Explanation for the Decline in Coronary Heart Disease Mortality Rates in Auckland, New Zealand, Between 1982 and 1993

Simon Capewell, MD; Robert Beaglehole, MD; Mary Seddon, MD; John McMurray, MD

Background—We sought to determine how much of the recent, substantial fall in coronary heart disease (CHD) mortality rates in New Zealand can be attributed to “evidence-based” medical and surgical treatments and how much can be attributed to cardiovascular risk factor reductions.

Methods and Results—A cell-based mortality model was developed and refined. This model combined (1) the published effectiveness of cardiological treatments and risk factor reductions with (2) data on all medical and surgical treatments administered to all CHD patients and (3) trends in population cardiovascular risk factors (principally smoking, cholesterol, and hypertension) from 1982 to 1993 in Auckland, New Zealand (population 996,000). Between 1982 and 1993, CHD mortality rates fell by 23.6%, with 671 fewer CHD deaths than expected from baseline mortality rates in 1982. Forty-six percent of this fall was attributed to treatments (acute myocardial infarction 12%, secondary prevention 12%, hypertension 7%, heart failure 6%, and angina 9%), and 54% was attributed to risk factor reductions (smoking 30%, cholesterol 12%, population blood pressure 8%, and other, unidentified factors 4%). These proportions remained relatively consistent after a robust sensitivity analysis.

Conclusions—Approximately half the CHD mortality rate fall in Auckland, New Zealand, was attributed to medical therapies, and approximately half was attributed to reductions in major risk factors. These findings emphasize the importance of a comprehensive strategy that maximizes the population coverage of effective treatments and actively promotes a prevention program, particularly for smoking, diet, and blood pressure reduction. (Circulation. 2000;102:1511-1516.)

Key Words: coronary disease ■ mortality ■ drugs ■ risk factors ■ population

Coronary heart disease (CHD) is an important cause of death, complications, and resource use throughout the industrial and developing worlds. CHD mortality rates have been falling in some, but not all, industrial countries since the 1970s. Explanations for this remain controversial. Many authors attribute most of the CHD mortality rate fall to reductions in major risk factors such as smoking, cholesterol, and blood pressure. Conversely, others point to the increasingly widespread use of effective therapies such as thrombolysis, aspirin, ACE inhibitors, and CABG. A better understanding is crucial for future CHD strategies and resource allocation.

We developed and refined a CHD mortality model in Scotland. This cell-based model uses published evidence of the effectiveness of treatments and risk factor reductions combined with local data on patient numbers to calculate the expected mortality rate reduction. In Scotland between 1975 and 1994, ≈40% of the CHD mortality rate fall was attributable to the cumulative effect of all treatments, whereas ≈50% of the fall was attributed to reductions in major risk factors. The remaining 9% was attributed to other, unmeasured factors; these may include intrauterine effects, dietary antioxidants, exercise, and increased obesity. The findings remained remarkably consistent in a robust sensitivity analysis. However, there clearly was a need to replicate the model with independent data from a distant country.

New Zealand has experienced a severe CHD epidemic, and underlying risk factors appear to be the same as in other countries. Although mortality rates have been falling since the late 1960s, this has received surprisingly little detailed analysis. In 1986, ≈40% of the mortality rate fall was attributed to treatments, and in 1990, ≈40% of the fall was attributed to risk factor declines. However, the 2 components have never been considered simultaneously. Moreover, there has been no analysis since the widespread introduction of modern cardiological treatments.
In the present study, we therefore sought to test the Scottish CHD mortality model by determining how well it might explain the observed changes in treatments, risk factors, and mortality rates in New Zealand between 1982 and 1993. A better understanding of the CHD mortality rate fall is clearly essential to form CHD strategies in New Zealand and to cast further light on CHD trends in comparable countries elsewhere.

### Methods

#### Setting

The study was conducted in the Central Auckland Statistical Area (1993 population 996,000, one third of the total New Zealand population) from 1982 to 1993.

#### Identification and Assessment of Relevant Data

Information on population, demographic changes, mortality rates, acute myocardial infarction incidence, and treatment was based on routine health statistics and data from the Auckland Region Coronary Or Stroke (ARCOS) Study, a World Health Organization MONICA project, with ICD-9 codes 410 to 414.11,12,13 Patients eligible for secondary prevention after acute myocardial infarction, CABG, and angioplasty were calculated with routine statistics.14 The proportions of patients treated with aspirin, β-blockers, ACE inhibitors, rehabilitation programs, statins, and other therapies were based on local audit studies.15,16 (B. Arroll, personal communication). Precise patient numbers were available for those treated with CABG or angioplasty and for those with unstable angina (M. Vedder, personal communication).

