Life-Years Gained From Modern Cardiological Treatments and Population Risk Factor Changes in England and Wales, 1981–2000

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Objectives. We estimated life-years gained from cardiological treatments and cardiovascular risk factor changes in England and Wales between 1981 and 2000.

Methods. We used the IMPACT model to integrate data on the number of coronary heart disease patients, treatment uptake and effectiveness, risk factor trends, and median survival in coronary heart disease patients.

Results. Compared with 1981, there were 68 230 fewer coronary deaths in 2000. Approximately 925 415 life-years were gained among people aged 25–84 years (range: 745 195–1 138 655). Cardiological treatments for patients accounted for approximately 194 145 life-years gained (range: 142 505–259 225), and population risk factor changes accounted for approximately 731 270 life-years gained (range; 602 695–879 430).

Conclusions. Modest reductions in major risk factors led to gains in life-years 4 times higher than did cardiological treatments. Effective policies to promote healthy diets and physical activity might achieve even greater gains. (*Am J Public Health.* 2005;95:103–108. doi: 10.2105/AJPH.2003.029579)

Life expectancy at birth in England and Wales increased by 4.4 years in men and 3.2 years in women between 1981 and 2000.[1] Much of this increase has been attributed to reductions in coronary heart disease mortality rates, which have halved in the past 2 decades. The coronary heart disease mortality decline has been attributed to the widespread use of effective therapies such as thrombolysis, aspirin, angiotensin-converting enzyme inhibitors, statins, and coronary bypass surgery.[2] However, reductions in major risk factors such as smoking, cholesterol, and blood pressure[3] also have made substantial contributions.[4]

Furthermore, the majority of cardiology studies in other countries suggest that improvements in treatment explain less than half of the mortality decline[5,6]; for example, such improvements accounted for just 43% of the decline in the United States between 1980 and 1990[7]; 40% and 48% of the decline in New Zealand between 1974 and 1981[6] and between 1982 and 1993, respectively[8]; 40% of the decline in Scotland between 1975 and 1994[9]; and 42% of the decline in England and Wales between 1981 and 2000.[10]

However, previous analyses have generally reported reductions in mortality rather than gains in longevity. We therefore estimated the life-years gained as a result of cardiological treatments and changes in cardiovascular risk factor levels that occurred between 1981 and 2000 in England and Wales.

METHODS

Estimation of Deaths Prevented or Postponed in England and Wales in 2000

We used the IMPACT coronary heart disease mortality model to estimate the number of deaths prevented or postponed in 2000 that could be attributed to improved cardiac treatment uptake or risk factor changes since 1981.[10] The IMPACT mortality model was developed in 1996; published reviews of the model have appeared in several peer-reviewed journals since 1999 and provide more detailed methodology.[8,9,11] In brief, this model combines data describing (1) the number of coronary heart disease patients in each disease subcategory, (2) uptake of specific medical and surgical treatments, (3) population trends in major cardiovascular risk factors (smoking, cholesterol, blood pressure, obesity, diabetes, physical activity, and socioeconomic deprivation), and (4) effectiveness of specific cardiological treatments and risk factor reductions.[10]

The number of coronary heart disease deaths prevented or postponed in each treatment group was based on the relative mortality reduction reported in published trials and meta-analyses applied to the case fatality rates observed in unselected patient cohorts. To avoid sample overlap (double counting), we first made adjustments for overlaps between different treatment groups by subtracting the overlapping subgroup with the main group. For example, we reduced the myocardial infarction survivors total by 20% to allow for those patients who rapidly developed heart failure after myocardial infarction. (Details of the overlap assumptions and other aspects of the IMPACT Model are available at: http://www.liv.ac.uk/PublicHealth/sc/bua/IMPACT-Model-Appendices.pdf).

For risk factor changes, the model employs regression (β) coefficients obtained from meta-analyses and large cohort studies. Each β coefficient quantifies the relationship between population change for a specific risk factor (smoking, cholesterol, blood pressure) and the consequent change in population mortality rates from coronary heart disease. Published cohort and MONICA (Monitoring Trends in Cardiovascular Disease and Risk Factors) studies have generated a range of different coefficients that describe the relationship between each separate risk factor and coronary heart disease mortality.[12,13] First, we critically reviewed these coefficients; then, we used these coefficients to provide the best estimates as well as the minimum and maximum estimates.[12,13] For the decrease in each major risk factor, the subsequent reduction in deaths was then estimated as the product of 3 variables: the number of coronary heart disease deaths observed in 1981 (the baseline year), the relative reduction in that risk factor, and the regression coefficient.

