [1906] and 1916**

| | Male | Female |
|---|---|---|
| 1876 | 0.81571 | 0.83544 |
| 1896 | 0.88090 | 0.89520 |
| [1906] | [0.88839] | [0.90545] |
| 1916 | 0.91285 | 0.92883 |
| Absolute change 1876-1896 | 0.06519 | 0.05976 |
| Absolute change 1896-1916 | 0.03195 | 0.03363 |

Data for 1876 are linear interpolations between census years 1874 and 1878. From 1881, New Zealand joined the Empire census programme and censuses then became regular in years ending in a one or a six.

Finally, there is evidence of a sex crossover at 30-45 years in survivorship from 1891, when male rates at these ages exceeded female to later years when the reverse was true. Data on causes (Table 4) will allow further explanation of the crossover, and why the inverse had held true previously (1881). Male advantage in survivorship and even longevity may well have held true historically in pre-transition Europe (Stolnitz 1956: 23-24; see also Stolnitz 1955).

In Table 3 cause-specific death rates are presented for childhood ages. It should be clear that the diagnostic and certification standards at that time were far below present day best practice. This is most evident for the category “other” causes. The category “diseases of early infancy” was recorded in a particularly unsatisfactory way and detailed analysis of it produces peculiar results (Pool 1985: Note for Table 122). Equally well, it is unclear why so many deaths came into the cardiovascular and neoplasm category at 0-4 years.

**Table 3: Pakeha children, 0-4 and 5-14 years, cause-specific death rates (per 100,000), 1876, 1896, 1916**

| | | 0-4 years | | | 5-14 years | | |
|---|---|---|---|---|---|---|---|
| | | 1876 | 1896 | 1916 | 1876 | 1896 | 1916 |
| Infectious diseases, excluding tuberculosis, including influenza | M | 66.3 | 15.8 | 17.5 | 18.4 | 3.0 | 4.3 |
| | F | 84.2 | 18.2 | 18.7 | 21.6 | 4.4 | 5.9 |
| Tuberculosis, all forms | M | 15.1 | 3.1 | 3.1 | 2.2 | 1.3 | 1.7 |
| | F | 12.3 | 2.9 | 3.4 | 3.0 | 1.2 | 1.6 |
| Respiratory (excl. Tuberculosis and influenza) | M | 56.4 | 43.1 | 18.2 | 3.8 | 2.1 | 1.4 |
| | F | 41.3 | 32.5 | 14.8 | 3.4 | 2.5 | 1.1 |
| Heart diseases, neoplasms etc | M | 14.1 | 3.5 | 2.8 | 3.4 | 1.2 | 1.2 |
| | F | 10.3 | 1.9 | 1.4 | 3.4 | 0.4 | 1.1 |
| Accidents and violence | M | 15.8 | 11.5 | 6.8 | 6.4 | 4.7 | 4.3 |
| | F | 14.3 | 9.2 | 3.7 | 2.7 | 2.2 | 1.4 |
| Diarrhoeal and related diseases | M | 86.3 | 30.4 | 14.8 | 1.5 | -- | 0.3 |
| | F | 62.2 | 27.9 | 12.1 | 1.8 | 0.1 | 0.4 |
| Other causes (including congenital abnormalities, ill defined and diseases of early infancy) | M | 169.4 | 138.1 | 99.1 | 6.6 | 6.8 | 7.1 |
| | F | 138.3 | 117.3 | 76.1 | 6.4 | 7.1 | 5.6 |
| Total all causes | M | 423.4 | 245.5 | 162.3 | 42.3 | 19.1 | 20.3 |
| | F | 362.9 | 209.9 | 130.2 | 41.4 | 17.9 | 17.1 |

Source: Pool 1985, Table 122

Nevertheless, some clear and interesting trends are evident. There were dramatic decreases over the period 1876-96 in the “acute infective diseases” (Lancaster 1952a and b) of childhood, tuberculosis and the diarrhoeal diseases, less significant changes in the heart diseases etc. category, and radical but still significant declines in accidents and violence, respiratory diseases and other causes. At 5-14 years the decreases 1876-96 in infectious causes alone effectively almost halved mortality from all causes at these ages.