The number of patients with heart failure who were receiving ACE inhibitor treatment in the hospital and in the community was based on routine statistics14 and local and national surveys.15,16 Hypertension prevalence and treatment were also based on national surveys and on extensive ARCOS prevalence studies.16,17 Further validation of community treatment levels was provided by additional independent information from national prescription monitoring agencies.18,19

#### The Model

The Microsoft Excel cell-based mortality model has been described in detail elsewhere.9 In brief, 1982 was taken as the base year. The number of CHD deaths prevented or postponed in Auckland in 1982 and again in 1993 were calculated for specific interventions, such as thrombolysis, CABG, aspirin use, and so on. Each specific mortality rate reduction was derived from the relative and absolute mortality risk reduction.19

### Table 1.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>No. of Eligible Patients</th>
<th>Uptake of Intervention</th>
<th>Compliance</th>
<th>Absolute Risk Reduction</th>
<th>No. of Deaths Prevented or Postponed (minimum and maximum estimates)</th>
<th>Proportion of Total Mortality Reduction, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute myocardial infarction</td>
<td>1429</td>
<td></td>
<td></td>
<td></td>
<td>78 (28, 124)</td>
<td>12</td>
</tr>
<tr>
<td>Community CPR</td>
<td>377</td>
<td>0.5</td>
<td>1.00</td>
<td>0.180</td>
<td>21 (6, 39)</td>
<td>...</td>
</tr>
<tr>
<td>Hospital CPR</td>
<td>137</td>
<td>0.95</td>
<td>1.00</td>
<td>0.150</td>
<td>19 (8, 36)</td>
<td>...</td>
</tr>
<tr>
<td>Thrombolysis and aspirin</td>
<td>1106</td>
<td>0.41</td>
<td>1.00</td>
<td>0.052</td>
<td>24 (10, 32)</td>
<td>...</td>
</tr>
<tr>
<td>Aspirin alone</td>
<td>1106</td>
<td>0.29</td>
<td>1.00</td>
<td>0.024</td>
<td>8 (2, 10)</td>
<td>...</td>
</tr>
<tr>
<td>β-Blocker</td>
<td>1106</td>
<td>0.3</td>
<td>1.00</td>
<td>0.013</td>
<td>4 (2, 5)</td>
<td>...</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>1106</td>
<td>0.2</td>
<td>1.00</td>
<td>0.007</td>
<td>2 (1, 2)</td>
<td>...</td>
</tr>
<tr>
<td>Secondary prevention</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post–myocardial infarction</td>
<td>3749</td>
<td></td>
<td></td>
<td></td>
<td>46 (10, 156)</td>
<td>7</td>
</tr>
<tr>
<td>Aspirin alone</td>
<td>3749</td>
<td>0.35</td>
<td>0.7</td>
<td>0.007</td>
<td>6 (1, 17)</td>
<td>...</td>
</tr>
<tr>
<td>β-Blocker alone</td>
<td>3749</td>
<td>0.10</td>
<td>0.7</td>
<td>0.023</td>
<td>6 (1, 25)</td>
<td>...</td>
</tr>
<tr>
<td>Aspirin and β-blocker</td>
<td>3749</td>
<td>0.20</td>
<td>0.7</td>
<td>0.025</td>
<td>13 (4, 37)</td>
<td>...</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>3749</td>
<td>0.20</td>
<td>0.7</td>
<td>0.006</td>
<td>5 (2, 58)</td>
<td>...</td>
</tr>
<tr>
<td>Simvastatin</td>
<td>3749</td>
<td>0.20</td>
<td>0.7</td>
<td>0.006</td>
<td>3 (1, 10)</td>
<td>...</td>
</tr>
<tr>
<td>Warfarin</td>
<td>3749</td>
<td>0.05</td>
<td>0.85</td>
<td>0.010</td>
<td>2 (0, 6)</td>
<td>...</td>
</tr>
<tr>
<td>Rehabilitation including exercise</td>
<td>3749</td>
<td>0.19</td>
<td>0.7</td>
<td>0.020</td>
<td>11 (3, 35)</td>
<td>...</td>
</tr>
<tr>
<td>Post-CABG/PTCA</td>
<td>3099</td>
<td></td>
<td></td>
<td></td>
<td>37 (9, 115)</td>
<td>5</td>
</tr>
<tr>
<td>Angina</td>
<td>21 864</td>
<td></td>
<td></td>
<td></td>
<td>60 (24, 200)</td>
<td>9</td>
</tr>
<tr>
<td>CABG</td>
<td>2906</td>
<td>1.00</td>
<td>1.00</td>
<td>0.010</td>
<td>28 (18, 89)</td>
<td>...</td>
</tr>
<tr>
<td>PTCA</td>
<td>1713</td>
<td>1.00</td>
<td>1.00</td>
<td>0.002</td>
<td>3 (0, 8)</td>
<td>...</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>1743</td>
<td>1.00</td>
<td>1.00</td>
<td>0.005</td>
<td>9 (2, 17)</td>
<td>...</td>
</tr>
<tr>
<td>Aspirin in community</td>
<td>21 864</td>
<td>0.33</td>
<td>0.70</td>
<td>0.004</td>
<td>19 (4, 86)</td>
<td>...</td>
</tr>
<tr>
<td>Heart failure</td>
<td>6,983</td>
<td></td>
<td></td>
<td></td>
<td>42* (7, 147)</td>
<td>6</td>
</tr>
<tr>
<td>Hospital</td>
<td>778</td>
<td>0.35</td>
<td>1.00</td>
<td>0.125</td>
<td>33 (8, 104)</td>
<td>...</td>
</tr>
<tr>
<td>Community</td>
<td>6120</td>
<td>0.21</td>
<td>0.72</td>
<td>0.02</td>
<td>18 (1, 97)</td>
<td>...</td>
</tr>
<tr>
<td>Hypertension</td>
<td>57 994</td>
<td>0.63</td>
<td>0.48</td>
<td>0.003</td>
<td>47 (10, 155)</td>
<td>7</td>
</tr>
<tr>
<td>Total treatments</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>310 (101, 920)</td>
<td>46</td>
</tr>
</tbody>
</table>

