

Original Contribution

Explaining the Decline in Coronary Heart Disease Mortality in Finland between 1982 and 1997

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In Finland since the 1980s, coronary heart disease mortality has declined more than might be predicted by risk factor reductions alone. The aim of this study was to assess how much of the decline could be attributed to improved treatments and risk factor reductions. The authors used the cell-based IMPACT mortality model to synthesize effectiveness of treatments and risk factor reductions with data on treatments administered to patients and trends in cardiovascular risk factors in the population. Cardiovascular risk factors were measured in random samples of patients in 1982 ($n = 8,501$) and 1997 ($n = 4,500$). Mortality and treatment data were obtained from the National Causes of Death Register, Hospital Discharge Register, social insurance data, and medical records. Estimated and observed changes in coronary heart disease mortality were used as main outcome measures. Between 1982 and 1997, coronary heart disease mortality rates declined by 63%, with 373 fewer deaths in 1997 than expected from baseline mortality rates in 1982. Improved treatments explained approximately 23% of the mortality reduction, and risk factors explained some 53–72% of the reduction. These findings highlight the value of a comprehensive strategy that promotes primary prevention programs and actively supports secondary prevention. It also emphasizes the importance of maximizing population coverage of effective treatments.

cardiac surgical procedures; coronary disease; drug therapy; mortality; primary prevention; risk factors

Abbreviations: CABG, coronary artery bypass grafting; CHD, coronary heart disease.

Coronary heart disease (CHD) mortality has been declining in most developed countries since the 1960s (1). Only a few studies have estimated the extent to which changes in the major cardiovascular risk factors (blood pressure, serum cholesterol, and smoking) can explain the observed changes in mortality (2–4). In Finland, risk factor changes explained almost all of the decline in CHD mortality during the 1970s. However, in the late 1980s and 1990s, mortality declined much faster than might be expected simply from risk factor reductions. Between 1972 and 1992, altogether 80 percent

of the reduction in CHD mortality was explained by a decline in the major risk factors: smoking, cholesterol, and blood pressure. The biggest contribution was attributable to a large drop in cholesterol levels (3).

New cardiological treatments were increasingly used in the 1980s and 1990s both to treat CHD and to prevent complications. These therapies included coronary artery surgery, angioplasty, thrombolysis, statins, angiotensin-converting enzyme inhibitors, and other medications for secondary prevention and heart failure (5–10). In controlled clinical trials,

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all have been shown to reduce mortality, but how much have these treatments reduced mortality at the population level? This information is very important in terms of rational public health planning and health services development. However, the existing analyses are few and dated.

Few published studies have simultaneously considered both the role of risk factors and the impact of new treatments. Most of them consistently suggest that improvements in treatment explain less of the mortality decline than do risk factor reductions. For example, the proportion of the decline in mortality attributable to medical interventions was estimated as 40 percent in Auckland, New Zealand, between 1974 and 1981 (11) and 48 percent between 1982 and 1993 (12); 43 percent in the United States between 1980 and 1990 (13); and 46 percent in the Netherlands between 1978 and 1985 (14). More recently, IMPACT, a cell-based CHD mortality model, was developed in Britain and New Zealand to estimate the population impact of different treatments and risk factors (12, 15, 16). Results in both countries were very similar: just over half of the decline was attributable to risk factor changes and just under half to new treatments (40–48 percent).

We used this IMPACT model to examine how much of the decline in CHD mortality in Finland between 1982 and 1997 could be attributed to evidence-based medical and surgical treatments and how much to changes in major cardiovascular risk factors.

MATERIALS AND METHODS

Cardiovascular risk factors

Since 1972, risk factors in the Finnish population have been measured by using population risk factor surveys carried out with independent, random samples in 5-year intervals (17). The surveys were started in North Karelia and Kuopio provinces and were later expanded to cover six geographic areas of Finland. In this study, data from 1982 and 1997 were used from three areas of Finland—North Karelia province, Kuopio province, and southwest Finland—because these areas were included in both the 1982 and 1997 surveys. The total population aged 35–64 years in these areas was 221,892 in 1982 and 256,013 in 1997. Age- and sex-stratified random samples of persons aged 25–64 years were drawn from population registers. In this analysis, data were used for participants aged 35–64 years. The sample size in the three survey areas for this age group was 8,501 in 1982 and 4,500 in 1997. These Finnish risk factor surveys have been part of the World Health Organization (WHO) MONICA project (1982) and the National FINRISK Study (1997) (17–19).

