

Modelling the decline in coronary heart disease deaths in England and Wales, 1981-2000: comparing contributions from primary prevention and secondary prevention

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Abstract

Objective To investigate whether population based primary prevention (risk factor reduction in apparently healthy people) might be more powerful than current government initiatives favouring risk factor reduction in patients with coronary heart disease (CHD) (secondary prevention).

Design, setting, and participants The IMPACT model was used to synthesise data for England and Wales describing CHD patient numbers, uptake of specific treatments, trends in major cardiovascular risk factors, and the mortality benefits of these specific risk factor changes in healthy people and in CHD patients.

Results Between 1981 and 2000, CHD mortality rates fell by 54%, resulting in 68 230 fewer deaths in 2000. Overall smoking prevalence declined by 35% between 1981 and 2000, resulting in approximately 29 715 (minimum estimate 20 035, maximum estimate 44 675) fewer deaths attributable to smoking cessation: approximately 5035 in known CHD patients and approximately 24 680 in healthy people. Population total cholesterol concentrations fell by 4.2%, resulting in approximately 5770 fewer deaths attributable to dietary changes (1205 in CHD patients and 4565 in healthy people) plus 2135 fewer deaths attributable to statin treatment (1990 in CHD patients, 145 in people without CHD). Mean population blood pressure fell by 7.7%, resulting in approximately 5870 fewer deaths attributable to secular falls in blood pressure (520 in CHD patients and 5345 in healthy people) plus approximately 1890 fewer deaths attributable to antihypertensive treatments in people without CHD. Approximately 45 370 fewer deaths were thus attributable to reductions in the three major risk factors in the population: some 36 625 (81%) in people without recognised CHD and 8745 (19%) in CHD patients.

Conclusions Compared with secondary prevention, primary prevention achieved a fourfold larger reduction in deaths. Future CHD policies should prioritise population-wide tobacco control and healthier diets.

Introduction

Coronary heart disease (CHD) remains the largest cause of death in the United States, Europe, and Australasia.¹ However, since the 1980s, CHD mortality rates have halved in Britain and in many industrialised countries.¹ Studies in the US, Europe, and New Zealand consistently suggest that 50-75% of the falls in cardiac deaths can be attributed to population-wide improvements in the major risk factors, particularly smoking, cholesterol, and

blood pressure.²⁻⁵ Modern cardiological treatments for known CHD patients, such as thrombolysis, aspirin, angiotensin converting enzyme inhibitors, statins, and coronary artery bypass surgery, generally explain the remaining 25-50% of the fall in mortality.²⁻⁵

Risk factor reduction should thus be a central component of all CHD policies. However, disagreement continues about whether to prioritise risk factor reduction across the whole population, explicitly including all apparently healthy people (primary prevention), or mainly to target CHD patients (secondary prevention). Current funding in the US⁶ and the United Kingdom clearly favours secondary prevention. This reflects four perceived limitations of primary prevention as an "evidence based" intervention. Firstly, a much quoted Cochrane meta-analysis of 10 intervention studies in primary care that used counselling or education to modify more than one cardiovascular risk factor in adults found only modest changes in risk factors and no effect on mortality.⁷ Secondly, although confident about prescribing drugs, some clinicians may feel uncomfortable in a health promotion role, perhaps lacking the confidence or skills to influence complex behaviours such as diet or smoking.⁸ Thirdly, the very long time scales apparently involved may be a deterrent.⁹ Fourthly, the numbers needed to treat to prevent one event are substantial, often fivefold higher than those for targeting CHD patients.¹⁰

In truth, both primary and secondary prevention interventions are probably necessary to maximise population health.¹¹ Quantifying their relative contributions is clearly important but is difficult to do with disease registers or population cohorts.¹² Researchers have therefore used models to quantify the potential contribution of risk factor reductions before and after CHD is diagnosed in an individual. For example, use of the CHD policy model suggested that approximately 25% of the decline in CHD deaths in the US between 1980 and 1990 was explained by primary prevention and slightly more (29%) was explained by risk factor reductions in patients with CHD.²

A better understanding of the relative contributions of primary prevention and secondary prevention to the recent falls in deaths from CHD is clearly essential, in order to inform future CHD policy options in Britain and elsewhere.¹⁰ We have therefore used the only validated and comprehensive CHD model available in the UK.⁴ We analysed the decrease in CHD mortality in England and Wales between 1981 and 2000 to estimate the proportions attributable to changes in major cardiovascular risk factors in apparently healthy people (primary prevention) and in patients with CHD (secondary prevention).