*Adjusted for community/hospital overlap.
rate reductions reported in published randomized controlled trials and meta-analyses. Survival benefit over a minimum time interval of 1 year was calculated for all treatments and all patient groups in the hospital and in the community.

Treatment Combinations and Compliance
Where combination therapy was common, such as acute myocardial infarction and secondary prevention, cumulative benefit was estimated with the following formula: Relative Benefit = 1 - (1 - Treatment A) × (1 - Treatment B), and so on. Compliance, the proportion of treated patients actually taking therapeutically effective levels of medication, was assumed to be 100% in hospital patients, 70% in symptomatic community patients, and 50% in asymptomatic community patients.

Risk Factor Trends and Mortality Rate Benefits
Extensive high-quality, population-based risk factor data were available. Trends in the cohorts aged >65 years were calculated by extrapolation.

The CHD mortality rate reduction attributable to declines in specific risk factors was principally based on a regression method. This used the mean $\beta$-coefficients for smoking, cholesterol, and blood pressure derived from 8 pooled MONICA cohort studies in Finland, Iceland, and Australasia. The cholesterol effect was also calculated with a recent meta-analysis.

A second, independent method was used as validation, based on population-attributable risk (the mortality rate reduction expected with a given percentage fall in a specific risk factor).

Comparison With Observed Mortality Rate Falls
The model estimates for the total deaths prevented or postponed by all treatments plus all risk factor reductions were then compared with the observed falls in mortality rates for men and women in specific age groups.

As in Scotland, any shortfall in the overall model estimate was then formally attributed to other, unmeasured risk factors.

Sensitivity Analysis
Because of the uncertainties that surround some of the values, a multiway sensitivity analysis was performed (analysis of extremes method).

Worked examples are given in the Appendix.

Results
In 1993 in the Central Auckland Statistical Area, there were 1808 deaths from CHD. This represented an overall CHD mortality rate fall of 23.6%, 558 fewer deaths than expected, had there been no decline from 1982 mortality rates.

Medical and Surgical Treatments
Specific medical and surgical treatments were estimated to prevent or postpone 310 deaths (minimum estimate 101, maximum 920, Table 1).

Major Cardiovascular Risk Factors
Declines in the major cardiovascular risk factors together produced a best estimate of 361 fewer deaths (minimum estimate 204, maximum 596). The majority were attributable to reductions in smoking prevalence and to the secular fall in population blood pressure. The decline specifically attributable to a fall in cholesterol levels was 79, or 99 with the recent meta-analysis.

