How low can it go? Projecting ischaemic heart disease mortality in New Zealand to 2015

Martin Tobias, Kerry Sexton, Stewart Mann, Norman Sharpe

Abstract

Aims This study aims to identify how ischaemic heart disease (IHD) mortality rates in New Zealand have varied between successive cohorts and time periods. This information is then used to project IHD mortality rates and counts (burdens) out to year 2011–15.

Methods Age / period / cohort models were constructed (5-year periods and 5-year age groups, generating 10-year overlapping cohorts) using both frequentist and Bayesian methods. Data were available from 1956 for the total population and from 1981 for Māori. The projection period was 2001-5 to 2011-15. Uncertainty was quantified as the Bayesian 90% credible interval.

Results IHD mortality rates for all age by gender groups increased from 1956–60 to peak in 1966–70, then declined by more than 60% to current (1996–2000) levels. However, the decline has been much shallower for Māori.

This decline has resulted from increasingly favourable period effects since 1971-75 (less marked for Māori). However, no substantive cohort effects have been seen, at least from the 1891 to the 1951 cohort. Our model suggests that, for the first time, a substantive and unfavourable cohort effect may be emerging among recent birth cohorts.

Conclusions IHD mortality rates are projected to continue to fall from 2001-05 to 2011-15, albeit more slowly than in the past as the increasing (favourable) period effect is partly offset by an emerging (unfavourable) cohort effect. The result is a relatively small projected decline in absolute IHD mortality burden overall, but an actual increase among Māori.

Ischaemic heart disease (IHD, coronary heart disease) mortality has been falling steadily in New Zealand for at least the past three decades yet this disease still accounts for almost one-quarter of all deaths. Also, large ethnic inequalities continue to exist, with Māori IHD mortality rates at least twice those of non-Māori.

Projections of future trends in IHD mortality (overall and by ethnicity) are therefore of continuing interest to national health policy advisors and funders, District Health Board planners, and health service providers.

The aim of this study is to identify how IHD mortality rates have varied in the past between successive birth cohorts and time periods, and to use this analysis of cohort and period effects to project these rates (and counts) into the future.

Methods

Data sources—Unit record mortality data from 1956 to 2000 was made available by the New Zealand Health Information Service. To minimise diagnostic and coding bias over the study period, a broad
definition of IHD was used, corresponding to ICD-9 codes 410–414. These codes may still omit IHD deaths attributed to codes for ‘sudden death’, ‘heart failure’, ‘cardiovascular disease not otherwise specified’ and ‘(type 2) diabetes mellitus’. All deaths so coded over the study period were assessed using multiple cause of death coding, and (where possible) were also linked to hospital discharges (for mention of IHD). This process yielded relatively few additional IHD deaths, however (approximately an additional 10%).

Māori IHD deaths were adjusted for undercounting of Māori ethnicity on death certificates using the New Zealand Census – Mortality Study (NZCMS) adjustors. These adjustors were derived by linking census to mortality records for the 3 years following each census from 1981 onwards. So for the ethnic (Māori – non-Māori) analysis, the study was restricted to 1981–2000 (rather than 1956–2000).

Midyear population estimates for 1956–2000 and projections (series 4) for 2001–2015 were obtained from Statistics New Zealand. Māori populations were interpolated intercensally, with the 1996 censal population being re-estimated from the 1991 and 2001 censal populations (as the ethnicity item varied in 1996 from earlier and later censuses).

Rates were age standardised for summarisation by the direct method, with the World Health Organization (WHO) World population as the standard.

Age/period/cohort (APC) modelling—For readers unfamiliar with APC modelling, the following brief explanation is provided. Mortality rates can be thought of as realisations of three dimensions of time: age (at death), period (calendar year of death) and cohort (year of birth). Age, period and cohort are proxies for the real drivers of IHD mortality (e.g. ‘age’ captures the cumulative process of atherosclerosis over the life course, ‘period’ captures developments in treatment and prevention, and ‘cohort’ captures risk exposures related to birth cohorts such as tobacco use or diet). Given a sufficiently long time series of IHD mortality data, regression models can be constructed to project rates based on the historical trends in age, period and cohort effects.

