Explaining the Decline in Coronary Heart Disease Mortality in England and Wales Between 1981 and 2000

Belgin Unal, MD, MPH; Julia Alison Critchley, DPhil; Simon Capewell, MD

Background—Coronary heart disease mortality rates have been decreasing in the United Kingdom since the 1970s. Our study aimed to examine how much of the decrease in England and Wales between 1981 and 2000 could be attributed to medical and surgical treatments and how much to changes in cardiovascular risk factors.

Methods and Results—The IMPACT mortality model was used to combine and analyze data on uptake and effectiveness of cardiological treatments and risk factor trends in England and Wales. The main data sources were published trials and meta-analyses, official statistics, clinical audits, and national surveys. Between 1981 and 2000, coronary heart disease mortality rates in England and Wales decreased by 62% in men and 45% in women 25 to 84 years old. This resulted in 68 230 fewer deaths in 2000. Some 42% of this decrease was attributed to treatments in individuals (including 11% to secondary prevention, 13% to heart failure treatments, 8% to initial treatments of acute myocardial infarction, and 3% to hypertension treatments) and 58% to population risk factor reductions (principally smoking, 48%; blood pressure, 9.5%; and cholesterol, 9.5%). Adverse trends were seen for physical activity, obesity and diabetes.

Conclusions—More than half the coronary heart disease mortality decrease in Britain between 1981 and 2000 was attributable to reductions in major risk factors, principally smoking. This emphasizes the importance of a comprehensive strategy that promotes primary prevention, particularly for tobacco and diet, and that maximizes population coverage of effective treatments, especially for secondary prevention and heart failure. These findings may be cautiously generalizable to the United States and other developed countries. (Circulation. 2004;109:1101-1107.)

Key Words: coronary disease ■ mortality ■ modeling ■ risk factors ■ treatment

Coronary heart disease (CHD) remains the most common cause of death in the United States and the United Kingdom.1-4 However, CHD mortality rates have decreased by 50% in most industrialized countries since the 1970s. In the United States, the decline was steeper in the 1980s and then flattened slightly in the last decade. However, in the United Kingdom, the decline has been slower, and CHD mortality rates are still higher than in the United States (Figure 1).

Explanations for the mortality decreases remain controversial.5 Many authors credit the increasingly widespread use of effective therapies such as thrombolysis, aspirin, ACE inhibitors, statins, and coronary artery bypass surgery.5,6 Others highlight reductions in major cardiovascular risk factors such as cholesterol, smoking, and blood pressure.3,7 Although both components are probably important, answering this complex question seems difficult.

Some researchers have therefore used models of various degrees of sophistication to try to explain the observed declines in CHD mortality.8 The majority consistently suggest that risk factor improvements explain more of the mortality decline than do treatments. For example, it has been estimated that the proportion of mortality decline attributable to risk factor reductions was 57% in the United States between 1980 and 1990; 60% in Auckland, New Zealand, between 1974 and 1981 and 52% between 1982 and 1993; 60% in Scotland between 1975 and 1994.2 Since then, however, many effective therapies have been introduced.13 A better understanding of the CHD mortality decrease in Britain, the United States, and other countries is clearly essential both to predict future trends and to clarify policy options for CHD prevention.13,14 We have therefore examined how much of the CHD mortality decrease in England and Wales between 1981 and 2000 can be attributed to “evidence-based” medical and surgical treatments and how much to changes in major cardiovascular risk factors.

Methods

The cell-based IMPACT mortality model, previously validated in Scotland and New Zealand,12 was further developed and refined. We identified and incorporated data for men and women 25 to 84 years old in England and Wales, detailing (1) CHD patient numbers, (2) uptake of specific medical and surgical treatments, (3) population trends in major cardiovascular risk factors (smoking, total cholesterol, hypertension, obesity, diabetes, physical activity, and socioeco-
omic deprivation), (4) effectiveness of specific cardiological treatments, and (5) effectiveness of specific risk factor reductions.