As planned, the model shortfall of 28 was attributed to other, unmeasured factors. (Table 2).

Mortality Rate Reduction 1982 to 1993
The number of deaths prevented or postponed with all medical and surgical treatments plus all of the reductions in risk factors therefore totaled 671 (310+361). In 1982, an estimated 113 deaths were prevented or postponed with all medical and surgical treatments. The estimated reduction in CHD mortality rate between 1982 and 1993 was therefore 558 deaths (671-113). Comparison with the actual mortality rate reduction observed over that period showed good agreement overall (Table 3).

With the application of sensitivity analysis, the rankings and proportional contributions to the total mortality rate reduction by specific interventions remained relatively consistent across a wide range of assumptions and values (Figure 1). The best estimates suggested that ∼46% of the mortality rate reduction was attributable to treatments (acute myocardial-
dial infarction 12%, secondary prevention 12%, angina 9%, heart failure 6%, hypertension 7%) and 54% was attributable to reductions in population risk factors (smoking 30%, cholesterol 12%, population blood pressure reduction 8%). As planned, the 4% shortfall in the model estimate was attributed to other, unmeasured factors.

**Discussion**

Medical treatments and risk factor changes together prevented or postponed 670 CHD deaths in Auckland in 1993 compared with 1982. This estimate was consistent with the observed decline in deaths and with treatment effects in 1982. Medical treatments therefore have a substantial impact on CHD mortality rates, particularly secondary prevention and the initial treatment of myocardial infarction, heart failure, and hypertension.5,8,9,16,20–25 Although CABG and angioplasty may improve symptoms in individual patients, revascularization has a small overall contribution to mortality rate, reflecting the relatively few patients and relatively modest treatment effect.9,36,37

We previously highlighted low treatment uptakes. Appropriate therapy at adequate doses for the majority of Scottish patients would result in ~4000 fewer deaths each year.38 This is equally true in New Zealand and, we suggest, the United States, where, by extrapolation, an additional 100 000 deaths might be prevented or postponed.

The present study provided useful replication of the Scottish CHD mortality model9 (Table 4). It appears reasonable to credit medical and surgical treatments for up to half the CHD mortality rate fall in Scotland (40%, 1975 to 1994) and in New Zealand (Table 4). It appears reasonable to credit medical and surgical treatments for up to half the CHD mortality rate fall in Scotland (40%, 1975 to 1994) and in New Zealand

**TABLE 3. Comparison of Estimated and Observed CHD Mortality Falls (1982–1993) by Age and Sex**

<table>
<thead>
<tr>
<th>Deaths Prevented or Postponed</th>
<th>Men, y</th>
<th>Women, y</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>&lt;65</td>
</tr>
<tr>
<td>Total treatment effects in 1993</td>
<td>310</td>
<td>67</td>
</tr>
<tr>
<td>Total risk factor effects in 1982–1993</td>
<td>361</td>
<td>113</td>
</tr>
<tr>
<td>Combined treatment and risk factor effects, n</td>
<td>671</td>
<td>180</td>
</tr>
<tr>
<td>(% contribution)</td>
<td>(27)</td>
<td>(21)</td>
</tr>
<tr>
<td>Minus treatment effects in 1982, n</td>
<td>113</td>
<td>44</td>
</tr>
<tr>
<td>Estimated mortality reduction 1982–1993, n</td>
<td>558</td>
<td>136</td>
</tr>
<tr>
<td>(% contribution)</td>
<td>(24)</td>
<td>(21)</td>
</tr>
<tr>
<td>Observed mortality reduction 1982–1993, n</td>
<td>558</td>
<td>115</td>
</tr>
<tr>
<td>(% contribution)</td>
<td>(21)</td>
<td>(25)</td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th>Scotland</th>
<th>New Zealand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment, %</td>
<td>40</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>10</td>
</tr>
<tr>
<td>Secondary prevention</td>
<td>8</td>
</tr>
<tr>
<td>Heart Failure</td>
<td>8</td>
</tr>
<tr>
<td>Aspirin for angina</td>
<td>2</td>
</tr>
<tr>
<td>CABG surgery</td>
<td>2</td>
</tr>
<tr>
<td>Hypertension treatment</td>
<td>9</td>
</tr>
<tr>
<td>Risk factor reduction, %</td>
<td>60</td>
</tr>
<tr>
<td>Population blood pressure*</td>
<td>15</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>6</td>
</tr>
<tr>
<td>Deprivation</td>
<td>3</td>
</tr>
<tr>
<td>Other factors</td>
<td>9</td>
</tr>
<tr>
<td>Smoking</td>
<td>36</td>
</tr>
</tbody>
</table>