APC models were fit to the available data, using 5-year age groups and 5-year calendar periods, so defining 10-year overlapping birth cohorts, using the statistical package S Plus. However, the entire dataset could not be modelled because of poor fit. Instead, the data had to be truncated to the 35–74 age range to obtain good fits, omitting the substantial proportion of IHD deaths that occur in very old age. Both classical (frequentist) and Bayesian models were constructed. For the frequentist models, the assumption was made that the underlying risk of IHD mortality increases exponentially with age, allowing period-cohort models (rather than full APC models) with identifiable (i.e. unique) period and cohort effects to be fit. Projections were then obtained by linear regression of future period and cohort effects on the most recent three observed effects.

For the Bayesian models, the ‘random walk 2’ full APC models were fit. These models have unidentifiable effects but identifiable projections, and were used:

- To test the validity of the linearity assumption used in the frequentist projections; and
- To quantify uncertainty around the projected rates (presented here as the 90% credible interval, the Bayesian equivalent of the frequentist confidence interval).

Ex-post tests were carried out by fitting both sets of models to a reduced dataset that omitted the most recent observed period (1996–2000). Mortality for this period was then projected and the projections compared to the observed values.

Results

Descriptive

Period—For the total population, rates for almost all age by sex groups increased from 1956–61 to peak in 1966–70 and then declined steadily to 1996–2000 (Figure 1). Rates are now well below those seen at the beginning of the study period, having fallen on average about 60% from the peak in 1966–70 (slightly more for females and younger age groups). This corresponds to an average annual percentage change of approximately –3.5% over the observation period.
Figure 1. Ischaemic heart disease (IHD) mortality rates by period (1956–2000) and age (35–54), total population

<table>
<thead>
<tr>
<th>Age-specific rate per 100,000</th>
<th>Males</th>
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Note: Due to differences of scale, only rates for younger age groups are shown as these are of most interest (rates for older age groups are similar in pattern, though higher in level, to those for the 50–54 age group; these rates are available from the authors).

For Māori, age-specific rates have declined more slowly over the 1981–2000 period (both sexes). Only in the older age groups have substantial falls been seen, with rates in younger age groups declining relatively little over the observation period (data not shown).

Cohort—For the total population, each cohort experienced lower IHD mortality rates at corresponding ages than preceding cohorts (Figure 2).

For Māori, the pattern was essentially similar (for the included cohorts), although with less change in the age specific rates of successive cohorts (data not shown).
Figure 2. IHD mortality rates by cohort (1891–1961) and age (35–74), total population

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<th>Cohort mid-point</th>
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Note: Log scale is used to allow all ages to be represented (focus is on parallelism of the age specific curves).

Modelling

Period effects—Period effects derived from the frequentist model are presented in Figure 3. The effects are expressed as relative risks ie an effect <1 is protective (lowers the IHD mortality rate) while an effect >1 is adverse (increases the IHD mortality rate).

For the total population, period effects become progressively less adverse from the 1971–75 period onwards and by the most recently observed period are protective and large This steady trend in the period effect over three or more decades provides support for our assumption of linear projection over the 2001–2015 period.

For Māori, there has also been an improving (ie downward) trend in period effects from 1981–85 onwards, although the trend is shallower than that for non-Māori, especially among males. The period effect reaches 1 in the most recent observed period (1996–2000) or shortly before this, and is thereafter projected to become protective, more so for females than males.
Figure 3. Period effects, 1961–2015 (projected), ages 35–74

Males:

![Graph showing period effects for males, with data points for 1961-1965 to 2006-2010 for total population and Māori.]

Females:

![Graph showing period effects for females, with data points for 1961-1965 to 2006-2010 for total population and Māori.]