Methods

IMPACT CHD model

The cell based IMPACT CHD mortality model, previously validated in Scotland,³ New Zealand,⁵ and Beijing,¹³ has been described elsewhere.^{3 5 14} Briefly, we used the model to synthesise data for the adult population of England and Wales—35.5 million people aged between 25 and 84—describing numbers of CHD patients, uptake of specific treatments, trends in major cardiovascular risk factors in apparently healthy people in populations and in specific patient groups, and the effectiveness (mortality benefits) of the reductions in specific risk factors in people with and without recognised CHD.⁴

Data sources

Sources of data included national surveys, official statistics, clinical audits, controlled trials, and meta-analyses. These are detailed on our website.¹⁵

Primary prevention: risk factor trends and mortality benefits in the general population

For risk factor changes, the model uses regression (β) coefficients obtained from large meta-analyses, cohort studies, and MONICA analyses (appendix 10 on website).^{4 15} Each β coefficient quantifies the independent relation between population change in a specific CHD risk factor (such as smoking, cholesterol, or blood pressure) and the consequent percentage change in population mortality from CHD. We then estimated the subsequent reduction in the number of deaths produced by the decrease in each major risk factor as the product of three variables: the number of CHD deaths observed in the base year (1981), the relative reduction in that risk factor, and the β coefficient, each stratified by age and sex.^{4 15}

To estimate the impact of the population-wide reduction in cholesterol due to dietary change, we subtracted the estimated effect of statins for primary prevention from the overall number of deaths prevented or postponed in the population due to change in mean cholesterol concentration. We explicitly considered demographic change by using age and sex specific population CHD mortality and CHD patient numbers for 1981 and for 2000.⁴

Secondary prevention: risk factor trends and mortality benefits in CHD patients

We then estimated the mortality benefit attributable to reductions in each major risk factor (smoking, total cholesterol, and blood pressure) in each group of CHD patients as the number of deaths prevented or postponed.⁴ We categorised CHD patients according to disease groups: acute myocardial infarction, survivors of myocardial infarction, revascularisation patients, and patients with unstable angina, chronic angina, and chronic heart failure. We did not consider the impact of changes in risk factors in patients with an initial acute myocardial infarction or unstable angina, because both are transient states. To avoid double counting, we firstly made adjustments for overlaps between different treatment groups by subtracting the overlapping subgroup from the main group, as detailed in appendix 8 on the website.^{4 15}

We based age and sex specific smoking cessation rates on local surveys and audits. We initially assumed that age and sex specific changes in cholesterol attributable to diet and changes in blood pressure attributable to population secular trends would mirror the changes seen in the general population. We then used rigorous sensitivity analyses to test the effect of much smaller (–50%) and much larger (+50%) changes.

Statins and other treatments

The model aimed to include all medical and surgical treatments provided in 2000. This included statins as primary prevention (in people without recognised CHD) and as secondary prevention (in CHD patients). We calculated the absolute reduction in mortality by using the relative reduction in mortality reported in the most recent meta-analysis¹⁵ applied to the age specific case fatality rate observed in unselected patient cohorts. The effect of all other “secondary prevention” drugs (aspirin, β blockers, and angiotensin converting enzyme inhibitors) has been previously reported and was explicitly excluded from this analysis.⁴ We did not consider over the counter statins, as these only became available in 2003.

We estimated the number of deaths prevented or postponed for specific age and sex groups. We used survival benefit over a one year time interval throughout.

Sensitivity analyses

Because of the uncertainties surrounding some values, we did a multiway sensitivity analysis using the analysis of extremes method.¹⁶ Illustrative examples are shown in appendix 7 on the website.¹⁵

Apportioning deaths prevented or postponed between primary and secondary prevention

We then estimated the deaths prevented or postponed in apparently healthy people as the deaths prevented or postponed in the entire population minus the deaths prevented or postponed in each CHD patient group. Illustrative examples are given in the results section.