CHD patients and uptake of cardiological treatments

Mortality data were obtained from the National Causes of Death Register. CHD deaths were defined by using *International Classification of Diseases*, Ninth Revision, codes 410–414 and *International Classification of Diseases*, Tenth Revision, codes I20–I25 as underlying causes of death. Morbidity data and prevalence rates for cardiac patients in the

survey areas were obtained from the Hospital Discharge Register, the National FINRISK Survey, the social insurance data, and a myocardial infarction register (the FINAMI register). The Hospital Discharge Register is a national database maintained by the National Research and Development Centre for Welfare and Health. The social insurance data, including information on all reimbursed medicine prescriptions, are collected and maintained by the Social Insurance Institution of Finland. The National Public Health Institute collects and maintains the FINAMI register (20) in the same three survey areas according to the WHO MONICA protocols (18).

Treatment data on cardiac patients were obtained from the National FINRISK Survey, the social insurance data, and a special database created for this survey by checking individual patients' medical records. Patients eligible for secondary prevention therapies after myocardial infarction, coronary artery bypass grafting (CABG) surgery, or angioplasty were determined by using the Hospital Discharge Register data and special registers on CABG surgery and angioplasties maintained by the Finnish Heart Association. In the community, the numbers of treated and untreated patients with angina, hypertension, or heart failure were obtained from the social insurance data, the National FINRISK Survey data, and the Hospital Discharge Register data. Information on treatment prescription and uptake was obtained from the National FINRISK Survey and from the National Health 2000 Survey (17, 21).

The IMPACT model

The cell-based IMPACT mortality model first used in Scotland and later validated in England, Wales, and New Zealand was further developed and refined (12, 15, 16, 22, 23). The model was described earlier in detail elsewhere (15). In brief, 1982 was taken as the baseline year. The number of CHD deaths expected in 1997 if the 1982 mortality rates had not declined was calculated by indirect age standardization. The number of deaths actually observed in 1997 were then subtracted to give the decline in deaths observed between 1982 and 1997. The number of CHD deaths prevented or postponed in Finland in 1982 and again in 1997 were calculated for specific interventions, such as thrombolysis, CABG, and drug treatments, as well as for risk factor reduction. Each specific mortality rate reduction was derived from the relative and absolute mortality rate reductions by using data from controlled trials and meta-analyses (23). To calculate the number of prevented or postponed CHD deaths, this model combines 1) the published effectiveness of cardiological treatments and risk factor reductions with 2) data on all medical and surgical treatments administered to all CHD patients in 1982 and 1997 in three regions of Finland: North Karelia, Kuopio province, and southwest Finland; and 3) trends in the major cardiovascular risk factors (smoking, cholesterol, and blood pressure) obtained from the National FINRISK surveys in 1982 and 1997.

As distinct from the original IMPACT model, blood pressure treatments were omitted from the treatment category in the Finnish model. Treatment for high blood pressure is considered mainly primary prevention, and its effect on

mortality is mediated through population blood pressure reduction, which was already included in the model. Because population risk factor data were available for all survey areas, all age groups, and both years, we were able to use Finnish β coefficients and a logistic regression formula to assess the effect of risk factor reduction and the Taylor formula (24) to calculate the confidence intervals for risk factor effects.

Treatment effects

The number of CHD deaths prevented or postponed for at least 1 year was calculated for each specific cardiac intervention in Finland in 1982 and again in 1997. To avoid double counting, adjustments were made for potential overlaps between different groups of patients. For example, approximately half of the patients who have had CABG surgery have also had a myocardial infarction. Adjustments made are described in detail in earlier publications (12, 16, 22).

Adherence, the proportion of treated patients actually taking therapeutically effective levels of medication, was available from patient records in many instances. Otherwise, if only the prescription rate was available, compliance was assumed to be 50–70 percent in community patients (25). This assumption was used for secondary prevention treatments after a myocardial infarction and CABG or angioplasty and for treatment of heart failure in community patients.