Median Survival Data

Deaths prevented or postponed by medical and surgical treatments. For each treatment category, median survival was obtained from the best available population-based data.[14,15] Most age-specific median survival values came from large retrospective cohort studies of unselected patients with acute myocardial infarction[14] or with heart failure.[15] Median survival estimates for patients with hypertension were based on the Glasgow Blood Pressure Clinic cohort (stratified by age and gender).[16] Estimates of survival after coronary surgery were obtained from local sources[17] (J. Astbury, Information and Statistics Division, National Health Service, Edinburgh, Scotland; unpublished data, 1991) and a cohort study in Scotland.[17] We assumed that angioplasty for angina had no additional survival benefit.[18]

Deaths prevented or postponed by reductions in coronary heart disease risk factors. Coronary atheroma generally begins early in life; symptomatic manifestations occur late in life and even then may go undiagnosed. Risk factor reductions such as smoking cessation therefore may prevent mortality either before or after the onset of symptomatic disease. Age-specific median survival was derived for 3 separate groups of patients. Among (1) patients with recognized coronary heart disease, median survival was assumed to be very similar to that in age-matched myocardial infarction survivors; among (2) asymptomatic individuals, median survival was based on age-specific life expectancy for the general population[1]; among (3) patients with symptomatic but unrecognized coronary heart disease, median survival was assumed to lie midway between the values for myocardial infarction survivors[14] and the general population.[1] We tested all assumptions in a sensitivity analysis.

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 gender, as the product of the number of deaths prevented or postponed in 2000 and the age-specific median survival for that age–gender group. Estimates of life-years gained were adjusted to take into account the modest influence of "competing causes of mortality" and generally amounted to less than 1 extra year of life.[19,20]

Sensitivity Analyses

We used the analysis of extremes method[21] to perform a sensitivity analysis. Our analysis addressed the uncertainties surrounding the key variables (patient numbers, treatment uptake, 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.[21]

RESULTS

In 2000, 68 230 fewer coronary heart disease deaths occurred than expected from mortality rates in 1981, the baseline year. We compared the age-specific model estimates for deaths prevented or postponed by all interventions with the observed declines in mortality in each age and gender category. The model estimated 61 595 fewer deaths, representing 90% of the observed coronary heart disease mortality decline.[10] These 61 595 fewer deaths resulted in a gain of approximately 925 415 life-years among people aged 25–84 years (range: 745 195–1 138 655) (Tables 1 and 2).

Life-Years Gained From Medical and Surgical Treatments

Specific medical and surgical treatments for patients with coronary heart disease prevented or postponed approximately 25 745 deaths in England and Wales in 2000.[10] Such treatments therefore achieved a gain of approximately 194 145 life-years (range: 142 505–259 225) (Table 1). The largest contributions came from secondary preventive treatments for patients after myocardial infarction or revascularization (32%), heart failure treatments (13%), and hypertension treatments (9%) (Table 1). Coronary artery bypass surgery and angioplasty procedures together accounted for 17% of the life-years gained from treatments (Table1).

TABLE 1—Life-Years Gained From Medical and Surgical Treatments of Coronary Heart Disease: England and Wales, 2000

