Original Contribution

Life-Years Gained Among US Adults From Modern Treatments and Changes in the Prevalence of 6 Coronary Heart Disease Risk Factors Between 1980 and 2000

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Has the recent US decline in coronary heart disease (CHD) mortality increased life expectancy? The authors estimated the number of life-years gained from CHD treatments and changes in the prevalence of cardiovascular disease risk factors for the US population between 1980 and 2000. The previously validated IMPACT model was used to integrate data on numbers of CHD patients, treatment uptake, treatment effectiveness, population risk factor trends, and median survival among US adults. There were 308,900 fewer CHD deaths in 2000 among Americans aged 25–84 years than if 1980 mortality rates had applied. These 308,900 fewer deaths represented approximately 3,147,800 life-years gained (sensitivity analysis range, 2,448,900–3,744,900). Treatments for patients accounted for approximately 1,092,400 (751,700–1,387,000) life-years gained, whereas changes in the prevalence of population risk factors accounted for a gain of 2,055,500 (1,697,200–2,346,300) life-years. However, the 2,770,500 life-years gained through decreased levels of smoking, cholesterol, blood pressure, and physical inactivity were diminished by a loss of 715,000 life-years attributable to increased rates of obesity and diabetes. Therefore, modest reductions in the prevalence of several major cardiovascular disease risk factors accounted for more than twice as many life-years gained as did treatments. Unfortunately, these gains were partially offset by substantial increases in obesity and diabetes.

coronary disease; mortality; risk factors; therapeutics; United States

Abbreviations: CHD, coronary heart disease; DPP, deaths prevented or postponed; NHANES, National Health and Nutrition Examination Survey.

Between 1980 and 2003, the life expectancy of US newborns increased by 4.8 years in men and 2.7 years in women (1). Much of this substantial and important increase has been attributed to dramatic reductions in rates of US coronary heart disease (CHD) mortality, which decreased by approximately 50% between 1980 and 2000 (2). This decline in CHD mortality rates has in turn been attributed to increased use of evidence-based therapies—principally for acute coronary syndromes (such as thrombolysis, aspirin, statins, angiotensin-converting enzyme inhibitors, and revascularization), chronic CHD angina, and heart failure (aspirin, statins, angiotensin-converting enzyme inhibitors, beta-blockers, coronary artery bypass surgery, and angioplasty)—and to population-wide reductions in the prevalence of cardiovascular disease risk factors—principally smoking, high cholesterol, high blood pressure, and physical inactivity (3). Quantification of increased life expectancy is clearly more relevant and informative than a crude estimation of delayed mortality.

While investigators in several studies have examined reductions in CHD mortality rates (4–7), few have assessed gains in life-years associated with these reductions. In 1 study, researchers estimated that reductions in CHD and cerebrovascular disease death rates accounted for 10%–20% of the 7.1-year increase in life expectancy seen in the United States between 1950 and 1989 (8). In a more recent study, Tsevat et al. (9) attributed a 1.0- to 1.2-year increase in life expectancy among 35-year-old US men to lower blood
pressure and a 0.5- to 1.2-year increase to smoking cessation. A Canadian study produced similar estimates (10). Researchers from the Framingham Study suggested that participation in moderate and high levels of physical activity was associated with 1.3 and 3.7 more life-years, respectively, among men and 1.5 and 3.5 more life-years among women (11).

We recently estimated (12) that in England and Wales in 2000, combined CHD therapies were associated with a gain of 195,000 life-years but reductions in the prevalence of major CHD risk factors were associated with a much larger gain of nearly 800,000 life-years, much as in Ireland (13). However, to our knowledge, no previous studies have attempted to estimate life-expectancy gains attributable to the recent, substantial reductions in the number of CHD deaths in the United States or have distinguished between life-years gained in youth and those gained in old age. Therefore, in this study, we estimated the increase in life-years among US residents aged 25–84 years associated with CHD treatments and with changes in the prevalence of cardiovascular disease risk factors from 1980 to 2000.

MATERIALS AND METHODS

Estimation of numbers of DPP in the United States in 2000

We used the IMPACT CHD mortality model to estimate the number of deaths prevented or postponed (DPP) in 2000 that could be attributed either to increases in the use of cardiac treatments or decreases in the prevalence of CHD risk factors since 1980 (3). This model has been continuously developed since 1996. A detailed description of the method used in building the model can be found on the IMPACT Web site (http://www.liv.ac.uk/PublicHealth/sc/bua/impact.html) and in several publications (3, 5–7). In brief, the IMPACT model combines data describing:

- the number of CHD patients in each disease subcategory;
- the percentage of patients who received specific medical and surgical treatments;
- population trends in the prevalence of major cardiovascular disease risk factors (smoking, high cholesterol, high systolic blood pressure, obesity based on body mass index, diabetes, and physical inactivity); and
- the effectiveness of specific treatments and major risk factor reductions in preventing or postponing CHD deaths.