Results

Fall in CHD mortality between 1981 and 2000

Between 1981 and 2000, age specific CHD mortality in England and Wales fell by 62% in men and 45% in women aged 25–84. This resulted in 68 230 fewer deaths in 2000, compared with the 1981 baseline.⁴

Medical and surgical treatments together prevented or postponed approximately 25 805 deaths (minimum estimate 17 110, maximum estimate 49 040). This represented 42% of the total decrease in CHD deaths estimated by the model.⁴

Approximately 58% of the fall in mortality was attributable to reductions in the risk factors—mainly smoking, cholesterol, and blood pressure.⁴ Changes in the three major cardiovascular risk factors together produced a best estimate of 45 370 (29 570–76 835) fewer deaths. In contrast, adverse trends in diabetes, obesity, and physical activity together generated approximately 7645 (5395–10 730) additional deaths.⁴

Risk factor reductions in the general population and in CHD patients

Overall smoking prevalence fell by 35% between 1981 and 2000. This resulted in approximately 29 715 (20 035–44 675) fewer deaths. Approximately 5035 (17%; 3100–8255) fewer deaths resulted from smoking cessation in CHD patients, leaving the remainder—approximately 24 680 (83%; 16 935–36 420)—attributable to reduced smoking prevalence in “healthy people” (table 1).

Population total cholesterol concentrations fell by 4.2% between 1981 and 2000, resulting in approximately 7900 (5285–16 695) fewer deaths. Approximately 5770 fewer deaths were attributable to dietary reduction—approximately 1205 (645–2455) in CHD patients, leaving some 4565 (3290–9650) fewer deaths attributable to dietary cholesterol reduction in healthy

Table 1 Fall in coronary heart disease mortality attributable to changes in risk factors in people with and without recognised coronary heart disease: England and Wales, 1981-2000

Risk factor	Change in population risk factor level (%)	Deaths prevented or postponed				Total
		Primary prevention*		Secondary prevention*		
		No (min-max)†	%	No (min-max)‡	%	
Smoking:						
Overall	-35	24 680 (16 935-36 420)	83	5 035 (3 100-8 255)	17	29 715 (20 035-44 675)
Cholesterol:						
Overall	-4.2	4 710 (3 335-10 060)	60	3 190 (1 950-6 635)	40	7 900 (5 285-16 695)
Attributable to diet		4 565 (3 290-9 650)		1 205 (645-2 455)		5 770 (3 935-12 105)
Attributable to statins		145 (45-410)		1 990 (1 305-4 180)		2 135 (1 350-4 590)
Blood pressure:						
Overall	-7.7	7 235 (3 965-14 525)	93	520 (285-940)	7	7 755 (4 250-15 465)
Secular trend		5 345 (3 125-11 740)		520 (285-940)		5 870 (3 410-12 680)
Antihypertensive treatment		1 890 (840-2 785)		‡		1 890 (875-3 165)
All three major risk factors		36 625 (24 235-61 005)	81	8 745 (5 335-15 830)	19	45 370 (29 570-76 835)

*Primary prevention=in healthy people; secondary prevention=in people with coronary heart disease.

†All numbers rounded to nearest 5; min-max=minimum and maximum estimates.

‡Antihypertensive treatment in coronary heart disease patients was subsumed in secondary prevention medication component of IMPACT model.

people. Approximately 2135 (1350-4590) fewer deaths were attributable to statin treatments: 1990 (1305-4180) in CHD patients and 145 (45-410) in healthy people (table 1).

Mean population diastolic blood pressure fell by 7.7% between 1981 and 2000, resulting in approximately 7755 (4250-15 465) fewer deaths. Approximately 520 (285-940) fewer deaths were attributable to the secular fall in blood pressure in CHD patients, leaving some 5345 (3125-11 740) in healthy people, and 1890 (840-2785) fewer deaths were attributable to antihypertensive treatments in around 7.1 million hypertensive people (table 1).⁴

All secondary prevention interventions (risk factor reductions in CHD patients) together accounted for approximately 8745 (5335-15 830) fewer deaths (table 1). This represented 19% of the total number of 45 370 deaths prevented or postponed by change in the three major risk factors between 1981 and 2000 (minimum contribution 18.0%, maximum contribution 21.0%).

The remaining reduction in deaths (45 370 minus 8745) could then be attributed to primary prevention in healthy people, which thus accounted for approximately 36 625