Data on the efficacy of treatments were reviewed from published randomized controlled trials and meta-analyses (23). The absolute mortality benefit determined from trials was applied to the appropriate patient group for each treatment (12, 15, 16). For situations in which combination therapy is common, such as drug treatments in secondary prevention, cumulative benefit was estimated by using the approach suggested by Mant and Hicks (26), where the cumulative relative benefit = $1 - (1 - \text{treatment A}) \times (1 - \text{treatment B})$, and so on.

Deaths prevented or postponed by medical and surgical treatments in 1982 and 1997

A number of effective therapies were already in limited use in 1982, including CABG surgery, in-hospital cardiopulmonary resuscitation, beta blockers for acute myocardial infarction, and beta blockers, acetylsalicylic acid, warfarin, and rehabilitation as secondary prevention. Precise patient data for these interventions were available from the data sources detailed above.

The number of deaths prevented or postponed with each treatment used in 1982 and also in 1997 was then estimated by multiplying 1) the number of patients in that group by 2) the uptake of cardiological treatments, 3) patient adherence, and 4) the absolute mortality benefit. The treatment effect in 1997 over and above that in 1982 was then estimated by subtracting the deaths prevented or postponed by treatments in 1982 from the deaths prevented or postponed by treatments in 1997. Concerning new treatments and treatment modalities introduced between 1982 and 1997—thrombolysis, angiotensin-converting enzyme inhibitors, statins, angioplasty, and use of acetylsalicylic acid during

the acute phase of myocardial infarction and as treatment for angina patients—any deaths prevented and postponed in 1997 were a gain compared with 1982, when these treatments were not used.

Risk factor effects

For risk factor changes, the model made use of logistic regression (β) coefficients estimated from Finnish cohorts in the National FINRISK Study (3). Each β coefficient quantifies the relation between population changes in a specific CHD risk factor (smoking, cholesterol, or blood pressure) and the consequent change in population mortality rates from CHD separately for men and women (12, 15, 16).

For the decline in each major risk factor, the subsequent reduction in deaths was estimated as the product of three variables: 1) the number of CHD deaths observed in 1982 (the baseline year), 2) the reduction seen in that risk factor between 1982 and 1997, and 3) the logistic regression β coefficient. The number of deaths prevented or postponed was calculated by using the following formula: $d = (1 - 1/e^{\beta(x_i - x)})n$, where d = deaths prevented or postponed, x_i = initial risk factor mean or prevalence (1982), x = risk factor mean or prevalence in 1997, and β = β value from the logistic regression model in the Finnish cohorts (3). The risk factor estimates were then repeated for cholesterol and smoking by using international β coefficients obtained from recent meta-analyses (15, 16, 27, 28).

Comparison with observed mortality decline

The model estimate for the total deaths prevented or postponed by all treatments plus all risk factor changes was then compared with the observed decline in mortality, stratified by age and sex. On an a priori basis, any shortfall in the overall model estimate was then formally attributed to other, unmeasured risk factors (12, 15, 16).

Sensitivity analysis

Because of the uncertainties surrounding some of the values, a sensitivity analysis was performed by using the analysis of extremes method (29). This method has been described in detail previously (15, 16). The 95 percent confidence limits for risk factor effects were calculated by using the Taylor formula, taking into account the variation in both the β coefficient and the risk factor changes (24).

Ethical issues and information protection

Ethical committees approved both the original and more recent studies used in the analyses. The information protection rules in the National Public Health Institute were followed throughout.

RESULTS

Mortality decline

Between 1982 and 1997, CHD mortality rates declined by 56 percent among men and 64 percent among women aged

TABLE 1. Mortality and risk factors for coronary heart disease in Finnish survey areas by year, gender, and age group