Identification and Assessment of Relevant Data
Information on population, demographic changes, mortality, and myocardial infarction incidence was obtained principally from routine health statistics from the Office for National Statistics and the British Heart Foundation’s Annual CHD Statistics. The number of patients admitted to hospital with myocardial infarction, angina, and heart failure was obtained from Hospital Episode Statistics. Patients undergoing cardiopulmonary resuscitation in the community or in hospital were enumerated from various surveys. Information on patients undergoing CABG surgery and angioplasty came from the United Kingdom Cardiac Surgical Register and the British Cardiovascular Intervention Society’s audit returns, respectively. Surviving patients eligible for secondary prevention therapies after myocardial infarction, CABG surgery, or angioplasty were calculated by use of routine statistics and revascularization registers.

The number of patients in the community with treated or untreated hypertension or angina was calculated using the 1998 Health Survey for England and the British Regional Heart Study. The number of treated and untreated heart failure patients in the community was obtained from General Practice returns and survey data.

Information on treatment prescription and uptake was obtained from various national and local clinical audits and surveys. Population risk factor trend data were obtained primarily from the British Regional Heart Study, the General Household Survey, and the Health Survey for England.

Data on the efficacy of therapeutic interventions and the mortality reduction from specific population cardiovascular risk factor changes were obtained from published randomized controlled trials, meta-analyses, and cohort studies. Full details of data sources are provided in Appendices 1 through 4 (see the online-only Data Supplement).

Decrease in CHD Deaths
The number of CHD deaths expected in 2000 if the mortality rates in 1981 had persisted was calculated by indirect age standardization, using 1981 as a base year. The CHD deaths actually observed in 2000 were then subtracted to provide the decrease in CHD deaths between 1981 and 2000.

IMPACT Model
This Microsoft Excel cell-based mortality model has been described in detail elsewhere. The numbers of CHD deaths prevented or postponed by each specific cardiac intervention and by each risk factor change were calculated for England and Wales for the year 1981 and again for the year 2000.

The model aimed to include all medical and surgical treatments provided in 1981 and 2000. These interventions are listed in Table 1 and included all the interventions considered in earlier versions of the IMPACT model plus primary angioplasty and stenting for myocardial infarction, statins for primary prevention, platelet glycoprotein IIb/IIIa inhibitors for unstable angina, and spironolactone and β-blockers for heart failure (Table 1; see also Appendices 2 and 3 in the online-only Data Supplement).

The mortality reduction for each treatment was calculated by use of the relative mortality reduction reported in published meta-analyses and trials applied to the case-fatality observed in unselected patient cohorts. Survival benefit over a 1-year time interval was used for all treatments.

Polypharmacy Issues
The potential effect of multiple treatments in an individual patient was examined using the Mant and Hicks cumulative relative benefit approach: relative benefit = (1−[1−(1−treatment A)×(1−treatment B)×…]).

Treatment Compliance and Overlaps
To avoid double counting, potential overlaps between different groups of patients were identified and adjustments were made (see Appendix 6 in the online-only Data Supplement). For example, ~50% of the patients having CABG surgery have a previous myocardial infarction.

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.

Deaths Prevented or Postponed in 1981
A number of effective therapies were already in limited use in 1981. These included CABG surgery, cardiopulmonary resuscitation, β-blockers for acute myocardial infarction, diuretics for acute left ventricular heart failure, and therapy for moderate and severe hypertension (defined as a diastolic blood pressure >105 mm Hg). Precise patient data for some of these interventions, such as CABG, and eligible hypertensives were available from the data sources detailed above. Others were estimated after consultation with cardiologists in practice in 1981.