*Population blood pressure includes effect of treatment of individuals for hypertension.
(46%, 1982 to 1993). This is consistent with the few comparable studies in other countries. Unsurprisingly, this treatment contribution tended to be a little lower in earlier studies\(^3\) and higher in the 1990s, with the more widespread use of effective therapies.\(^7\)-\(^9\),\(^3\)\(^9\)-\(^4\) In Finland, only 30% of the 1972-to-1992 mortality rate fall was attributable to treatments.\(^3\) However, the Finnish comprehensive national prevention program achieved particularly large risk factor reductions.\(^3\)

Half of the Auckland CHD mortality rate fall was apparently attributable to reductions in major risk factors, particularly smoking. This is a substantial achievement for health promotion. However, further reductions in smoking, cholesterol, and blood pressure would still be beneficial.\(^3\)\(^6\)

Surprisingly, declines in “other,” unmeasured risk factors apparently accounted for only 4% of the mortality rate fall in Auckland. The available \(\beta\)-coefficients may overestimate the effect of major risk factors. Opposing trends in other risk factors may also have tended to cancel out each other, such as increases in obesity, exercise levels, and dietary intake of antioxidants.\(^3\)\(^6\)

The model estimates with the regression and population-attributable risk method were reassuringly consistent for smoking, but, as in Scotland, the population-attributable risk method produced substantially lower estimates than the regression method for cholesterol and blood pressure.\(^9\)\(^3\)\(^3\) The existing coefficients may not be truly independent and may overlap with treatment effects. Methodological development work is clearly needed.\(^2\),\(^1\),\(^2\),\(^1\),\(^7\),\(^3\),\(^0\),\(^3\),\(^2\)

Studies such as this have a number of limitations. Few local CHD data are generally available on recent risk factor trends and treatments. Assumptions therefore must be made to fill the gaps, and a sensitivity analysis becomes essential.\(^3\)\(^5\) However, even when extreme minimum and maximum values are used, the ranking and the proportional contributions to the overall mortality rate reduction changed very little (Figure 1). Furthermore, the individual best estimates produced a total mortality rate reduction consistent with the fall actually observed. This analysis focused on mortality, not incidence and neglected symptomatic relief, a major modern therapy goal.\(^6\)-\(^9\) The model also assumed that efficacy in randomized trials can be generalized to effectiveness in clinical practice. Effects may vary with the level of risk.\(^3\)\(^7\)

In conclusion, up to half of the recent large falls in CHD mortality rates in New Zealand, Scotland, the United States, and elsewhere may be attributable to medical therapies and half may be attributable to reductions in major risk factors. This emphasizes the importance of primary prevention strategies, particularly for diet and smoking, and secondary prevention strategies, particularly those that maximize treatment uptake.

### Appendix

#### Calculation Examples

**An Example of the Estimation of CHD Mortality Rate Reduction Attributable to Declines in Specific Risk Factors: Smoking in Men Aged 45 to 64 Years**

Dobson et al\(^1\) recently pooled data from 8 MONICA populations to generate regression values. For smoking in men aged 45 to 64, they derived a mean \(\beta\)-coefficient of 0.4; that is, a 0.4% relative decline in mortality rates was attributable to a 1% relative fall in population smoking rates.

In Auckland between 1982 and 1993, smoking prevalence in men aged 45 to 64 years fell from 28.6% to 16.9%, a relative decline of 40.8%.

The CHD deaths prevented or postponed were therefore calculated as

\[
\text{CHD deaths prevented or postponed} = (\text{Patient number}) \times (\text{treatment uptake}) \times (\text{compliance}) \times \text{(absolute mortality rate reduction)}
\]

Best estimate

\[
573 \times 53\% \times 0.052 = 15.79 \text{ deaths prevented or postponed}
\]

Minimum estimate

\[
544 \times 42\% \times 0.04 = 9.36 \text{ deaths prevented or postponed}
\]

Maximum estimate

\[
602 \times 64\% \times 0.069 = 26.58 \text{ deaths prevented or postponed}
\]

This sensitivity analysis was then repeated for each specific treatment and risk factor reduction.

**Acknowledgments**

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References
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