Intervention	No. of Eligible Patients	No. of Deaths Prevented or Postponed[a]	Life-Years Gained,[a] Best Estimate (Range)	Proportion of Total Life-Years Gained From Treatments, %
Acute myocardial infarction	66 195	5750	38 330 (20 795–57 880)	19.7
Secondary prevention				
After myocardial infarction	313 380	3580	24 520 (11 900–37 140)	12.6
After coronary surgery or angioplasty	315 680	3055	37 660 (35 360–39 960)	19.4
Angina				
Coronary surgery	187 415	1935	25 805 (22 550–31 695)	13.3
Angioplasty	112 405	560	7905 (5405–10410)	4.1
Unstable angina	72 600	910	5530 (4700–9400)	2.8
Aspirin in never-hospitalized patients	2 114 665	1105	9690 (4845–14 535)	5.0
Hospital treatment	41 385	4755	6120 (4895–7340)	3.2
Community treatment	242 090	3210	19 240 (7605–21 140)	9.9
Hypertension treatments	12 592 120	1890	17 775 (15 290–25 485)	9.2
Statins for primary prevention	7 630 760	145	1570 (1370–2285)	0.8
Total treatment effects in 2000		25 765	194 145 (142 505–259 225)	100

[a]All deaths prevented or postponed and life-years gained estimates were rounded to the nearest 5.

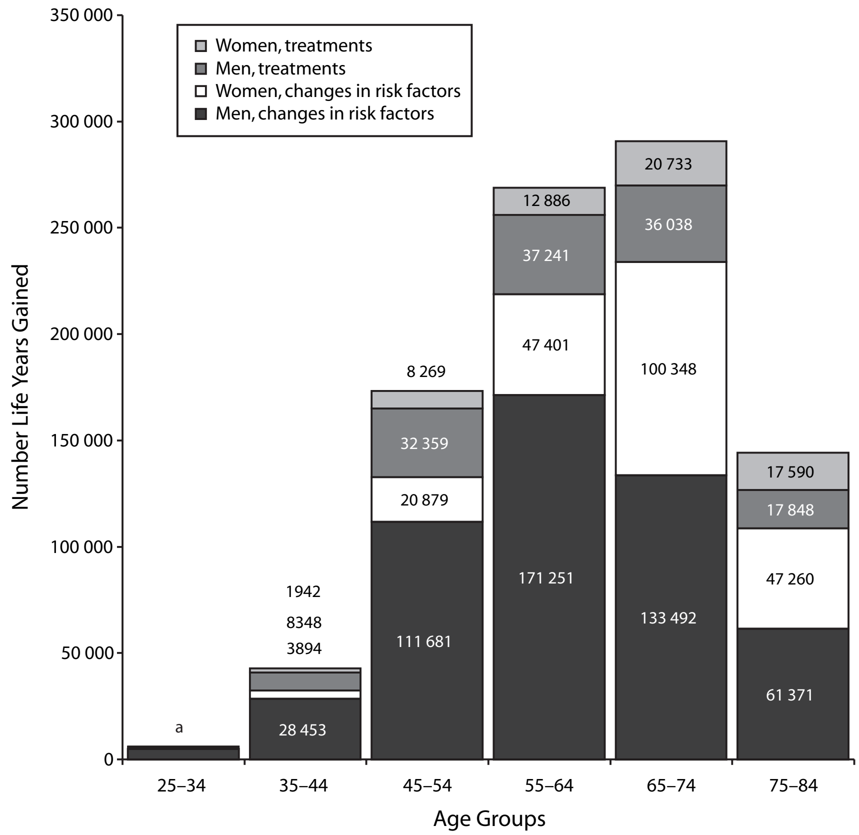
Life-Years Gained From Risk Factor Changes in the Population

In England and Wales, approximately 35 830 deaths were prevented or postponed as a result of risk factor changes in the population between 1981 and 2000. We therefore estimated that risk factor changes accounted for approximately 731 270 life-years gained (minimum 602 695, maximum 879 430) and represented 79% of all life-years gained in 2000. The largest contribution came from reductions in smoking (54%) and high blood pressure (28%) (Table 2).

Adverse trends between 1981 and 2000 were seen for obesity, physical inactivity, and diabetes. Together, these factors caused approximately 7645 additional coronary heart disease deaths (Table 2), resulting in a change of approximately 92 640 life-years (range: [−68 355]–[−100 770]), effectively halving the gain from population cholesterol reductions (Table 2).

Age and Gender Distribution of Life-Years Gained

The majority of life-years were gained among individuals aged 55–74 years. More life-years were gained by men than by women in all age groups; 68% (life-years gained in men divided by life-years gained in men and women: 132 505/194 145) of the life-years gained were from medical and surgical treatments, and 69% (life-years gained in men divided by life-years gained in men and women: 510 915/731 270) of the life-years gained were from risk factor reductions (Figure 1).