The data on causes in Table 3 thus show that improvements in survivorship at childhood occurred across a wide range of causes, including accidents and violence. In the absence of either modern chemotherapeutics, or an efficient health system,

explanations must be sought in social and economic trends. These will be analysed below, but suffice to say that the fertility declines, to be noted there, would have had an effect in numerous ways: for example, increases in levels of per capita resources within families, decreases in cross-sibling infection and in overcrowding, especially in the number of persons per bedroom, and shifts in child care responsibilities from older siblings to parents.[7]

**Table 4: Pakeha adults, sex-specific death rates (per 10,000) by cause,[a] ages 25-44 and 45-64 years, 1876, 1896, 1916**

| | | 25-44 years | | | 45-64 years | | |
|---|---|---|---|---|---|---|---|
| | | 1876 | 1896 | 1916 | 1876 | 1896 | 1916 |
| Tuberculosis | M | ***14.5*** | ***13.6*** | 11.8 | 19.3 | 14.2 | 7.7 |
| | F | ***16.7*** | ***14.7*** | 10.2 | 12.9 | 8.4 | 4.7 |
| Chronic non-communicable diseases and maternal mortality[b] | M | ***21.4*** | ***20.6*** | ***22.0*** | 115.5 | 106.4 | 94.9 |
| | F | ***52.3*** | ***30.6*** | ***27.3*** | 104.2 | 99.7 | 92.2 |
| Respiratory (excl. Tuberculosis and influenza) | M | ***6.2*** | 5.4 | 3.8 | 28.2 | 17.5 | 12.4 |
| | F | ***6.5*** | 4.4 | 2.5 | 17.2 | 11.5 | 6.9 |
| Infections (excl. Tuberculosis and influenza) | M | ***6.9*** | *3.6* | 2.5 | 7.9 | 5.9 | 2.9 |
| | F | ***8.0*** | *5.3* | 2.0 | 6.0 | 5.6 | 1.7 |
| Accidents and violence[c] | M | 22.8 | 14.7 | 12.5 | 30.5 | 20.2 | 17.0 |
| | F | 2.9 | 2.0 | 2.0 | 4.3 | 4.1 | 3.1 |
| Other causes (including ill-defined[d] and diarrhoeal diseases) | M | 15.7 | 0.2 | 0.4 | 7.0 | 0.8 | 1.4 |
| | F | 2.9 | 0.2 | 0.2 | 4.4 | 0.8 | 0.7 |
| Total all causes | M | ***87.5*** | 58.1 | 53.0 | 208.4 | 165.0 | 136.3 |
| | F | ***89.3*** | 57.2 | 44.2 | 149.0 | 130.1 | 109.3 |

Source: Pool 1985: Table 12.2. figures in bold italics are for cause-specific rates where female exceed male.

Notes: a. Tuberculosis and accidents and violence were probably the most reliably reported. Influenza was often put in the infectious rather than respiratory categories. For all years fitting categories to match modern ICD's is very difficult.

b A very wide group which includes cardiovascular diseases, cancers, senility and, for females, maternal mortality. Maternal mortality ratios and rates can be computed but produce counter-intuitive results suggesting that maternal causes were classified elsewhere.

c. It seems that in 1916 war deaths were not included.

d. In 1876 mainly ill-defined for males.

[7] For explanations of declines in acute infective diseases in Australia in the 1920s see Lancaster 1952a and b.

Changes in the proximate causes of the adult sex crossover in survivorship noted earlier can be seen in patterns of decline for numerous causes at reproductive ages and menopause, as seen in Table 4. For females, the "physiological burden of childbearing" would have been reduced by the fertility declines affecting deaths from non-communicable disorders. But there are also associations between communicable diseases such as tuberculosis and childbearing in high mortality, high fertility societies (Waldron 1982:98). By contrast, male rates of accidental and violent death far exceeded female especially in 1876, but, as New Zealand's economy shifted from dangerous exploitative industries (mining; timber, especially the native hardwoods) to intensive pastoralism, and the forest clearing for farming was drawing to a close (late 1890s), so too did exposure to risk of accidental death decrease. Thus the male rate for ${}_{30}p_{15}$ in 1881 was lower than the female, but the reverse became true in 1891. In spite of this, in 1876 male death rates overall at 25-44 had still been lower than female.

**Towards Explanation: Artefact or Reality**

A first operational hypotheses to explain the high levels of life expectation for Pakeha at this time would be:

> That rates were in part an artifact of under-registration of Pakeha deaths (Maori deaths were not registered at this time).

Sceats and Pool (1985a: Table 141; Figure 34), using trends in neonatal deaths, suggested this as a possibility, but identified no corresponding under-registration at post-neonatal ages. Their reasoning was based on (i) an apparent increase in rates around 1885-90; and (ii) comparisons with the more regular curve in the rates for Sweden, another low mortality population at that time. The occurrence of many births would have taken place outside hospitals (Pool 1982: 13), and this meant a means for the enforcement of registration was often missing. But, there might also be a "real" explanation: the period around 1890 (Pool 1982: Fig.4, 26-27) saw the arrival of a virile pandemic of influenza – the first outbreak world-wide since the late 1840s – that unlike its successor in 1918, followed classic age-specific patterns.