Risk Factor Trends and Mortality Benefits
For risk factor changes, the model uses regression (β) coefficients obtained from large cohort studies and MONICA analyses (Appendix 8 in the online-only Data Supplement). Each β coefficient quantifies the independent relationship between population change in a specific CHD risk factor (such as smoking, cholesterol, or blood pressure) and the consequent change in population mortality rate from CHD. These coefficients were reviewed and updated. The subsequent reduction in deaths produced by the decrease in each major risk factor was then estimated as the product of 3 variables: the number of CHD deaths observed in 1981 (the base year), the relative reduction in that risk factor, and the β coefficient.
A separate method was used for obesity, diabetes, physical activity, and socioeconomic deprivation, given the absence of suitable β coefficients (Appendix 5 in the online-only Data Supplement). Population-attributable risk fraction (PAR) was calculated by use of the conventional formula: \( \text{PAR} = \frac{\text{prevalence} \times (\text{relative risk} - 1)}{\text{prevalence} \times (\text{relative risk} - 1) + 1} \).

The number of CHD deaths attributable to each specific risk factor was then calculated for 1981 and for 2000. The difference between the two values then represented the deaths prevented or postponed because of the change in that specific risk factor in the population.

### Model Validation: Comparison With Observed Mortality Decreases

The model estimate for the total deaths prevented or postponed by all treatments plus all risk factor changes was summed and then compared with the observed decreases in mortality for men and women in each specific age group. On an a priori basis, any shortfall in the overall model estimate was then formally attributed to other, unmeasured risk factors.

### Sensitivity Analyses

Because of the uncertainties surrounding many of the values, a multway sensitivity analysis was performed using the analysis-of-extremes method. Illustrative examples of specific analyses and calculations are shown in Appendix 5 in the online-only Data Supplement.

### Results

In England and Wales between 1981 and 2000, CHD mortality rates decreased by 62% in men and 45% in women to 35 years old. There were 68,230 fewer CHD deaths than expected from baseline mortality rates in 1981.

### Medical and Surgical Treatments

Medical and surgical treatments together prevented or postponed \( \approx 25,805 \) deaths (minimum estimate, 17,110; maximum estimate, 49,042) (Table 1). This represented \( \approx 42\% \) of...
the total mortality decrease, after allowance for treatments given in 1981 (Figure 2). Substantial contributions came from specific treatments in individuals for secondary prevention (11.2%), heart failure (12.6%), acute myocardial infarction (7.7%), angina (7.0%), and hypertension (3.1%).

Approximately 4779 deaths were prevented or postponed by immediate treatments for acute myocardial infarction; the biggest contributions came from cardiopulmonary resuscitation, aspirin, and thrombolysis (Table 1). Coronary artery bypass surgery and angioplasty were estimated to prevent or postpone 1935 and 559 deaths, respectively, accounting for 3.8% of the total (Table 1).

Adjustment for Polypharmacy in Individual Patients
Applying the Mant and Hicks equation to the uptake of multiple medications in individual patients would reduce the total deaths prevented or postponed (25 830) by \( \approx 2118 \) (395 in acute myocardial infarction, 800 in heart failure patients, and 923 in secondary prevention) (Appendix 7 in the online-only Data Supplement).

Major Cardiovascular Risk Factors
Changes in the major cardiovascular risk factors together produced a best estimate of 35 944 fewer deaths (minimum estimate, 23 123; maximum, 62 195) (Table 2). This therefore accounted for some 58% of the total mortality decrease between 1981 and 2000. The biggest contribution came from the reduction in smoking (48.1%), along with decreases in serum total cholesterol levels (9.6%), blood pressure (9.5%), and deprivation (3.4%) (Figure 3). These mortality reductions reflected a substantial decline in smoking prevalence and smaller reductions in mean blood pressure, total cholesterol, and deprivation (Table 2).

Adverse trends were seen for obesity, physical activity, and diabetes. They together caused \( \approx 7650 \) additional CHD deaths (Table 2). The prevalence of obesity increased by 186%, resulting in an estimated additional 2095 CHD deaths. Diabetes prevalence increased by 66% with \( \approx 2890 \) additional CHD deaths, and indirect evidence suggested a 30% decrease in physical activity, with some 2660 additional deaths (Table 2).