Estimates of the number of CHD DPP in each treatment group were based on the mortality reduction associated with various treatments (reported in published trials and meta-analyses) applied to the case fatality rates observed in unselected patient cohorts (3, 5, 14). To adjust for sample overlaps between different treatment groups, we subtracted the number of people in overlapping subgroups from the number in the main group. Details on the IMPACT model, including overlap assumptions, are available in the supplementary appendix of a recent article (3). The specific treatment interventions are detailed in Table 1.

For risk factor changes, the model employs regression beta coefficients obtained from meta-analyses and large cohort studies that quantify the independent relations between changes in each risk factor (analyzed as a continuous variable) and subsequent changes in CHD mortality rates (15–17). After selecting the best estimates of these coefficients, as well as minimum and maximum estimates as previously described (3, 14–17), we then estimated the reduction in deaths associated with the decrease in each major risk factor as the product of 3 variables: the number of CHD deaths observed in 1980 (the baseline year), the changes in the prevalence of that risk factor, and the regression coefficient (3, 6, 12, 14).

For smoking, diabetes, and physical inactivity, analyzed as categorical variables, we used the population attributable risk fraction (PARF), which we calculated using the following formula (3, 6, 12, 14):

\[
\text{PARF} = \left[ \frac{\text{Prevalence} \times (\text{Relative Risk} - 1)}{(\text{Prevalence} \times (\text{Relative Risk} - 1)) + 1} \right]
\]

We calculated the number of CHD deaths attributable to each factor for 1980 and again for 2000, using independent relative risk values from the INTERHEART study (18). The difference between the 2 values represented the number of DPP that were attributable to the change in the prevalence of that risk factor in the population, stratified by age and sex. Examples of such calculations of the population attributable risk fraction may be found on the IMPACT Web site (http://www.liv.ac.uk/PublicHealth/sc/bua/impact.html) and in previous reports (3, 6, 12, 19).

Because independent regression coefficients and relative risks for CHD death associated with each risk factor were taken from multivariate analyses, we assumed that there was no further synergy between the treatment and risk factor sections of the model or among the major risk factors. To account for potential differences in the effect, we systematically quantified the number of DPP as a result of risk factor changes for each specific patient group. We did not model lag times between changes in the prevalence of risk factors and changes in rates of CHD-related deaths because we assumed that the effect of these lag times would be relatively unimportant over a 2-decade period (6, 16, 20, 21).

We used National Health and Nutrition Examination Survey (NHANES) data from 1976–1980 and 1999–2000 to calculate changes in mortality rates attributable to changes in the prevalence of CHD risk factors. Details about NHANES are available online (http://www.cdc.gov/nchs/nhanes.htm). In the NHANES, current smokers were defined as people who had smoked more than 100 cigarettes during their lifetime and were still smoking. Serum total cholesterol was measured enzymatically with the use of commercial reagents. High cholesterol was defined as a total cholesterol concentration greater than or equal to 200 mg/dL, or the self-reported use of cholesterol-lowering medication. High blood pressure was defined as a blood pressure greater than or equal to 140/90 mm Hg or the self-reported use of antihypertensive medication. Body mass index was calculated from participants’ measured heights and weights (weight (kg)/height (m)^2), and obesity was defined as a body mass index greater than or equal to 30.

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Diabetes was defined as a fasting glucose concentration greater than or equal to 126 mg/dL or a self-reported physician diagnosis of diabetes. We used data from the Behavioral Risk Factor Surveillance System surveys for 1988 and 2000 (http://www.cdc.gov/brfss/index.htm) to assess changes in the prevalence of physical inactivity. Behavioral Risk Factor Surveillance System respondents were considered to be physically inactive if they did not report participation in any leisure-time physical activity. We assumed that changes in inactivity levels between 1988 and 2000 followed a linear trend back to 1980, because we could not locate appropriate estimates on physical inactivity for 1980.

Model calibration: comparison of estimated reductions in the number of DPP with observed reductions

After using the IMPACT model to estimate the total number of DPP associated with each treatment and risk factor change, we summed these estimates and compared the total with the difference between the observed number of deaths among men and women in each age group and the number of deaths expected had CHD mortality rates in 2000 been the same as rates in 1980.