Figure 4. Cohort effects, 1891 to 1976 (projected), ages 35–74

Males:

Cohort effects—Cohort effects derived from the frequentist model are presented in Figure 4. The effects are again expressed as relative risks ie an effect <1 is protective while an effect >1 is adverse.
For the total population (both sexes), the striking finding is the absence of any strong cohort effects (at least from the 1891 cohort onwards). Nevertheless, there are interesting albeit minor variations in cohort effects to be seen. From the 1891 cohort (females) or 1896 cohort (males), the cohort effects become increasingly adverse (greater than 1), although still small, up to the 1916 cohort, whereafter the trend reverses and the cohort effect becomes increasingly protective (although still small) up to the 1946 cohort.

The most recently observed cohorts (i.e. the 1951, 1956 and 1961 cohorts) reverse the trend once more, and become increasingly adverse—although only the 1956 and 1961 effects (males) or 1961 effect (females) actually exceed 1.

Although the recent trend is far less clear than that for the period effects (see Figure 3), linear projection over recent cohorts produces a substantive adverse cohort trend, at least for the next few cohorts (both sexes). Thus our projection indicates the possible emergence, for the first time ever, of a substantive (and adverse) cohort effect (as shown by the dotted line in Figure 4).

For Māori, the pattern is essentially similar, with no strong cohort effects being detected. Nevertheless, the projection is for an upward trend in cohort effects over the next fifteen years, although this is of much smaller magnitude for males than females.

**Projections**—Projections were done using both the frequentist model (which required the assumption that recent trends in period and cohort effects would continue linearly into the future) and the Bayesian model (which required no such assumption). In fact, both models gave almost identical projections across all age by sex by ethnicity groups, supporting the linearity assumption (data available from the authors). Further validation of both models was provided by the ex post test: using the reduced dataset, both models produced near identical projections for the 1996–2000 period, which agreed closely with the observed rates (data available from the authors). Only the Bayesian projections are reported here.

For the total population (both sexes), and for both ethnic groups (Māori and non-Māori), age-specific and age-standardised IHD mortality rates are projected to continue to decrease until 2015. However, the rate of decrease will progressively slow (Figure 5). This reflects the interaction of increasingly protective period effects with increasingly adverse cohort effects.

For both total and ethnic populations, age standardised within the 35–74 age range, IHD mortality rates are projected to continue their long-term downward trend for both sexes. It should be noted that the 90% credible interval is wide for Māori (reflecting small numbers), but does not encompass an actual increase in rates.

**IHD burden**—Projections of the IHD burden were done by applying the projected age-specific IHD mortality rates to the projected population (within the 35–74 age range); see Figure 6.

For the total population (both sexes), the count of IHD deaths (within the age range 35–74) is projected to continue to decrease, albeit slightly more slowly than in previous decades. The projected average annualised count in 2001–05 is 1447 (males) and 507 (females), decreasing to 1103 and 345 in 2011–15 respectively (reductions of 24% and 32% respectively).
Figure 5A. Age-standardised (35–74) IHD mortality rates and projections, 1956–2015, by sex, total population
Figure 5B. Age-standardised (35–74) IHD mortality rates and projections, 1981–2015, by sex, Maori and non-Maori population

Males

Females
Figure 6A. Average annualised IHD mortality count, ages 35–74, by sex, total population, 1956–2015 (projected)

Figure 6B. Average annualised IHD mortality count, ages 35 – 74, by sex, Maori population, 1981 – 2015 (projected)
This lesser reduction in burden than risk of IHD mortality results because the declining risk is partially offset by increasing population size together with a small contribution from population ageing (within the 35–74 age range) as the large ‘baby boom’ cohorts reach late middle and early old age.

For Māori, the decline in IHD mortality risk is (relatively) smaller and the growth (and ageing) of the population is (relatively) greater. As a result, the number of Māori deaths (both sexes) is projected to actually increase over the next ten to fifteen years. By 2011–15, the average annualised count of IHD deaths among Māori is projected to reach approximately 560, a 15% increase from the 480 (average annualised) deaths estimated for 2000–05.