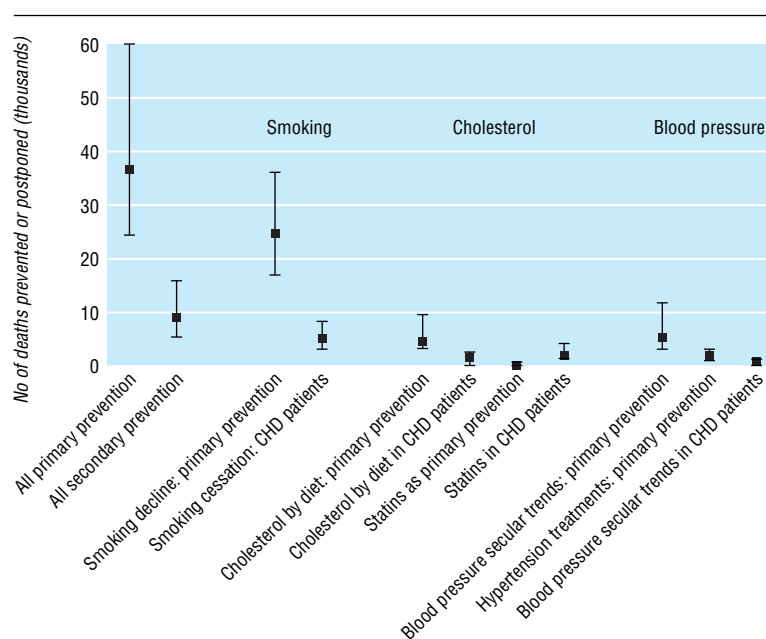
(24 235-61 005) fewer deaths (table 1). This 36 625 represented 81% of the total mortality decrease of 45 370 deaths prevented or postponed by change in the three major risk factors between 1981 and 2000 (minimum contribution 53.4%, maximum contribution 79.3%).

Sensitivity analyses

The relative contribution to the overall decline in CHD deaths from primary and secondary prevention for each risk factor was little changed by whether best, minimum, or maximum estimates were considered (figure).

Risk factor reduction benefits in specific groups of CHD patients

In 2000, approximately 30 530 (18 630-54 180) deaths were prevented or postponed in patients with CHD. Some 23 770 (77.9%) were attributable to medical and surgical treatments, and 6760 (22.1%) were attributable to reductions in the three major risk factors. Substantial contributions came from three patient groups: post-myocardial infarction and post-surgical intervention groups, angina in the community, and heart failure (table 2).



Sensitivity analysis comparing primary and secondary prevention: falls in coronary heart disease mortality attributable to changes in risk factors in people with and without recognised coronary heart disease in England and Wales, 1981-2000 (best estimate, with minimum and maximum estimates). CHD=coronary heart disease

Table 2 Fall in coronary heart disease mortality in England and Wales between 1981 and 2000: numbers of deaths prevented or postponed by treatments and by changes in risk factors in people with coronary heart disease, categorised into specific groups

Patient groups	No of deaths prevented or postponed*	Minimum and maximum estimates	Percentage
After acute myocardial infarction			
All treatments:	3 840	2 865-5 080	66.5
Statins	460	430-1 340	8.0†
Smoking cessation	1 450	880-2 315	25.1
Cholesterol reduction (diet)	335	250-560	5.8
Blood pressure reduction	140	70-230	2.4
Total	5 765	4 060-8 185	100
After CABG surgery or angioplasty			
All treatments:	3 055	1 740-7 620	74.0
Statins	675	380-1 495	16.3†
Smoking cessation	820	510-1 150	19.8
Cholesterol reduction (diet)	195	65-320	4.7
Blood pressure reduction	60	40-130	1.4
Total	4 130	2 355-9 225	100
Angina			
Revascularisation	2 320	1 185-3 495	100
Chronic angina in the community			
All treatments:	1 105	720-2 395	43.6
Statins	105	95-275	4.2†
Smoking cessation	1 115	715-1 605	44.0
Cholesterol reduction (diet)	200	95-460	7.5
Blood pressure reduction	115	75-170	4.5
Total	2 535	1 605-4 630	100
Acute myocardial infarction			
All treatments	4 780	3 225-8 290	100
Unstable angina			
All treatments	910	620-1 620	100
Heart failure requiring hospital admission			
All treatments:	4 535	2 295-7 680	85.2
Statins	415	290-575	7.7†
Smoking cessation	595	400-1 025	11.2
Cholesterol reduction (diet)	130	55-265	2.5
Blood pressure reduction	60	15-97	1.2
Total	5 325	2 765-9 070	100
Heart failure in the community			
All treatments:	3 220	1 950-6 345	67.6
Statins	335	110-490	7.0†
Smoking cessation	1 055	600-2 160	22.1
Cholesterol reduction (diet)	350	175-845	7.3
Blood pressure reduction	140	85-310	3.0
Total	4 770	2 810-9 665	100
All groups			
All treatments:	23 770‡	14 600-42 530	77.9
Statins	1 990	1 305-4 180	6.5†
Smoking cessation	5 035	3 100-8 255	16.5
Cholesterol reduction (diet)	1 205	645-2 455	3.9
Blood pressure reduction	520	285-940	1.7
Total	30 530	18 630-54 180	100

CABG=coronary artery bypass graft.