Median survival data

DPP by medical and surgical treatments. For each treatment category, we estimated patients’ median survival using the best available population-based data from unselected cohorts of Medicare patients following their hospital admission for acute myocardial infarction, heart failure, or revascularization. Additional age-specific median survival data for unstable angina patients were obtained from a large retrospective cohort study of unselected patients in the United Kingdom (19, 22, 23).

DPP by reductions in the prevalence of CHD risk factors. Coronary atheroma generally begins early in life; symptomatic manifestations occur late in life and even then may go undiagnosed. Because risk factor reductions such as

<table>
<thead>
<tr>
<th>Intervention or Condition</th>
<th>No. of Patients Eligible</th>
<th>No. of Deaths Prevented or Postponed</th>
<th>Median Survival, years</th>
<th>Life-Years Gained</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>670,700</td>
<td>21,600</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>(thrombolysis, cardiopulmonary resuscitation, aspirin, statins, ACE inhibitors, primary angioplasty, and CABG surgery)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unstable angina</td>
<td>665,300</td>
<td>13,600</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>(aspirin, heparin, clopidogrel, angioplasty, and CABG surgery)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secondary prevention</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-myocardial infarction</td>
<td>2,867,000</td>
<td>28,600</td>
<td>6.5</td>
<td>158,400</td>
</tr>
<tr>
<td>Post-CABG/angioplasty (aspirin, statins, beta-blockers, ACE inhibitors, rehabilitation)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic angina (total)</td>
<td>4,987,400</td>
<td>17,800</td>
<td>11.3</td>
<td>196,000</td>
</tr>
<tr>
<td>CABG surgery 1990–2000 (with CABG in 1980 subtracted)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angioplasty 1990–2000</td>
<td>2,206,000</td>
<td>4,400</td>
<td>9.4</td>
<td>41,300</td>
</tr>
<tr>
<td>Aspirin in community</td>
<td>2,119,500</td>
<td>1,000</td>
<td>10.1</td>
<td>9,900</td>
</tr>
<tr>
<td>Statins in community</td>
<td>2,119,500</td>
<td>1,000</td>
<td>10.1</td>
<td>9,900</td>
</tr>
<tr>
<td>Heart failure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Requiring hospitalization</td>
<td>258,700</td>
<td>11,700</td>
<td>2.1</td>
<td>24,100</td>
</tr>
<tr>
<td>Community treatment (ACE inhibitors, beta-blockers, aspirin, and statins)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension treatmentb</td>
<td>54,353,700</td>
<td>23,800</td>
<td>9.6</td>
<td>308,000</td>
</tr>
<tr>
<td>Statins, etc., for primary prevention lipid reductionb</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total treatments in 2000</td>
<td>159,500</td>
<td>1,092,400</td>
<td>34.7</td>
<td>1,387,000</td>
</tr>
</tbody>
</table>

Abbreviations: ACE, angiotensin-converting enzyme; CABG, coronary artery bypass graft; NA, not applicable.

a Acute myocardial infarction and unstable angina patients were only counted for 1 year; thereafter they were counted in the secondary prevention category.

b Hypertension treatments and statins, etc., for primary prevention lipid reduction were quantified as medical interventions.
smoking cessation may prevent death from CHD either before or after the onset of symptomatic disease, we calculated age-specific median survival values for 2 distinct groups: 1) patients with recognized CHD, whose median survival we assumed to be similar to that of age-matched myocardial infarction survivors (19), and 2) asymptomatic persons without recognized CHD, whose median survival we assumed to be similar to that of age-matched members of the general population.

**Estimation of life-years gained**

We estimated the number of life-years gained in 2000 for each treatment category and each risk factor change, stratified by age and sex, as the product of the number of DPP in 2000 and the age-specific median survival expectation for that age-sex group. We did not adjust estimates of life-years gained to account for “competing causes of mortality,” which generally amounted to less than 1 extra year of life (10, 24).

**Sensitivity analyses**

We tested all assumptions in a sensitivity analysis. We assumed that the minimum estimate for age-specific median survival among asymptomatic persons without recognized CHD lay midway between that for myocardial infarction survivors and that for the general population and that the maximum estimate equaled the projected mean survival time in the general US population. A detailed description of the best, minimum, and maximum values for age-specific median survival was provided in a recent publication (3).