To investigate this further, ${}_nP_x$ values observed Pakeha were compared with those in 'West" model tables. Under-registration at infancy would see lower observed ${}_1P_0$ than in the models, and the ratios between ${}_1P_0$ and ${}_5P_0$, and subsequent ${}_nP_x$ would be marked. The ratios are shown in Table 5.

**Table 5: Comparisons between model life table West levels 13, 15 and 17 (male) with 1881 and 1891 non-Maori survival rates**

| | Model life table | | | non-Maori males | | |
|---|---|---|---|---|---|---|
| | **Level 13** | **Level 15** | **Level 17** | **1874** | **1881** | **1891** |
| **Probability** | | | | | | |
| ${}_1P_0$ | 0.86058 | 0.90661 | 0.91379 | 0.87676 | 0.90043 | 0.9054 |
| ${}_4P_1$ | 0.92915 | 0.94999 | 0.96504 | 0.92983 | 0.95699 | 0.96747 |
| ${}_5P_5$ | 0.97941 | 0.98428 | 0.98822 | 0.97374 | 0.97942 | 0.98539 |
| ${}_5P_{10}$ | 0.98509 | 0.98781 | 0.99119 | 0.98361 | 0.98789 | 0.99019 |
| **Ratios** | | | | | | |
| ${}_4P_1 : {}_1P_0$ | 1.080 | 1.048 | 1.056 | 1.061 | 1.063 | 1.069 |
| ${}_5P_5 : {}_1P_0$ | 1.138 | 1.086 | 1.081 | 1.111 | 1.088 | 1.088 |
| ${}_5P_{10} : {}_1P_0$ | 1.145 | 1.090 | 1.085 | 1.122 | 1.097 | 1.094 |
| ${}_5P_5 : {}_4P_1$ | 1.054 | 1.031 | 1.024 | 1.048 | 1.023 | 1.019 |

The rate and ratio comparisons suggest that infant and childhood probabilities of survival estimated for non-Maori in 1881 and 1891 are largely in line with what would be expected under model life tables conditions. Given the levels of ${}_4P_1$, ${}_5P_5$ and ${}_5P_{10}$, ${}_1P_0$ might, if anything, be slightly under-estimated (ie. the registration data produce a rate which is too low, thus negating the hypothesis of under-registration in infancy), or alternatively ${}_4P_1$ has been slightly over-estimated. In both cases the differences are small. Thus, it can be concluded that if under-registration had occurred in the 1880s and 1890s, it would have been very slight.

At ${}_1P_0$ in 1874 (the first New Zealand table) there does, however, seem to be an indication of the effects of under-registration at infancy. The ratios are also lower in the New Zealand table than in "West 13".

This latter result, contrasting 1874 with 1881 and 1891, actually strengthens the argument about improving survivorship at infancy and childhood. Had ${}_{10}p_0$ in 1876 approximated (been a little higher than) that for West 13, say 0.80424, then the change between 1876 and 1896 would have been +0.07666 rather than +0.06519. All later analyses using 1876 data are based on observed rates rather than adjusted, and therefore provide conservative estimates of the speed and magnitude of change in infancy and childhood 1876-96. The case for momentum effects to be made later is thus understated.

A second "explanation" is more in the form of a caveat. The data putting New Zealand in a favourable light excluded Maori. Once Maori are included, New Zealand expectancies are seen to have been equal to or lower than those for Norway, as is shown in Table 6. That said, as noted earlier, the interesting comparison in this paper is really between the European-origin settler population and other West-European societies.

**Table 6: Female life expectation in years, 1881**

| | $e_0$ | $e_{20}$ | $e_{40}$ | $e_{60}$ |
|---|---|---|---|---|
| NZ Pakeha | 56.4 | 46.5 | 31.5 | 17.1 |
| NZ total (including Maori) | 51.3 | 43.2 | 28.6 | 13.8 |
| Norway | 51.3 | 45.6 | 31.5 | 16.9 |

Sources: Pakeha, Jit Cheung; Total, Pool 1982: Table 7; Norway, Central Bureau of Statistics (1969): Table 32 (mean of 1871-80 and 1881-90)

A third explanation and operational hypothesis relates to choice of indices:

> That Pakeha were favoured by high levels of survivorship and expectancies at younger ages, but not at older ages.