Risk factor and age group (years)	Men		Women	
	1982	1997	1982	1997
Coronary heart disease mortality rates (per 100,000)				
35–44	70	30	4	5
45–54	350	120	30	10
55–64	990	400	170	70
35–64	420	150	70	20
Diastolic blood pressure (mmHg)				
35–44	86.0	82.8	80.4	78.6
45–54	89.5	87.6	85.7	82.3
55–64	89.5	87.4	86.7	84.5
35–64	88.3	86.0	84.3	81.9
Cholesterol (mmol/liter)				
35–44	6.08	5.53	5.60	5.13
45–54	6.41	5.79	6.29	5.62
55–64	6.49	5.66	7.01	6.10
35–64	6.32	5.66	6.32	5.63
Smoking (%)				
35–44	39.3	35.7	19.0	21.8
45–54	36.1	33.6	13.1	16.6
55–64	36.6	23.3	9.4	11.4
35–64	37.4	30.6	13.7	16.5

35–64 years (table 1). In 1997, there were 373 fewer CHD deaths than expected from baseline mortality rates in 1982 (tables 2 and 3).

Changes in risk factors

A major decline was observed in mean serum cholesterol levels from 1982 to 1997 across all age and sex groups, from 6.2 mmol/liter to 5.6 mmol/liter overall. Declines in mean blood pressure and smoking prevalence were smaller (table 1).

Medical and surgical treatments

Several new medical treatments were introduced between 1982 and 1997 (table 2). Medical and surgical treatments together prevented or postponed approximately 99 deaths in 1997 (minimum estimate, 65; maximum estimate, 154). Treatments already in use in 1982 were estimated to have prevented or postponed approximately 13 deaths (table 2 and appendix table 1). The estimated decline in the number of deaths between 1982 and 1997 attributable to treatments over and above those achieved in 1982 was therefore approximately 86 (99 in 1997 minus 13 from 1982), which represented some 23 percent of the total CHD mortality decline of 373. Substantial contributions came from specific

treatments in individuals, immediate treatment of acute myocardial infarction (3.5 percent of the CHD total mortality decline), secondary prevention following acute myocardial infarction (3.5 percent) or revascularization (4.5 percent), revascularization and other treatments for angina (9.7 percent), and heart failure therapies in the hospital and in the community (1.9 percent) (table 2).

Major cardiovascular risk factors

Declines in the major cardiovascular risk factors together accounted for approximately 199 fewer deaths (minimum estimate, 162; maximum estimate, 235) (table 3), therefore accounting for some 53 percent of the total decline in CHD mortality between 1982 and 1997 (figure 1). The most substantial contributions came from reductions of 37.0 percent in serum cholesterol, 8.8 percent in smoking, and 7.5 percent in blood pressure. These mortality reductions reflected a substantial decline in mean population total cholesterol level and a smaller reduction in smoking prevalence and mean blood pressure (table 1). Smoking declined slightly in men, particularly older men (aged 55–64 years), but not in women (table 1).

Sensitivity analysis

When international β coefficients from recent meta-analyses were used (12, 15, 16, 27, 28), the mortality reduction attributable to the lower cholesterol level was increased from 138 to 198 fewer deaths and, likewise, from 33 to 42 fewer deaths for the decline in smoking, increasing the total contribution from risk factor reductions from 199 to 268 deaths prevented or postponed. Thus, the risk factor reduction then explained 71.8 percent of the decline in mortality.

Fifty percent compliance for secondary prevention treatments in Finland is probably a conservative estimate. If compliance in the community were assumed to be 70 percent, the treatment effect would rise from 99 deaths prevented or postponed to 115 in 1997 and from 13 deaths prevented or postponed in 1982 to 16. Then, treatments would explain 27 percent of the overall reduction in CHD mortality, and the share for secondary prevention would be nearly 11 percent.

The “analysis of extremes” method we used assumes that, for the minimum estimate, all lowest possible treatment effects occurred at the same time and, likewise, that all highest possible values occurred simultaneously for the maximum estimate. The proportional contributions of specific treatments and risk factor changes to the overall decline in CHD mortality in Finland between 1982 and 1997 remained relatively constant (figure 2). The overall mortality reduction from treatments in 1997 was approximately 23 percent (minimum, 14 percent; maximum, 37 percent). The risk factor changes explained approximately 53 percent of the mortality decline (when 95 percent confidence intervals were used, the minimum estimate was 46 percent and the maximum estimate was 60 percent).