[a]Changes in risk factors gained 4 665 life-years in men, 574 in women; treatments gained 672 life-years in men, 219 in women.

FIGURE 1—Life-years gained from coronary heart disease treatments and changes in population risk factors, by age and sex in England and Wales, 1981–2000.

TABLE 2—Life-Years Gained From Changes in Population Cardiovascular Risk Factors: England and Wales, 1981–2000

Population Risk Factors	Relative Change in Risk Factor, 1981–2000, %	No. of Deaths Prevented or Postponed[a]	Life-Years Gained,[a] Best Estimate (Range)	Proportion of Total Life-Years Gained From Risk Factor Changes,%
Smoking	−34.0	29 715	398 080 (304 020–446 260)	54.4
Blood pressure	−7.5	5870	207 525 (197 870–288 445)	28.4
Cholesterol	−5.6	7900	164,305 (128,310–188 145)	22.5
Socioeconomic deprivation	−6.6	2125	53 995 (40 845–57 350)	7.4
Obesity	186.2	−2095	−10 690 ([−8565]–[−13 470])	−1.5
Physical activity	−30.6	−2660	−37 055 ([−27 245]–[−39 450])	−5.1
Diabetes	65.6	−2890	−44,895 ([−32 545]–[−47 850])	−6.1
Total risk factor effects in 2000		35 830	731 270 (602 695–879 430)	100.0

[a]All model estimates were rounded to the nearest 5.

Sensitivity Analyses

The relative contributions from treatments and risk factor reductions remained relatively constant, regardless of whether best, maximum, or minimum estimates were considered (Figure 2).

DISCUSSION

Coronary heart disease mortality rates in England and Wales decreased by 50% between 1981 and 2000, and this decrease resulted in approximately 70 000 fewer deaths. Our model estimated 61 595 deaths pre-