There is an empirically supportable reason for this. It should be clear from the context described earlier that Pakeha were a recent migrant population. In 1873 the inflow was so high that a similar level was not reached again until about 1973. But later in the

1870s and 1880s the streams dried up as New Zealand was hit by a severe depression. The host population in the 1860s and early 1870s had been very small. Consequently, in the 1880s infants and children were typically native-born; adults, especially older ones were from overseas. Infants and children would have been the beneficiaries of any favourable conditions found in New Zealand, whereas arguably immigrant adults carried, as it were, "the baggage" of their countries of origin. There are indications, for example, that tuberculosis death rates may have been higher among the foreign-born than the native-born (Pool 1982).

In Table 7 data for 1911 are presented comparing life expectancies at various ages for New Zealand Pakeha, Norway and Sweden. At older ages Pakeha (alone, excluding Maori) are below those of these other populations. In contrast, for $e_x$ at younger ages, -- that is, the whole of life or the whole of adult life -- Pakeha are favoured. A disjunction between $e_0$ and adult $e_x$'s, where populations have relatively low $e_0$'s and high adult $e_x$'s, or vice-versa has been reported in the literature (eg. for Europe see Caselli and Egidi 1981; for within Italy see Caselli and Egidi 1980).

**Table 7: New Zealand Pakeha (1911), Norway (1910), Sweden (1911): Life expectation, selected ages**

| | Males | | | Females | | |
|---|---|---|---|---|---|---|
| | NZ | Norway | Sweden | NZ | Norway | Sweden |
| $E_0$ | 60.5 | 56.4 | 56.6 | 63.3 | 59.3 | 59.5 |
| $E_{20}$ | 47.4 | 45.8 | 46.4 | 49.2 | 48 | 48.5 |
| $e_{40}$ | 30.5 | 31.6 | 31.3 | 32.5 | 33 | 33 |

Sources: New Zealand, Jit Cheung; Norway, Sweden: Preston *et al* (1972), 628-30, 652-54.

## Towards Explanation: Cohort Momentum Effects into the 20th Century from 19th Century Survivorship Gains

As has been shown, Pakeha infant and childhood survival underwent significant gains rather rapidly in the late 19th Century. By the arithmetic logic of cohort life table

construction, it is evident that this would also have consecutively affected all $l_x$ and thus person-year exposure above childhood ages as the cohort moved through its life cycle. It has also been shown that Pakeha survivorship gains were slower after 1901 than prior to then, and that in 1911 older Pakeha were disadvantaged in terms of $e_x$ by comparison with their peers in Norway and Sweden. In terms of timing, such a trajectory, as George Stolnitz showed long ago (1955, 1956), is aberrant. Across Europe and the United States "improvements had been slower during the 19th Century than during the 20th, and decennial increases were most remarkable in the first two decades following the turn of the [20th] century and in the years immediately after World War II" (cited United Nations 1973: 111). With these observations in mind it is hypothesised:

> That cohort momentum effects by comparison with period effects had a disproportional impact on life expectations in the first three decades of the 20th Century.

This hypothesis is investigated using the data in Table 8. It is clear that rapid increases in cohort ${}_{10}p_0$ had occurred by 1896, but that gains were slower after that. This momentum carried into cohort ${}_{40}p_{10}$'s, although gains at this life-cycle stage were more marked in the next cohorts. For males this will be confounded by the effects of the 1914-18 war (see fn5 above), whereas for several female cohorts gains from sex cross-overs, and thus more rapid improvements than those achieved by men could have been a factor.

The changes by the 1931-36 cohort have further significance. From the cohorts of the 1920s deterioration seems to have set in,[8] whereby for several decades each succeeding cohort's probabilities were lower than its predecessor's across some of the active ages (Pool 1983; Cheung 1999).

[8] Cheung (1999: Chapts. 8 and 10) using a range of indices on cohort and synthetic data confirmed Pool's (1983) earlier work on this effect.