Sensitivity Analyses, Validation, and Model Fit
Figure 3 demonstrates the results of the sensitivity analysis. The proportional contributions of specific treatments and risk factor changes to the overall decrease in CHD mortality in England and Wales between 1981 and 2000 remained relatively consistent (Figure 3). Thus, all secondary prevention

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>% Change in Risk Factor 1981–2000</th>
<th>B Coefficient</th>
<th>Best Estimate</th>
<th>Minimum Estimate</th>
<th>Maximum Estimate</th>
<th>Proportion of Overall Deaths Prevented or Postponed, %, Best Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking</td>
<td>−34.0</td>
<td>0.51</td>
<td>29 715</td>
<td>20 037</td>
<td>44 677</td>
<td>48.1</td>
</tr>
<tr>
<td>Population blood pressure</td>
<td>−7.7</td>
<td>1.67</td>
<td>5868</td>
<td>4246</td>
<td>15 469</td>
<td>9.5</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>−4.2</td>
<td>2.46</td>
<td>7900</td>
<td>5284</td>
<td>16 692</td>
<td>9.6</td>
</tr>
<tr>
<td>Deprivation</td>
<td>−6.6</td>
<td>1.24</td>
<td>2126</td>
<td>1063</td>
<td>3189</td>
<td>3.4</td>
</tr>
<tr>
<td>Physical activity</td>
<td>−30.6</td>
<td>0.50</td>
<td>−2862</td>
<td>−1491</td>
<td>−3460</td>
<td>−4.3</td>
</tr>
<tr>
<td>Obesity</td>
<td>+186.2</td>
<td>1.57</td>
<td>−2097</td>
<td>−1339</td>
<td>−2587</td>
<td>−3.4</td>
</tr>
<tr>
<td>Diabetes</td>
<td>+65.6</td>
<td>4.24</td>
<td>−2888</td>
<td>−2567</td>
<td>−4685</td>
<td>−4.7</td>
</tr>
<tr>
<td>Total risk factor effects</td>
<td>...</td>
<td>...</td>
<td>35 944</td>
<td>23 123</td>
<td>62 195</td>
<td>58.2</td>
</tr>
</tbody>
</table>

Figure 2. CHD deaths prevented or postponed by treatments and risk factor changes in England and Wales population, 1981 to 2000.
treatments together accounted for \( \approx 11\% \) of the total mortality decrease of 68,230. The minimum contribution was 7% and the maximum, 21%. This contribution therefore remained consistently larger than that for acute myocardial infarction or hypertension (Figure 3).

The agreement between the estimated and observed mortality decreases for men and women in each age group was generally good (Table 3). Overall, the model accounted for 89% of the total mortality decrease in England and Wales between 1981 and 2000 (95% in men and 77% in women). As planned, the remaining 11% was attributed to other, unmeasured factors such as dietary changes and life-course effects.

**Discussion**

CHD mortality decreased by more than 50% between 1981 and 2000 in England and Wales. Approximately 40% of the UK decrease was attributable to the combined effects of modern cardiological treatments and almost 60% to reduction in major risk factors, particularly smoking. This is consistent with the majority of other studies in the United States,20 Europe,21 Scotland,12 and New Zealand.11 Although Hunink et al9 attributed 71% of the recent US decline to “treatments,” this exception was more apparent than real. It principally reflected the categorization of risk factor decreases in individual patients with recognized CHD as “treatment benefits.” In the entire US population, 50% of the CHD mortality decline was actually explained by risk factor reductions. Furthermore, Hunink et al did not report on specific medical therapies.

Modern cardiological treatments together prevented or postponed \( \approx 26,000 \) deaths in 2000. Irrespective of whether best, minimum, or maximum estimates were used, the most substantial contributions came from secondary prevention and heart failure treatments. However, although heart failure treatments resulted in \( >7700 \) deaths prevented or postponed because of the relatively short life expectancy in these patients, this gained only \( \approx 25,360 \) life-years (just 2% of the total life years gained by cardiological treatments and population risk factor changes in England and Wales in 2000).22
Revascularization from CABG surgery and angioplasty together accounted for only 4% of the total mortality decrease, much as in the United States.23,24 This is a disappointingly small contribution, particularly when considering the large financial and political resources being consumed.13,24,25

Thrombolysis likewise accounted for only 25% of the deaths prevented by initial treatments for acute myocardial infarction. This was much less than aspirin and cardiopulmonary resuscitation, as in other studies.26 Furthermore, treating angina patients with aspirin in the community prevented almost twice as many deaths as treating unstable angina patients in hospitals, principally reflecting the far greater numbers involved (Table 1).