*All numbers rounded to nearest 5, so sums may differ from totals in table 1.

†Statin percentages already included within treatments.

‡This total mirrors the treatment effect (25 805) reported in our previous publication,¹⁵ which included antihypertensive treatment in people without coronary heart disease (1888) and statin treatment as primary prevention (145).

Approximately 1990 (1305-4180) fewer deaths were attributable to statin treatments in CHD patients. The biggest contributions came from statin treatment for patients after acute myocardial infarction (460), revascularisation (675), or heart failure (750) (table 2).

Risk factor reduction benefits by age and sex

Of the 45 370 (29 570-76 835) deaths prevented or postponed by reductions in risk factors, 73.7% occurred in men and 26.3% in women (table 3). The relative contribution from secondary prevention was consistently higher in women (27.3%) than in men (16.4%). The contribution from secondary prevention was relatively consistent across age groups in men, but in women this contribution was higher in the youngest and oldest groups than in those in between (table 3).

Discussion

Impact of primary compared with secondary prevention

Mortality from coronary heart disease in England and Wales fell by 54% between 1981 and 2000.^{1 4} Approximately half of this large fall could be attributed to primary prevention, defined as reductions in the three major risk factors in people without recognised CHD. Furthermore, primary prevention had a fourfold greater impact than secondary prevention (risk factor reductions in CHD patients). This was much as predicted by Rose and others.^{11 12} The fourfold advantage apparently contrasted with the 25% to 29% split estimated by using the CHD policy model for the US population 1980-90.² However, the difference may substantially reflect a different categorisation of primary versus secondary prevention rather than true differences between populations.

The fourfold advantage of primary prevention becomes 12-fold greater when life years gained are considered, rather than simply deaths postponed.¹⁴ We have previously shown that a death prevented or postponed in a patient with recognised CHD gained an additional 7.5 years of life, on average. In contrast, each death prevented or postponed by primary prevention gained an additional 21 years of life, on average.¹⁴

Contributions of individual risk factors

The biggest single contribution reflected a large decrease in overall smoking prevalence, from 39% in 1981 to 28% in 2000.¹ This resulted in approximately 29 715 fewer deaths, of which 83% were in "healthy people" and 17% resulted from smoking cessation in CHD patients. The evidence base is now particularly solid for smoking cessation, with 50 year data from the Doll cohort and the recent Cochrane meta-analysis showing a 36% reduction in mortality in CHD patients who stop smoking.^{17 18} However, opportunities for smoking cessation in secondary prevention are frustratingly limited, as around 50% of myocardial infarctions are rapidly fatal.¹⁹ It therefore makes sense to energetically target smokers before they develop clinical disease. In Britain, substantial resources are now being spent on smoking cessation services, and four week results look promising. However, there is no room for complacency; subsequent relapse is common, and only a minority of all smokers have yet been reached. Furthermore, although the recent advertising ban is welcome, further tobacco control measures must be implemented.

Primary prevention also had an almost fourfold bigger impact on mortality than did secondary prevention for dietary based cholesterol reduction (some 4565 fewer deaths in healthy people compared with 1205 in CHD patients) and a 10-fold bigger impact through blood pressure reduction (5345 fewer deaths in healthy people compared with 520 in patients with CHD). Antihypertensive treatments contributed somewhat fewer deaths—only about 1890.

UK population levels of both cholesterol and blood pressure declined modestly between 1981 and 2000. The factors underlying these falls are clearly complex. Having explicitly considered

Table 3 Numbers of deaths prevented or postponed* by reductions in risk factors: relative contributions from primary prevention and secondary prevention, by age and sex. Values are numbers (percentages)

	Age groups (years)						Total
	25-34	35-44	45-54	55-64	65-74	75-84	
Men							
Overall	85	815	3 635	7 750	10 835	10 320	33 440
Secondary prevention	10 (11.8)	115 (14.1)	545 (15.0)	1 460 (18.8)	1 875 (17.3)	1 485 (14.4)	5 490 (16.4)
Primary prevention	75 (88.2)	705 (86.5)	3 090 (85.0)	6 285 (81.1)	8 960 (82.7)	8 835 (85.6)	27 950 (83.6)
Women							
Overall	10	95	510	2 400	5 085	3 835	11 930
Secondary prevention	5 (50.0)	35 (36.8)	175 (34.3)	560 (23.3)	1 120 (22.0)	1 365 (35.6)	3 255 (27.3)
Primary prevention	5 (50.0)	60 (63.2)	335 (65.7)	1 840 (76.7)	3 970 (78.0)	2 470 (64.4)	8 675 (72.7)