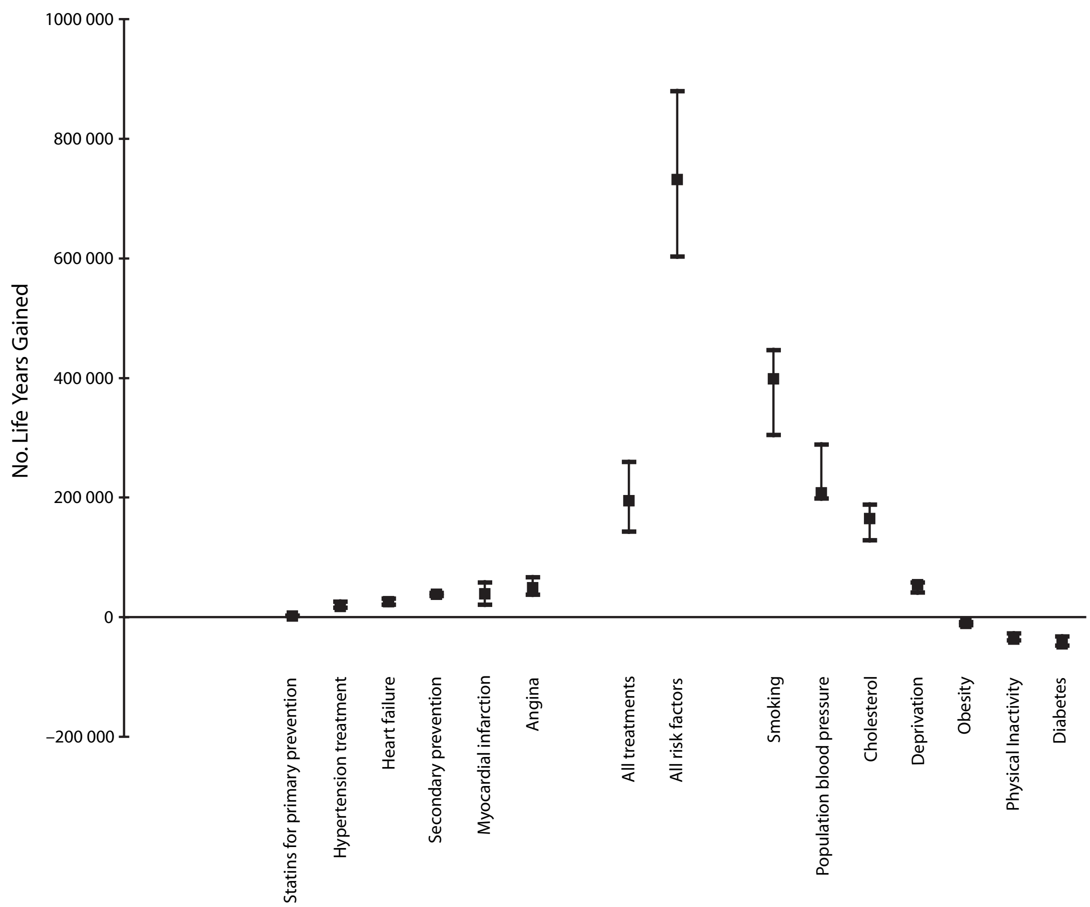


FIGURE 2—Proportional contributions of specific treatments and risk-factor changes to the decline in coronary heart disease in England and Wales, 1981–2000.

vented or postponed, which represents almost 1 million additional years of life gained. Each death avoided by treating a patient with recognized coronary heart disease thus yielded an average additional 7.5 years of life (total life-years gained divided by total deaths prevented or postponed). By contrast, each death avoided by a risk factor reduction yielded an average additional 20 years of life. Even more years of life were gained by younger individuals. These findings are generally consistent with those of previous studies.[22]

Medical and surgical treatments gained approximately 195 000 life-years in 2000. Much of this gain came from 4 categories: secondary prevention, hypertension, angina, and heart failure. The life-years gained from angiotensin-converting enzyme inhibitors, beta=blockers, and spironolactone were particularly impressive, given the relatively low prescribing rates in 2000 and the high case fatality rate in heart failure patients.[23] This evidence further emphasizes that simple, inexpensive treatments applied to all eligible patients can potentially produce huge gains.[24] Conversely, whereas the substantial resources devoted to revascularization in 2000 undoubtedly improved quality of life, the resulting gains in life-years were relatively modest (Table 1).

Risk factor reductions accounted for a massive 79% of the total life-years gained in 2000. Gains would have been even greater were it not for the adverse trends in physical activity, obesity, and diabetes. These risk factors represent a major public health target for coronary heart disease in the new millennium.[25] Substantial gains came from the reduction in smoking. These data highlight the substantial benefits gained from smoking cessation,[26] as well as the longer-term gains potentially achievable from primary prevention in children. The recent UK abolition of tobacco advertising will be valuable.[27] However, additional measures will remain essential, particularly for populations with low income and limited access to health services.[28]

This is the first published comprehensive coronary heart disease mortality model for England and Wales. However, the results of our mortality analyses are consistent with findings from other studies conducted in Europe,[29] New Zealand,[8] Scotland,[11] and the United States.[30] Bunker examined the 7.1-

year increase in life expectancy seen in the United States between 1950 and 1989.[31] Changes in coronary and cerebrovascular disease death rates accounted for 10%–20% of this increase.[31] Tsevat et al. attributed a 1.0- to 1.2-year increase in population life expectancy to the lowering of blood pressure among all men and a 0.5- to 1.2-year increase to smoking cessation among 35-year old men.[19] Using similar assumptions, Grover et al. estimated that reductions in coronary heart disease and stroke risk through blood pressure reduction would result in a 0.9- to 1.2-year increase in life-years in men aged 40 years and a 0.6- to 1.3-year increase in women aged 40 years.[22] All such modeling studies have limitations, being dependent on the variable quality and extent of data available on coronary heart disease patients, treatment uptake rates, and risk factor trends.[32] Assumptions must be made, and a robust sensitivity analysis therefore becomes essential.[21] Our model included only patients aged 84 years and younger, because data for older subjects were severely limited in quality and quantity. We considered deaths from coronary heart disease and ignored the possibility of "competing causes" such as cancer.[4] However, such competing cause effects are likely to be small,[19,20] and reductions in smoking would actually decrease deaths from lung and other cancers.[3,12,33]