Treatment uptake levels were often poor (Table 1). Earlier work suggested that if even 80% of eligible patients had received appropriate therapy, ≈30 000 additional deaths might have been prevented or postponed each year33 in the United Kingdom, equivalent to 100 000 fewer deaths in the United States.

Reductions in the major risk factors between 1981 and 2000 accounted for ≈35 000 fewer deaths in England and Wales in 2000. The biggest single contribution reflected a large decrease in smoking prevalence, from 39% to 28% overall. Almost 10% of the mortality decrease came from a relatively small reduction (4.2%) in population total cholesterol level. This emphasizes the large β coefficient (1.9 to 5.4, depending on age27) and highlights the potential gains from larger reductions in population cholesterol.

The adverse trends in obesity, diabetes, and physical inactivity together contributed ≈8000 additional deaths in 2000. These canceled out 2 decades of improvement in cholesterol. Furthermore, continuing deteriorations are expected.13,14,28

We used relatively conservative β coefficients for smoking, cholesterol, and blood pressure. Even so, there was relatively little space left in the model, ≈11%, for potential mortality benefits from other, unquantified factors such as life-course effects, alcohol, and other dietary improvements.29

All the β coefficients and relative risk values used in the model were independent, being obtained from multiple regression analyses. The interaction between the major risk factors should therefore have been accounted for. However, these estimates may still overestimate, because most models, of necessity, entered data into the model on only a limited range of risk factors. For the MONICA study, for instance, these are smoking (yes or no), systolic blood pressure, total cholesterol, and body mass index.4 There are many other potentially important risk factors for CHD, including diet (such as consumption of fish oils, antioxidants, and alcohol) and life-course factors. Some novel risk factors may be highly correlated with the 7 risk factors measured and considered in the model. It is therefore possible that the calculated coefficients contain the effects of some of these other, unmeasured risk factors.

Modeling studies have a number of potential strengths. They transparently integrate and simultaneously consider huge amounts of data from many sources. Explicit assumptions can then be tested by sensitivity analyses.8

Modeling studies also have limitations. They are dependent on the variable quality and extent of data available on CHD risk factor trends and treatment uptakes.30 Assumptions and robust sensitivity analyses therefore become essential.19 However, the relative contribution of each risk factor and treatment to the overall CHD mortality decline was little changed whether considering best, minimum, or maximum estimates (Figure 2). The model included only those 25 to 84 years old because of very limited data in older groups. We considered only deaths from CHD and ignored “competing causes” such as cancer.6 However, reductions in smoking would actually decrease deaths from lung cancer and other cancers.2,7 This analysis focused on mortality rather than symptomatic relief, “life-years-gained,” or disease incidence.22,31 These all merit attention in future work.

The IMPACT model assumes that estimates of efficacy from randomized controlled trials can usually be generalized to effectiveness in clinical practice. This seems reasonable.32 Further development work is clearly needed.8 Finally, although lag times were not explicitly considered, they may be relatively unimportant over a 20-year analysis. Substantial mortality reduction occurs within 1 to 4 years of quitting smoking or reducing cholesterol.27,34

In conclusion, more than 50% of the recent CHD mortality decrease in England and Wales was attributed to reductions in major risk factors, and some 40% to medical therapies. These findings might be cautiously generalizable to the United States and other comparable industrialized countries. Comprehensive CHD strategies should therefore actively promote primary prevention as well as maximizing the population coverage of effective treatments.

Acknowledgments
Belgin Unal was funded by a North West Regional Research and Development Training Fellowship. We also thank the UK Data Archive and many colleagues for constructive comments, particularly Tim Doran, Dogan Fidan, and Margaret Whitehead.

References