TABLE 2. Contribution of medical and surgical treatments to the decline in coronary heart disease mortality in Finland between 1982 and 1997

Treatments in 1997	Patients eligible (no.)	Uptake of intervention (%)	Absolute risk reduction	Deaths prevented or postponed in 1997		Deaths prevented or postponed in 1982		Deaths prevented or postponed in 1997 vs. 1982		Proportion of 373 deaths prevented or postponed	
				No.	Minimum, maximum estimates	No.	Minimum, maximum estimates	No.	Minimum, maximum estimates	%	Minimum, maximum
Myocardial infarction	412			17	9, 26	4	3, 5	13	6, 21	3.5	1, 6
In-hospital CPR*		5	0.300	7							
Thrombolysis and acetylsalicylic acid		35	0.038	5							
Acetylsalicylic acid alone		50	0.013	3							
Thrombolysis alone		5	0.024	0							
β blocker		91	0.004	1							
ACE* inhibitor		34	0.006	1							
Secondary prevention after myocardial infarction	1,893			21	13, 36	8	6, 10	13	7, 26	3.5	2, 7
Acetylsalicylic acid alone		4†	0.006	0							
β blocker alone		13†	0.010	1							
Acetylsalicylic acid and β blocker		74†	0.016	11							
ACE inhibitors		34†	0.010	4							
Statins		31†	0.012	3							
Warfarin		13†	0.006	1							
Rehabilitation		14†	0.011	1							
Secondary prevention after CABG* surgery/angioplasty	1,400			17	10, 27			17	10, 27	4.5	3, 7
Acetylsalicylic acid alone		7†	0.006	0							
β blocker alone		7†	0.010	1							
Acetylsalicylic acid and β blocker		81†	0.016	9							
ACE inhibitors		25†	0.010	2							
Statins		48†	0.012	4							
Warfarin		10†	0.006	0							
Rehabilitation		10†	0.011	1							
Angina				37	29, 46	1	1, 1	36	28, 45	9.7	8, 12
CABG surgery and angioplasty	2,830‡	100	0.013	30							
Acute treatment of unstable angina in the hospital	828	38	0.009	6							
Acetylsalicylic acid in the community	9,851	38†	0.002	1							
Heart failure				7	4, 19			7	4, 19	1.9	1, 5
Hospital	80	27	0.072	4							
Community	6,731	52†	0.018	3							
Total treatment effect, 1997–1982				99	65, 154	13	10, 16	86	55, 138	23.1	14, 37

* CPR, cardiopulmonary resuscitation; ACE, angiotensin-converting enzyme; CABG, coronary artery bypass grafting.

† Prescription rate. In addition, patient compliance, assumed to be 50% in the community, was taken into account.

‡ No. of patients who had had CABG surgery or angioplasty during the last 5 years.

TABLE 3. Contribution of risk factor changes and treatments to the decline in coronary heart disease mortality in Finland between 1982 and 1997

Risk factors, total treatment effect	Population (no.)	β coefficient		Deaths prevented, postponed, or observed		Proportion of overall deaths prevented or postponed	
		Men	Women	No.	Minimum, maximum	%	Minimum, maximum
Effects of risk factor changes, 1982–1997							
Smoking	256,013	0.81	1.24	33	20, 46	8.8	5, 12
Cholesterol	256,013	0.38	0.37	138	124, 151	37.0	33, 41
Blood pressure	256,013	0.02	0.03	28	18, 37	7.5	5, 10
Total risk factor effect				199	162, 235	53.3	46, 60
Total treatment effect, 1997–1982 (from table 2)				86	55, 138	23.1	14, 37
Unexplained				88		23.6	
Observed decline in mortality				373		100	