*All numbers rounded to nearest 5.

therapeutic interventions in our model, we then attributed the remainder of the cholesterol and blood pressure declines to life-style changes; this could be either physical activity or diet. Because the proportion of people who were moderately physical active probably decreased slightly,⁴ these falls in cholesterol concentrations might reasonably be cautiously attributed to the well documented positive trends in dietary intake of fruit, fibre, and unsaturated fats.¹ Reduced saturated fat and salt intake will both have benefited population blood pressure.^{1 20}

Effectiveness of population approach

Substantial and sustained change in dietary habits is hard to achieve in individuals.²¹ In contrast, substantial 0.5-1.0 mmol/l falls in average total cholesterol have been reported in entire populations in Finland and Mauritius.^{22 23} These almost certainly reflect a combination of factors: policy, finance, health promotion, and multisectoral collaboration, as well as advice to individuals.^{22 23}

These findings support the population prevention approach.¹¹ It has been repeatedly shown that achieving apparently small mortality benefits across the entire population would produce far larger overall gains than merely targeting people at high risk in order to achieve big reductions in mortality in a relatively small number of people.^{11 12}

The international evidence is also increasingly powerful. The most effective and cost effective interventions to reduce major risk factors have come from comprehensive cardiovascular strategies underpinned by robust national policies, as in Finland, Mauritius, and Singapore.^{1 7} The US has promoted an increasingly cholesterol conscious culture, with clear gains. Many American states now have complete public bans on smoking.²⁴ Ireland has followed this very successfully. Smoke-free campaigns in Liverpool, Birmingham, London, and Scotland are all gaining momentum, with further support from the recent public health white paper, which also proposes legislation to reduce saturated fat and salt in processed food.²⁰

Strengths and limitations of modelling studies

Modelling studies have several potential strengths. They can transparently integrate and simultaneously consider huge amounts of data from many sources. Explicit assumptions can then be tested by using sensitivity analyses. Modelling studies also have limitations. They are dependent on the variable quality and extent of data available on CHD risk factor trends and treatment uptakes. Rigorous sensitivity analyses are therefore essential.¹⁶ However, for each risk factor, the relative contribution of primary and secondary prevention to the overall decline in CHD deaths was reasonably consistent whether considering best, minimum, or maximum estimates (figure). Finally, lag times may be relatively unimportant over two decades. Substantial

mortality reductions occur within one or two years of reducing cholesterol or stopping smoking.¹⁸

Contribution of statin treatment

Approximately 1990 fewer deaths were attributable to statin treatment in CHD patients, and 145 to statins as primary prevention. This estimate is less than the recently quoted UK government figure of “7000 lives saved by statins in 2003.”¹⁰ However, although hospital discharge prescribing may be increasing, realistic assumptions about long term prescribing and compliance are essential.

Conclusions

Approximately half the recent large falls in CHD deaths in England and Wales can be attributed to primary prevention: reductions in the three major risk factors in people without recognised CHD. Much as predicted, primary prevention had a fourfold bigger impact on mortality than did secondary prevention. These findings might be cautiously generalisable to other comparable industrialised countries. Comprehensive CHD strategies should therefore focus on primary prevention, particularly tobacco control and healthier diets.

Contributors: BU developed the protocol; collected and critically reviewed the data; built the IMPACT model for England and Wales; analysed and interpreted the results; drafted and wrote the paper; finalised, approved, and submitted the paper; and is the guarantor. JAC 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,

What is already known on this topic

Coronary heart disease (CHD) mortality has halved since 1981 in the UK, resulting in 68 230 fewer deaths in 2000

Current government initiatives favour risk factor reduction in CHD patients (secondary prevention), but population based primary prevention (risk factor reduction in apparently healthy people) might be more powerful

What this paper adds

Approximately 45 370 fewer CHD deaths were attributable to reductions in smoking, cholesterol, and blood pressure in the whole population

Some 36 625 (81%) of these fewer deaths occurred in people without recognised CHD and 8745 (19%) in CHD patients

Compared with secondary prevention, primary prevention achieved a fourfold larger reduction in deaths

revised the paper, and approved the final version. SC 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, analysed and interpreted the results, revised and contributed to the paper, and approved the final version.

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