We used the “analysis of extremes” method (12, 13, 25) to perform a sensitivity analysis. To address the uncertainties surrounding the key variables (patient numbers, treatment usage, treatment efficacy, and median survival), we used 95% confidence intervals, where available, or the minimum and maximum plausible values for each variable to generate minimum and maximum estimates of life-years gained (12, 13, 25). All results were rounded to the nearest 100.

**RESULTS**

Some 341,700 fewer CHD deaths occurred in the United States in 2000 than would be expected had mortality rates stayed the same as in 1980, the baseline year. The IMPACT model accounted for a reduction of approximately 308,900 CHD deaths (90.4% of the observed reduction of 341,700 CHD deaths). The remaining 8.6% of the decrease was probably explained by model imprecision and by changes in other, minor risk factors.

We estimated that these 308,900 fewer deaths resulted in a gain of approximately 3,147,800 life-years (range: 2,448,900–3,744,900) among persons aged 25–84 years.

**Life-years gained from medical and surgical treatments among patients with CHD**

We estimated that specific medical and surgical treatments for approximately 12 million patients with CHD prevented or postponed approximately 159,500 CHD deaths in the United States in 2000. These treatments (specified in Table 1) were together associated with an estimated gain of 1,092,400 life-years (range: 751,700–1,387,000) (Table 1). Thus, each death postponed by treating a patient with CHD yielded, on average, an additional 6.9 years of life (total life-years gained divided by total deaths postponed). The largest numbers of life-years gained were associated with secondary preventive treatments administered after myocardial infarction (185,400 life-years) and after revascularization (72,700 life-years), heart failure treatments (121,900 life-years), coronary artery bypass surgery (135,000 life-years), and angioplasty procedures (41,300 life-years) (Table 1).

### Table 2. Summary of Life-Years Gained in 2000 Among US Adults Aged 25–84 Years \((n = 177,745,100)\) Attributable to Changes in the Prevalence of 6 Coronary Heart Disease Risk Factors Between 1980 and 2000

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>No. of Deaths Prevented or Postponed</th>
<th>Median Survival, years</th>
<th>Life-Years Gained</th>
<th>Best Estimate</th>
<th>% of Total</th>
<th>Minimum Estimate</th>
<th>%</th>
<th>Maximum Estimate</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Risk factors whose prevalence decreased</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>High cholesterol</td>
<td>82,800</td>
<td>13.6</td>
<td>1,102,100</td>
<td>35.0</td>
<td>29.3</td>
<td>1,241,700</td>
<td>39.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High systolic blood pressure</td>
<td>68,800</td>
<td>13.4</td>
<td>924,200</td>
<td>29.4</td>
<td>24.0</td>
<td>1,081,800</td>
<td>34.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>39,900</td>
<td>14.5</td>
<td>577,100</td>
<td>18.3</td>
<td>15.0</td>
<td>672,200</td>
<td>21.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical inactivity</td>
<td>17,400</td>
<td>9.6</td>
<td>167,100</td>
<td>5.3</td>
<td>4.3</td>
<td>198,700</td>
<td>6.3</td>
<td></td>
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</tr>
<tr>
<td>Subtotal</td>
<td>208,900</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td><strong>Risk factors whose prevalence increased</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td>–25,900</td>
<td>29.4</td>
<td>–346,200</td>
<td>–11.0</td>
<td>–9.2</td>
<td>–409,800</td>
<td>–13.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>–33,500</td>
<td>11.1</td>
<td>–368,800</td>
<td>–11.7</td>
<td>–9.5</td>
<td>–438,300</td>
<td>–13.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subtotal</td>
<td>–59,400</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total risk factors</td>
<td>149,600</td>
<td></td>
<td></td>
<td></td>
<td>53.9</td>
<td>2,346,300</td>
<td>74.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Primary prevention medications produced a gain of approximately 308,000 life-years from hypertension therapies and 173,200 life-years from lipid reduction therapies (Table 1).

Life-years gained from risk factor changes in the population

We estimated that approximately 149,600 CHD deaths were prevented or postponed as a result of secular changes in the population prevalence of CHD risk factors between 1980 and 2000 and that these changes accounted for approximately 2,055,500 life-years gained (range, 1,697,200–2,346,300) (Table 2). Thus, each death postponed by the reduction in the prevalence of a risk factor yielded an additional 13.7 years of life, on average. Increases in life-years attributable to decreases in the prevalence of 4 risk factors (smoking: 577,100; high cholesterol: 1,102,100; high systolic blood pressure: 924,200; and physical inactivity: 167,100) were partially offset by decreases in life-years attributable to increases in the prevalence of 2 risk factors (diabetes and obesity, which together were associated with an increase of 59,400 deaths and hence the loss of 715,000 life-years (range, 587,000–848,100)).