**Table 8: Changes in $_{10}p_0$ and $_{40}p_{10}$, for Pakeha between cohorts of 1871-76 to 1931-36**

| | Males | | Females | |
|---|---|---|---|---|
| | $_{10}p_0$ | $_{40}p_{10}$ | $_{10}p_0$ | $_{40}p_{10}$ |
| Cohort born | | | | |
| 1871-76 | 0.81693 | 0.81759 | 0.83955 | 0.83025 |
| 1891-96 | 0.87564 | 0.85621 | 0.89329 | 0.87377 |
| 1911-16 | 0.91035 | 0.90245 | 0.92655 | 0.92663 |
| 1931-36 | 0.94733 | 0.92284 | 0.95814 | 0.95188 |
| Inter-Cohort difference (Absolute) | | | | |
| (1891-96) - (1871-76) | 0.05871 | 0.03845 | 0.05374 | 0.04352 |
| (1911-16) - (1891-96) | 0.03471 | 0.04624 | 0.03326 | 0.05286 |
| (1931-36) - (1911-16) | 0.03698 | 0.02039 | 0.03159 | 0.02525 |
| Ratio of differences in changes between pairs of cohorts | | | | |
| Late C19/Early C20 | 1.69 | 0.84 | 1.62 | 0.82 |
| Early C20/Depression | 0.94 | 2.27 | 1.05 | 2.09 |
| Ratio of Differences averaged for each 10 years of life in order to account for durations across the life-cycle | | | | |
| Late C19/Early C20 | 1.69 | 0.21 | 1.62 | 0.21 |
| Early C20/Depression | 0.94 | 0.57 | 1.05 | 0.52 |

**Table 9: Pakeha synthetic life tables, $_{10}p_0$ and $_{40}p_{10}$, 1876, 1896, 1916 and 1936**

| | Male | | Female | |
|---|---|---|---|---|
| Year | $_{10}p_0$ | $_{40}p_{10}$ | $_{10}p_0$ | $_{40}p_{10}$ |
| 1876 | .81571 | .72609 | .83544 | .74372 |
| 1896 | .88090 | .79967 | .89522 | .81170 |
| 1916 | .91285 | .82620 | .92883 | .85570 |
| 1936 | .94576 | .88107 | .95700 | .89855 |
| Inter-table difference, same age | | | | |
| 1876 | -- | -- | -- | -- |
| 1896 | +0.06519 | +0.07358 | +0.05978 | +0.06798 |
| 1916 | +0.03195 | +0.02653 | +0.03361 | +0.04400 |
| 1936 | +0.03291 | +0.05487 | +0.02817 | +0.04885 |
| Intra-table difference | $_{10}p_0$ - $_{40}p_{10}$ | | $_{10}p_0$ - $_{40}p_{10}$ | |
| 1876 | 0.09177 | | 0.09172 | |
| 1896 | 0.05654 | | 0.08352 | |
| 1916 | 0.08665 | | 0.07313 | |
| 1936 | 0.06469 | | 0.05845 | |

The cohort data in Table 8 can be compared with the results from synthetic tables in Table 9. These approximate birth periods for the selected cohort tables. The rapid changes at childhood and then the delayed impact at older ages also shows up here.

The synthetic tables also highlight the gender difference. The male momentum effect at 10 to 49 years shows up in the 1916 table (difference ${}_{10}p_0 - {}_{40}p_{10}$), whereas the sex cross-over noted earlier affects females across a number of tables.

**Towards Explanation: Social and Economic Factors**

*Background*

When analysing changes in mortality over the period under review here, particularly rapid changes 1876 - 1896 it is impossible to argue that bio-medical or public health factors played a role of any significance. The medical and public health infrastructures were primitive by 20th century standards, and medical administration was inadequate. Before 1901, for example, "what information there is shows that the local authorities were allowing sanitation to look after itself" (MacLean 1963:90). Following the *Public Health Act* (1900) a wide range of sequential Acts and Regulations were passed. The main Act and its ancillary legislative changes essentially set up the basic public health, and medical infrastructures and regulations that were to remain in place, until the 1980s. But, for the period prior to 1901, socio-economic or other non-medical explanations of change must be sought.

The last general hypothesis posited at the start of the paper thus comprises a more disparate set of determinants, the basic features of which were outlined earlier in this paper when the context was being set out. The hypothesised relationships between mortality change and these determinants are, however, not conducive to direct testing, but rather by pointing to associations with trends in various factors co-varying over time with mortality changes.

The period being covered in this paper started from a base that was favourable economically and demographically. By 1865 “incomes in New Zealand... were on average significantly higher than in Australia which in turn were higher than in the United Kingdom or North America” (Hawke 1985: 76). The immigrants around the 1870s typically had their passage paid, they were selected for job experience and other factors, and, to the extent that this is meaningful, were tested for phthisis. They came to farm, or, if they lived in cities this was in separate dwellings or cottages on sufficient an area of land to allow gardening – the congested urban slums of Montreal, New York or even Sydney never existed in New Zealand. Dr Newman (1882) was also right in pointing out that the temperate climate of much of the country allowed crop production all year. Before refrigeration in 1882 opened up meat exporting to the United Kingdom market, a lack of an outlet for sheepmeat meant there was a super-abundance of mutton on the domestic market, and thus a meat protein rich diet (Pool 1982, 1985).