The IMPACT model also assumed that estimates of efficacy from randomized controlled trials can be generalized to effectiveness in clinical practice, regardless of the baseline level of risk. This assumption appears reasonable.[34] Finally, risk factor lag times were not explicitly considered. However, they may be relatively unimportant over a 20-year analysis, because mortality reduction occurs relatively quickly—within 1–5 years of quitting smoking or reducing cholesterol.[26,35]

In conclusion, modern cardiological treatments in England and Wales produced gains in many thousands of life-years in 2000. However, 4 times that number of life-years was generated by relatively modest reductions in major risk factors, principally smoking, cholesterol, and blood pressure levels. Therefore, effective policies to promote healthy diets and control tobacco use might yield substantial additional years of life. These findings may be generalizable to the United States and other comparable industrialized countries. ■

About the Authors

At the time of the study, B. Ünal, J.A. Critchley, and S. Capewell were with the Department of Public Health, University of Liverpool, UK. D. Fidan is with the London School of Hygiene and Tropical Medicine, London, UK.

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Contributors

B. Ünal developed the protocol, collected the data, critically reviewed the data, built the IMPACT model for England and Wales, analyzed and interpreted the results, and wrote the article. J.A. Critchley contributed to the conception and design of the study and the building of the IMPACT model for England and Wales, critically reviewed the data and interpreted the results, and revised the article. D. Fidan contributed to the conception and design of the study, critically reviewed the data and interpreted the results, and revised the article. S. Capewell built the original IMPACT model and supervised its adaptation for England and Wales, contributed to the conception and design of the study, acquired and critically reviewed the data, analyzed and interpreted the results, and revised and contributed to the article.

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Human Participant Protection

No protocol approval was needed for this study, as no human participants were involved in this study.

References

1. Office for National Statistics. *Annual Abstract of Statistics.* London, UK: The Stationery Office; 2002.

2. Sackett DL. Evidence-based medicine and treatment choices. *Lancet.* 1997;349:570–573.

3. Fichtenberg C, Glantz S. Association of the California Tobacco Control Program with declines in cigarette consumption and mortality from heart disease. *N Engl J Med.* 2001;343:1772–1777.

4. McGovern PG, Jacobs DR, Shahar E, et al. Trends in acute coronary heart disease mortality, morbidity, and medical care from 1985 through 1997: the Minnesota heart survey. *Circulation.* 2001;104:19–24.

5. Critchley J, Capewell S. Why model coronary heart disease? *Eur Heart J.* 2002;23:110–116.

6. Beaglehole R. Medical management and the decline in mortality from coronary heart disease. *BMJ.* 1986;292:33–35.

7. Hunink MG, Goldman L, Tosteson AN, et al. The recent decline in mortality from coronary heart disease, 1980–1990. The effect of secular trends in risk factors and treatment. *JAMA.* 1997;277:535–542.

8. Capewell S, Beaglehole R, Seddon M, McMurray J. Explaining the decline in coronary heart disease mortality in Auckland, New Zealand between 1982 and 1993. *Circulation.* 2000;102:1511–1516.

9. Capewell S, Morrison CE, McMurray JJ. Contribution of modern cardiovascular treatment and risk factor changes to the decline in coronary heart disease mortality in Scotland between 1975 and 1994. *Heart.* 1999;81:380–386.

10. Ünal B, Critchley J, Capewell S. Explaining the decline in coronary heart disease mortality in England and Wales, 1981–2000. *Circulation.* 2004;109:1101–1107.

11. Critchley JA, Capewell S, Ünal B. Life-years gained from coronary heart disease mortality reduction in Scotland: prevention or treatment? *J Clin Epidemiol.* 2003;56:583–590.

12. Tunstall-Pedoe H, Kuulasmaa K, Mahonen M, Tolonen H, Ruokokoski E, Amouyel P. Contribution of trends in survival and coronary-event rates to changes in coronary heart disease mortality: 10-year results from 37 WHO MONICA project populations. Monitoring trends and determinants in cardiovascular disease. *Lancet.* 1999;353:1547–1557.