![Figure 1](image1.png)

**Figure 1.** Life-years gained among US adults aged 25–84 years from coronary heart disease treatments and changes in the prevalence of coronary heart disease risk factors between 1980 and 2000, by age and sex.

![Figure 2](image2.png)

**Figure 2.** Results from a sensitivity analysis of the contributions of specific coronary heart disease treatments (left) and risk factor changes (right) to life-years gained in the United States, 1980–2000. Squares, best estimate; diamonds, overall best estimate; bars, maximum and minimum estimates. Rx, prescription.
Age and sex distribution of life-years gained

Overall, almost two-thirds of the life-years gained were among men (Figure 1). Reductions in the prevalence of CHD risk factors accounted for 68.4% of all life-years gained among men and 58.7% of all life-years gained among women. People under age 65 years accounted for half (51%) of the total life-years gained through treatments and risk factor reductions (Figure 1 and Appendix Table).

Sensitivity analyses

The relative contributions of treatments among CHD patients and population-wide risk factor reductions to life-years gained remained fairly constant, irrespective of whether we used best, maximum, or minimum estimates of life-years gained (Figure 2).

DISCUSSION

The CHD mortality rate in the United States was 43% lower in 2000 than in 1980. This resulted in approximately 341,700 fewer deaths attributable to CHD than would have occurred had the CHD mortality rate not changed (3). Our model accounted for approximately 90% of the total reduction in the number of CHD deaths and translated this reduction into some 3 million additional life-years. Thus, each postponed death attributable to treatment of a patient with recognized CHD yielded, on average, an additional 6.9 years of life (total life-years gained divided by total deaths postponed). By contrast, each postponed death attributable to the reduction in the prevalence of a cardiovascular disease risk factor in the population yielded double the benefit—on average, an additional 13.7 years of life. These findings are generally consistent with those from previous studies in other countries (12, 13). For example, in a recent study in Ireland, Kabir et al. (13) reported 7.7 life-years gained for each postponed death attributable to treatment and 16.5 life-years gained for each postponed death attributable to risk factor reduction.

Estimates derived from our model indicated that medical and surgical treatments for patients with CHD accounted for approximately 1 million life-years gained among US adults in 2000. Much of this gain came from just 2 treatment categories: secondary prevention and hypertension therapies. These results are consistent with previous findings that simple, inexpensive treatments applied to the majority of eligible patients can produce huge gains (3). Thus, primary prevention medications for hypertension and hyperlipidemia together produced a gain of almost 500,000 additional life-years.

Conversely, revascularization (coronary bypass surgery or angioplasty) undeniably improves quality of life and also prolongs life in some patients. However, because it applied to a relatively small number of selected patients, revascularization accounted for less than 6% of the total number of life-years gained, a percentage very similar to that of previous studies (5, 6, 26).

Reductions in the prevalence of 4 major risk factors (smoking, high cholesterol, high blood pressure, and physical inactivity) accounted for an increase of almost 3 million life-years among Americans in 2000. Therefore, future risk factor reductions through environmental and behavioral changes might be implemented in childhood or early adult life, thereby gaining far more additional life-years than therapies commenced after CHD has manifested.

However, this beneficial increase was partially offset by dramatic rises in the prevalences of obesity and diabetes, which together accounted for a decrease of more than 0.7 million life-years, consistent with recent studies (27). The combination of excessive caloric intake and poor nutrition, compounded by low activity levels, has been identified as a major public health challenge in the new millennium (27–29).

Large gains in life-years came from substantial reductions in the prevalence of smoking in the United States between 1980 and 2000; these were most likely due to public health campaigns and smoking cessation strategies. However, in 2007, the prevalence of smoking among US adults remained around 24% in men and 18% in women, and twice that in certain demographic groups (30). Furthermore, even though US physical inactivity rates have been decreasing, as of 2005, only half of US adults engaged in regular physical activity: approximately 50% of men and 47% of women (31).

Overall, almost two-thirds of the life-years gained were among men, principally reflecting their earlier age of onset of CHD.