As was shown earlier in this paper, life expectation was already high by the 1870s. Pakeha women arguably were the first population in history to experience 55 year life-expectation at birth. In the 1870s levels were as shown in Table 10.

**Table 10: Life expectation at birth, first Pakeha life tables**

| | 1874 | 1878 | 1881 |
|---|---|---|---|
| Males | 48.0 | 52.0 | 52.9 |
| Females | 49.9 | 55.6 | 56.4 |

In common with North America and Australia, New Zealand’s fertility levels were very high in the 1870s. Crude birth rates exceeded 40 per 1,000 and Gibson (1972) estimated that the total fertility rate may have been around 6.7 births per woman. Marital age specific rates were exceptionally high (over 500/1,000 at 15-19 years, 463 at 20-24 years) and marriage was not only almost universal (81 per cent of women

aged 25-29 were currently married), but was also early (Sceats and Pool 1985b). Fertility rates then changed dramatically (Gibson 1972: Chapt.VII).

*Changes in Life-Expectation and Co-Varying Factors*

Between 1876 and 1901, both crude birth rates (directly computed) and other fertility rates (estimates) changed very significantly. The crude birth rate declined from 41.7 per 1,000 in 1876, to 23.3 in 1901, from levels up with Australia to lower than Sweden's (26 per 1,000) (Khawaja 1985: Table 73; Sceats and Pool 1985b: 179); from among the highest levels in the industrialised countries to one of the lowest (Gibson 1972: Chapt.VII). The proximate determinants of fertility decline were changes in patterns of nuptiality. By 1901, Pakeha women had adopted the British pattern of late marriage and significant levels of spinsterhood. It has been estimated that 64 per cent of the change from 1876 to 1901 came from these shifts. Modern contraceptive technology, even condoms, did not really exist at that time, while induced abortion and ex-nuptial levels fertility levels appear to have been low (Sceats and Pool 1985b: 183-86).

The more causally remote determinants of the fertility decline seem to be threefold. The first factor was the low level of mortality by comparison with other populations, and above all the relatively high level of childhood survivorship relative to other societies at that time (cf. $e_0$ Norway and $e_0$ Pakeha in Table 7). As noted earlier, however, this did not carry through to ages 40 plus years (again see Table 7). A second factor was the high level of economic development. In this context, it is worth noting that the fertility decline spread northwards and outwards from the most developed sheep and grain farming regions and urban areas, to the newly developing dairying regions, and finally to the pioneering zones of the inland North Island (Pool and Tiong 1991). The third factor was the reinforcement of economic change by the policy environment and structures put in place at the end of the 19$^{th}$ century.

The link of reproduction to mortality is twofold. Firstly, as noted earlier lower fertility was linked to rapid improvements in survival at infancy and childhood, the causal links being through acute infective causes (infections, respiratory and diarrhoeal) and accidents. Secondly, as the physiological burden of childbearing decreased, so too was there a sex-crossover in mortality, and rates for females at reproductive ages declined for a wide range of causes.

A second set of co-variates of mortality relate to standards of living. As noted earlier Gross Domestic Product was already high by the 1870s:

> "The late nineteenth-century economy produced only a modest expansion in real income per head, but... it started from a very high level relative to other countries, and maintained a high level relative to other countries, and maintained a high level while population expanded from one-quarter of a million to one million" (Hawke 1985: 77).

Moreover the new pastoral export economy, dependent on refrigeration and on the British market, had a favourable impact on the balance of trade. It was "mostly negative before 1886 and mostly positive in later years" (Hawke 1985: 73). Beyond this, within the Pakeha population there was a high degree of social equality, and particularly the absence of a large population of urban poor (Gibson 1972).

In the 1890s the Seddon government provided the policy environment to reinforce these advantages. They improved frameworks for the redistribution of income including through industrial legislation and by land reform through splitting up large estates, they introduced various benefits, they extended the power of the state into a range of market activities (eg. banking and insurance), they encouraged cooperatives in the primary sector, and they developed policy and planning structures in service delivery sectors such as health (Sinclair 1959: Part II, Chapt II; Hawke 1985: Chapts. 4 & 5; Pool 1982).

What this section of the paper has described then, is a population that was favoured

already in the 1870s. But from the 1870s to 1900 the economic and social policy environment reinforced that advantage. Perhaps more importantly, a rapid fertility decline occurred and was closely related to the mortality changes.