13. van den Hoogen PC, Feskens EJ, Nagelkerke NJ, Menotti A, Nissinen A, Kromhout D. The relation between blood pressure and mortality due to coronary heart disease among men in different parts of the world. Seven Countries Study Research Group. *N Engl J Med.* 2000;342:1–8.

14. Capewell S, Livingston BM, MacIntyre K, et al. Trends in case-fatality in 117 718 patients admitted with acute myocardial infarction in Scotland. *Eur Heart J.* 2000;21:1833–1840.

15. MacIntyre K, Capewell S, Stewart S, et al. Evidence of improving prognosis in heart failure. Trends in case fatality in 66 547 patients hospitalized between 1986 and 1995. *Circulation.* 2000;102:1126–1131.

16. Isles CG, Walker LM, Beevers GD, et al. Mortality in patients of the Glasgow Blood Pressure Clinic. *J Hypertens.* 1986;4:141–156.

17. Pell JP, MacIntyre K, Walsh D, et al. Time trends in survival and readmission following coronary artery bypass grafting in Scotland, 1981–96: retrospective observational study. *BMJ.* 2002;324:201–202.

18. Pocock SJ, Henderson RA, Rickards AF, et al. Meta-analysis of randomised trials comparing coronary angioplasty with bypass surgery. *Lancet.* 1995;346:1184–1189.

19. Tsevat J, Weinstein MC, Williams LW, Tosteson AN, Goldman L. Expected gains in life expectancy from various coronary heart disease risk factor modifications. *Circulation.* 1991;83:1194–1201.

20. Yudkin JS. How can we best prolong life? Benefits of coronary risk factor reduction in non-diabetic and diabetic subjects. *BMJ.* 1993;306:1313–1318.

21. Briggs A, Sculpher M, Buxton M. Uncertainty in the economic evaluation of health care technologies: the role of sensitivity analysis. *Health Econ.* 1994;3:95–104.

22. Grover SA, Paquet S, Levinton C, Coupal L, Zowall H. Estimating the benefits of modifying risk factors of cardiovascular disease: a comparison of primary vs secondary prevention. *Arch Intern Med.* 1998;158:655–662.

23. Stewart S, Jenkind A, Buchan S, et al. The current cost of heart failure to the National Health Service in the UK. *Eur J Heart Fail.* 2002;4:361–371.

24. Department of Health. National Service Framework for Coronary Heart Disease. 2000. Available at: http://www.doh.gov.uk/pdfs/chdnsf.pdf. Accessed September 12, 2003.

25. Department of Health. *Saving Lives: Our Healthier Nation.* London, UK: The Stationery Office; 1999.

26. Critchley J, Capewell S. Mortality risk reduction associated with smoking cessation in patients with coronary heart disease: a systematic review. *JAMA.* 2003; 290:86–97.

27. The Tobacco Advertising and Promotion Act 2002. Chapter 36 (2002).

28. *Smoking Kills: A White Paper on Tobacco.* London, UK: The Stationery Office; 1998.

29. Bots ML, Grobbee DE. Decline of coronary heart disease mortality in The Netherlands from 1978 to 1985: contribution of medical care and changes over time in presence of major cardiovascular risk factors. *J Cardiovasc Risk.* 1996;3:271–276.

30. Goldman L, Cook E. The decline in ischemic heart disease mortality rates. An analysis of the comparative effects of medical interventions and changes in lifestyle. *Ann Intern Med.* 1984;101:825–836.

31. Bunker JP. The role of medical care in contributing to health improvements within societies. *Int J Epidemiol.* 2001;30:1260–1263.

32. Ünal B, Critchley J, Capewell S. Missing, mediocre, or merely obsolete? An evaluation of UK data sources for coronary heart disease. *J Epidemiol Community Health.* 2003;57:530–535.

33. British Heart Foundation Statistics Database. Coronary Heart Disease Statistics. 2002. Available at: http://www.bhf.org.uk/professionals/uploaded/bhf%20heartstats%202003%20-%204-page.pdf. Accessed September 12, 2003.

34. McAlister FA. Commentary: relative treatment effects are consistent across the spectrum of underlying risks, usually. *Int J Epidemiol.* 2002;31:76–77.

35. Law MR, Wald NJ, Thompson SG. By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease? *BMJ.* 1994;308:367–372.