Model fit

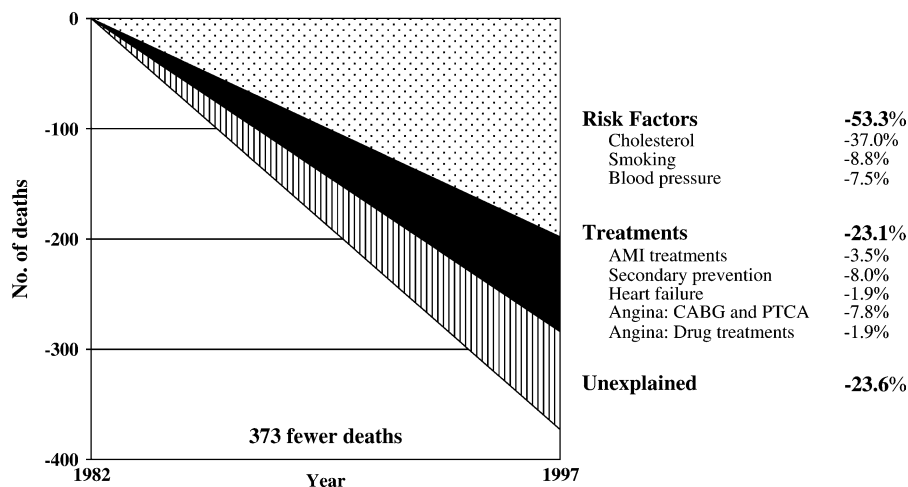
The estimated decline in the number of CHD deaths between 1982 and 1997 attributable to risk factor reductions and treatments over and above those achieved in 1982 was therefore approximately 285 (86 + 199). Thus, the IMPACT model explained 76 percent of the observed reduction in CHD mortality (figure 1). This fit improved to 95 percent when the 268 deaths prevented or postponed were used as risk factors based on international β coefficients, as described above. Thus, 5–24 percent of the observed decline for 373 subjects remained unexplained (table 3).

DISCUSSION

Over half of the substantial decline in CHD mortality in Finland between 1982 and 1997 was attributable to reduc-

tions in major risk factors. In addition to the remarkable decline in CHD mortality, the incidence of first coronary events also declined in Finland during this period, indicating successful implementation of primary preventative measures (30, 31). The biggest single risk factor contribution was from the large decline in total cholesterol levels. This change mainly reflects energetic and comprehensive prevention policies over many decades, including promotion of more healthy diets (32, 33).

Approximately 23 percent of the decline was attributable to medical and surgical therapies, which is lower than the 40–48 percent reported in other studies from the United States (2, 13), New Zealand (12), and Europe (14–16). However, the difference between these studies is partly explained by three factors: 1) a substantially greater decline in cholesterol in Finland than elsewhere; 2) an analysis confined to subjects aged less than 65 years (treatments may have

**FIGURE 1.** IMPACT model of the decline in coronary heart disease mortality in Finland between 1982 and 1997. AMI, acute myocardial infarction; CABG, coronary artery bypass grafting; PTCA, percutaneous transluminal coronary angioplasty.

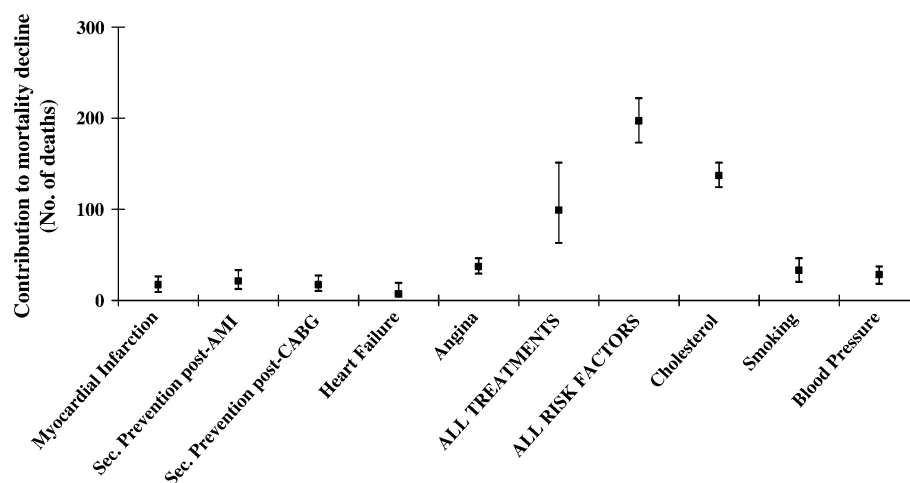


FIGURE 2. IMPACT model sensitivity analysis of the decline in coronary heart disease mortality in Finland between 1982 and 1997. Black squares, best estimate; T-shaped bars, minimum and maximum estimates for treatments and 95% confidence interval for risk factors. Sec., secondary; AMI, acute myocardial infarction; CABG, coronary artery bypass grafting.