**Conclusion**

This case-study has touched on both demographic transitions in general, and epidemiological transitions in particular. On the one hand the mortality history of Pakeha New Zealand in the late 19th and early 20th centuries could be seen simply as documenting another "exception" to epidemiological transition (Casselli, Meslé and Vallin 2002), or it could raise more fundamental theoretical questions. These seem to be three in number

Firstly, the most rapid changes occurred prior to 1900. As noted earlier, this contrasts with the normal case where improvements were radical after 1900. The pattern of acceleration followed by deceleration over the next 20-25 years suggests that, as a general rule, interpreting changes in life expectation and other indices from trends over a few decades may produce misleading conclusions. That is, even a strong trend in a particular direction may not presage longer-term trends. Moreover, cohort trends may imply a different perspective from synthetic.

Secondly, as Chesnais has clearly documented, demographic transitions normally follow a sequence: mortality decline and then fertility decline (1990: 327). This case-study on Pakeha seems to suggest a somewhat different pattern. The gaining of relatively low levels of mortality was followed by a rapid fertility decline, but it, in turn, seems to have effected further rapid mortality declines.

Finally, rapid mortality declines at infancy and childhood in the late 19th century appear to have been followed by changes at older ages as momentum effects moved up through the age structure increasing cohort in $p_x$ at sequential ages. This raises

another more general issue: once a change occurs at some ages this may produce an impetus that carries changes forward at subsequent ages. This is of significance in the sense that the older cohorts of today in most developed countries (but not Pakeha New Zealanders) benefited from rapidly improving survivorship at young ages (ie. between 1900 and 1930). The momentum may still be being felt today when these cohorts are at older ages. If the New Zealand model were to apply, then these rapid cohort gains will then be followed by rather smaller gains.

## REFERENCES


Belich, J. (1996) *Making Peoples*, Auckland: Allen Lane, The Penguin Press.

Belich, J. (2001) *Paradise Reforged: A History of the New Zealanders from the 1880s to the year 2000*, Auckland: Allen Lane, the Penguin Press.

Calot, G. and Sardon, J-P. (1999) “Les facteurs du vieillissement démographique”. *Population* 54(3) : 509-54.

Caselli, G. and Egidi, V. (1980) *Le differenze territoriali di mortalità in Italia*, Rome: Inst. Di demografia, Univ. di Roma.

Caselli, G. and Egidi, V. (1981) “Géographie de la mortalité en Europe :…”. *International Population Conference, Proceedings, Manila, 1981* Liège, IUSSP, vol 2 :165-202.

Casselli, F., Meslé, F. and Vallin, J. (2002) “Epidemiological Transition Theory Exceptions”, *Genus*, LVIII, 1:9-52.

Chesnais, J-P. (1990) “Demographic Transition Patterns and the Age Structure”, *Population and Development Review*, 16(2): 327-36.

Cheung, J. (1999) “Mortality, Morbidity and Population Health Dynamics”. Unpublished PhD Thesis, University of Waikato.

Cheung, J. (2001) “The Long-Term Trend of Non-Maori Mortality and its More Recent compression Effect”. Paper presented at Population Association of New Zealand bi-annual conference.

Crimmins, E., Saito, Y. and Ingegneri, D. (1989) “Changes in Life-Expectation and Disability in the United States”. *Population and Development Review*, 15(2): 235-67.

Donovan, J.W. (1969) “A Study in New Zealand Mortality 5: Tuberculosis”. *NZ Medical J*, 70(4): 94-103.

Fries J.F. (1980) "Aging, Natural Death, and the Compression of Morbidity". *New England Journal of Medicine*, 303:130-135.

Gibson, C. (1972) “Demographic History of New Zealand”. Unpublished PhD, Univ. of Calif, Berkeley (University Microfilms).

Hawke, G. (1985) *The Making of New Zealand*. Cambridge: Cambridge University

Press.

Kannisto, V. (1994) *Development of Oldest-Old Mortality: Evidence from 28 Developed Countries*, Odense Monographs on Population Ageing, Odence Univ. Press.

Khawaja, M. (1985) “Trends and Differentials in Fertility”. In ESCAP, Population Div. (eds) *Population of New Zealand: Country Monograph Series No 12*, 2 vols, NY, United Nations: Vol 1, 152-177.

Lancaster, H.O. (1952 a and b) “The Mortality in Australia from Acute Infective Diseases”. *Medical J. of Aust*, 1(6):451-70 and 2(8):272-76.

MacLean, F.S. (1963) *Challenge for Health*, Wellington: NZ Government Printer.