In our view, the IMPACT model is the first published CHD mortality model for the United States that comprehensively considers the effects of all standard CHD treatments and all major CHD risk factors (3). However, the results of our analyses using this model are reassuringly consistent with findings derived from earlier modeling studies conducted in the United States (4), Europe (6, 7, 32), and New Zealand (5). The accuracy of all such modeling studies is dependent on the quality of data used in the models (3, 6). Explicit assumptions are made, and robust sensitivity analyses become essential for accurate results (25).

One limitation is that this model excluded people over age 84 years, because data for older subjects are severely limited in quality and quantity. Another limitation is that we did not precisely quantify the effects of “competing causes” of death, such as cancer, on estimates of life-years gained (8). However, such effects are likely to have been small, representing less than 1 year (10, 24). Moreover, reductions in the prevalence of smoking would contribute to decreases in the numbers of deaths from lung cancer and several other cancers, as well as to decreases in the number of deaths from CHD (6, 20). The IMPACT model also assumed that estimates of treatment efficacy from randomized controlled trials accurately depicted the effectiveness of such treatments in clinical practice, regardless of the baseline level of risk. Although this assumption appears reasonable (33, 34), it may have led to overestimation of some treatment benefits.

In this paper, we focused on control of risk factors. In future studies, researchers should quantify the effect of policy interventions and lifestyle changes on specific risk factor levels, including the potential gains derived from primordial prevention (if a low risk state were maintained from youth) (35). Furthermore, risk factor changes occur in both persons with CHD and persons without recognized CHD. The concepts and numbers are actually complex; therefore, we plan...
to address them in a separate analysis comparing mortality gains from risk factor reductions in CHD patients and subjects without recognized CHD. This research will follow previously tested methods (21).

Values for median survival were taken from the best available sources but can only be considered crude estimates. However, the main findings remained secure in a rigorous sensitivity analysis. Finally, the model did not consider the effect of lag time in assessing how changes in the prevalence of risk factors were associated with CHD mortality rates. However, such lag times are probably relatively unimportant over a 20-year analysis, because in previous research mortality rates declined substantially within 1–2 years among persons who stopped smoking or reduced previously elevated cholesterol levels (15, 20, 34).

These important limitations will merit further attention and methodological development.

In this study, we showed that modern treatments were associated with an increase of approximately 1 million life-years among US CHD patients in 2000. However, recent changes in the prevalence of CHD risk factors in the wider population accounted for a gain of approximately 2 million life-years (modest decreases in the prevalences of smoking, high cholesterol, high blood pressure, and physical inactivity, partially offset by increases in the prevalences of obesity and diabetes). This association between cardiovascular disease risk factor reduction and an increase in life-years emphasizes the importance of federal, state, and local public health efforts to promote healthier diets, facilitate physical activity, and control tobacco use.

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Author contributions: S. Capewell developed the protocol, built the IMPACT model for the United States, analyzed and interpreted the results, and drafted the article. D. Hayes helped obtain and critically review the data, interpret the results, and finalize the article. E. Ford helped build the US IMPACT model, obtain and critically review the data, interpret the results, and finalize the article. J. Critchley helped develop successive versions of the IMPACT model, contributed to the conception and design of the study, critically reviewed the data and interpreted the results, and revised the article. J. Croft, K. Greenlund, and D. Labarthe contributed to the design of the study, critically reviewed the data and interpreted the results, and approved the final article.

The findings and conclusions in this article are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention. S. Capewell and E. Ford had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the analyses. No protocol approval was needed for this study, since no human participants were involved in it.

Conflict of interest: none declared.

REFERENCES


Appendix Table. Life-Years Gained Among US Adults Through Treatments and Risk Factor Reductions, by Age Group and Sex, 1980–2000a

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Age Group, years</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25–34</td>
<td>35–44</td>
</tr>
<tr>
<td>Treatment effects in 2000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Risk factor effects in 2000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total treatment and risk factor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>factor effects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of total</td>
<td>0.8</td>
<td>8.0</td>
</tr>
<tr>
<td>Risk factor:treatment ratio</td>
<td>5.9</td>
<td>4.1</td>
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<tr>
<td>Treatment effects in 2000</td>
<td>5,800</td>
<td>7,700</td>
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<tr>
<td>Risk factor effects in 2000</td>
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<td>31,700</td>
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<tr>
<td>Total treatment and risk factor</td>
<td>8,100</td>
<td>39,400</td>
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<tr>
<td>factor effects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of total</td>
<td>0.8</td>
<td>3.9</td>
</tr>
<tr>
<td>Risk factor:treatment ratio</td>
<td>4.1</td>
<td>4.3</td>
</tr>
</tbody>
</table>

a Data for Figure 1.