Discussion

Although the analysis had to be limited to the 35–74 age group, this study nevertheless provides new and valuable information. It confirms that the peak of the IHD epidemic occurred in New Zealand in the late 1960s, as was already known from earlier research. Since then, rates have fallen substantially (by approximately 60%) at all ages, although much less steeply for Māori. However, our models project that the rate at which IHD mortality declines in the next decade will progressively slow among both sexes and both major ethnic groups. To avoid this outcome, improvements in the coverage, quality, and effectiveness of prevention and treatment interventions will be required over and above those anticipated by projecting the historical trend.

This projected slower decline in IHD mortality risks, coupled with a growing and ageing population, leads us to forecast that the burden of IHD mortality (i.e. counts as opposed to rates) will decrease by only 25–30% (approximately) over the next decade. Indeed, the burden (and corresponding need for preventive and therapeutic coronary care services) is projected to increase for Māori. This finding has major policy implications, not least the need to urgently improve access for Māori to and through coronary care, if worsening of inequality in heart health between Māori and non-Māori is to be avoided.

Our projections relate only to the burden of IHD mortality. Trajectories for non-fatal burdens (including need for acute coronary care and management of people with heart failure) may be very different. Furthermore, trends in the 35–74 age group may differ from those in the 75+ age group.

Our study reveals an interesting pattern with regard to cohort effects. The absence of any strong cohort effects from the 1891 to the 1951 cohorts contradicts the hypothesis advanced by Barker (which states that the risk of IHD is largely predetermined in utero), at least at the population level. Under the fetal origins hypothesis, dramatic increases followed by decreases in cohort effects should have been detected—yet no strong cohort effects were found at all. This finding does not mean that the hypothesised relationship does not exist at the individual level, merely that it is unlikely to have had a substantive impact on the IHD epidemic at the population level.

Our model does, however, suggest the possible emergence of a rising cohort effect among those born since the early 1950s. If confirmed, this would provide an explanation for the projected slowing in the secular trend of IHD mortality over the
next 10 to 15 years. That is, recent cohorts are projected to experience higher underlying risks of IHD mortality than their preceding cohorts—so partially offsetting the benefits that would otherwise accrue to them from the projected continuing (protective) trend in period effects.

What might explain these projected trends in period and cohort effects? Continuing improvement in period effects is likely to reflect better coverage and quality of preventive and therapeutic interventions for IHD. Our model is unable to disaggregate the period effect into incidence reduction and case fatality reduction components. However, analysis of Auckland MONICA data suggests that approximately half may be attributable to downshifts in population risk factor distributions and half to more effective and accessible treatments (including secondary prevention and thrombolysis in particular).

The emergence of a substantive adverse cohort effect is harder to explain. Firstly, it may simply be an artefact of our frequentist model, specifically the linear regression of future cohort effects. However, the Bayesian model, which requires no such assumption, gave almost identical projections. Secondly, it could reflect changing proportions of different ethnic groups in the population. This explanation is unlikely as the same pattern is seen in the ethnic specific analyses (although less convincingly so for Māori males). Thirdly, it could reflect the emergence of a ‘core’ of people who are less responsive to health promotion messages such as not smoking cigarettes—although this would be expected to produce stabilisation of cohort effects rather than actual reversal of the prior trend.

A more likely explanation relates to the emergence, since the 1970s, of the epidemic of obesity (and consequential type 2 diabetes) in New Zealand and indeed throughout the developed world. In fact, similar slowing in IHD mortality declines has been observed recently in some other developed countries, and a rising cohort effect has been detected in both Australia and Sweden. If this explanation is confirmed, our study will have provided the first signal of an impact of the obesity epidemic on IHD mortality rates and burdens in New Zealand.

Regardless of the explanation, our projections for the next decade have clear implications for policy. At the very least, these projections imply that there is no room for complacency in regard to the prevention and treatment of IHD—especially if we are concerned about reducing inequalities in health between Māori and non-Māori.

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