Manton K.G. (1982) "Changing Concepts of Mortality and Morbidity in the Elderly Population". *Milbank Memorial Fund Quarterly / Health and Society*, 60:183-244.

Newman, A.K. (1881) “A Study of the Causes Leading to the Extinction of the Maori”. *Transactions and Proceedings NZ Institute*, 14:59-77.

Newman, A.K. (1882) “Is New Zealand a Healthy Country? – An Enquiry”. *Transactions and Proceedings NZ Institute*, 15:493-510.

Norway (1969) *Historical Statistics*, Oslo: Central Bureau of Statistics.

Olshansky S.J. Carnes, B.A.and Cassel, C.K. (1993) "The Aging of the Human Species". *Scientific American*, 268 (4):18-24.

Oeppen, J. and Vaupel, J.W. (2002) “Broken Limits to Life Expectancy”. *Science*, 296, 10 May: 1029-31.

Papps, T. (1985) “Growth and Distribution of Population: Historical Trends”. In ESCAP *op cit.*: vol. 1, 10-29.

Pool, I. (1977) *The Maori Population of New Zealand*. Auckland: Auckland University Press.

_____ (1982) “Is New Zealand a Healthy Country?”. *NZ Population Review*, 8(2):2-27.

_____ (1983) “Changing Patterns of Sex Differential in Survival:…”. In Lopez, A.D. & Ruzicka, L.T. (eds) *Sex Differentials in Mortality*, Canberra: Dept of Demography, Australian National University, 193-220.

_____ (1985) “Mortality Trends and Differentials:. In ESCAP *op cit.*: vol. 1, 209-42.

_____ (1991) *Te Iwi Maori. A New Zealand Population Past, Present and Projected*. Auckland: Auckland University Press.

_____ (1994) “Cross-Comparative Perspectives on New Zealand's Health”. In Spicer, J., Trlin, A. & Walton, J.A. (Eds.), *Social Dimensions of Health and Disease*. Palmerston North: The Dunmore Press Ltd, 16-50.

_____ (2002) “Transfers of Capital and Shifts in New Zealand's Regional Population Distribution, 1840-1996”. *Population Studies Centre Discussion Paper No. 42*. Hamilton: University of Waikato.

Pool, I. and Bedford, R. (1997) “Population Change: From Dynamics and Structures to Policies”. *Background Paper, Plenary Session, the Population Conference, Wellington, 12-14 November*, Hamilton: Population Studies Centre, University of Waikato.

Pool, I., & Tiong, F. (1991) “Sub-National Differentials in the Pakeha Fertility Decline, 1876-1901”. *NZ Population Review*, 17: 46-64.

Preston, S., Keyfitz, N. and Schoen, R. (1972) *Causes of Death: Life-Tables for National Populations*, NY: Seminar Press.

Robine, J. M. (2001) “Redéfinir les phases de la transition épidémiologique à travers l'étude de la dispersion des durées de la vie: le cas de la France”. *Population*, 56, 199-223.

Robine J.M. and Romieu, I. (1998) "Healthy Active Ageing: Health Expectancies at Age 65 in the Different Parts of the World". *REVES* paper 318, May 1998.

Sceats, J. and Pool, I. (1985a) “Perinatal and Infant Mortality”. In ESCAP *op cit*.: vol. 1, 243-68.

Sceats, J. and Pool, I. (1985b) “Fertility Regulation”. In ESCAP *op cit*.: vol. 1, 178-92.

Sinclair, K. (1959) *A History of New Zealand*, Harmondsworth: Penguin.

Stolnitz, G. (1955) “A Century of International Mortality Trends Part I”. *Population Studies*, 9 July, 24-55.

Stolnitz, G. (1955) “A Century of International Mortality Trends Part II”. *Population Studies*, 10 July, 17-42.

Tuljapurkar, S., Li, N. and Boe, C. (2000) “A Universal Pattern of Mortality Decline in the G7 Countries”. *Nature*, 405, 15 June, 789-92.

United Nations Population Division (1953) *Determinants and Consequences of Population Trends*, NY: Dept of Social Affairs, United Nations.

United Nations Population Division (1973) *Revision: Determinants and Consequences of Population Trends*, NY: Dept of Social Affairs, United Nations.

Waldron, I. (1982) “An Analysis of Causes of Sex Differences in Mortality and Morbidity”. In Gove, W.R. and Carpenter, G.R. (eds) T*he Fundamental Connection Between Nature and Nurture*, Lexington, Mass: Lexington Books, 69-116.

Williams, D. (1999) ‘*Te Kooti Tango Whenua’: The Native Land Court, 1864-1909*, Wellington: Huia.