a relatively greater impact in those aged 65–74 years) (16); and 3) our decision to omit blood pressure treatments from the treatment category in the present study. Although Hunink et al. (13) apparently attributed 71 percent of the recent US CHD mortality decline to “treatments,” this difference mainly reflects a different categorization of risk factor decline in individual patients with recognized CHD. In Finland, myocardial infarction register studies have estimated that, between 1983 and 1992, approximately half of the decline in CHD mortality was due to the decline in the occurrence of recurrent myocardial infarction events, which reflects the effect of secondary prevention and treatment of chronic, symptomatic CHD (34).

Although mortality and cholesterol levels declined considerably during the study period, there is still room for improvement. Smoking rates remained higher among younger men compared with those in other Western countries, and blood pressure was also high and declined only slightly between 1982 and 1997. Recent analyses from the United Kingdom suggest that, with the modest risk factor reductions already achieved in the United States and Scandinavia (19), CHD mortality could be halved by 2010 (35).

On the other hand, on the basis of evidence from elsewhere (16, 17, 21, 36, 37), there have been adverse trends over this time period regarding several cardiovascular risk factors and contributing disorders such as obesity, physical inactivity, and diabetes. Together, these disorders probably contributed a number of additional deaths in 1997 (16).

Modern cardiological treatments together prevented or postponed approximately 100 deaths in 1997 in the study population. Irrespective of whether best, minimum, or maximum estimates were used, the most substantial contributions were from secondary prevention treatments. Revascularization from CABG surgery and angioplasty together postponed approximately 30 deaths and thus accounted for only about 8 percent of the total decline in mortality, much

as in other studies (12, 15, 16, 38, 39). Although disappointing, this consistently small contribution highlights the contrasting mortality benefits between expensive interventional therapies (principally aimed at relatively few symptomatic and severe cases) and relatively inexpensive population interventions aimed at reducing the burden of current and future disease. Furthermore, the overwhelming majority of coronary deaths occur suddenly out of hospital, which naturally limits the effects of hospital interventions at the population level (40).

Thrombolysis (in combination with acetylsalicylic acid) likewise accounted for approximately only five fewer deaths in 1997, cardiopulmonary resuscitation for seven. This finding is consistent with those from other studies (12, 15, 16).

Treatment of heart failure patients in the community prevented as many deaths as treatment of heart failure patients admitted to hospitals. However, treatment uptake levels in the community were generally poor. Earlier work using this model suggested that if 80 percent of eligible patients had received appropriate therapy in England and Wales in 2000, approximately 21,000 additional deaths might have been prevented or postponed (41, 42).

Overall treatment benefits were similar for both men and women, as were the effects of blood pressure and cholesterol reduction. However, smoking rates declined among men between 1982 and 1997 but increased slightly among women. Additional initiatives are clearly needed.

Modeling studies such as ours have a number of strengths. They provide transparent integration of the best available information from many sources. Furthermore, no important treatments were excluded from this model (15, 16). Gender-specific β coefficients were obtained from Finnish sources. These coefficients were relatively conservative compared with those from other studies (23). Modeling can also provide a rapid and inexpensive way of assessing different policies for interventions (35, 41, 43).

All such studies also have limitations. They are dependent on the variable quality and extent of data available on cardiovascular risk factor trends and CHD treatment uptakes (44). Assumptions have to be made to fill the gaps, and a robust sensitivity analysis therefore becomes essential (29). However, the quality of the Finnish data was generally very good (17, 20, 34, 40). The National Causes of Death Register data have been available since 1936 and the Hospital Discharge Register data since 1967. Population risk factor data for 30 years are available from the National FINRISK Study, which follows international data collection standards. The accuracy of Finnish databases concerning the registering of CHD deaths and events has proven to be good (45). Furthermore, the relative contribution of each risk factor decline and each treatment category to the overall decline in CHD mortality changed very little whether we considered best, minimum, or maximum estimates (figure 2).

Some 5–24 percent of the mortality decline was not explained by the existing model. This limitation may well reflect the relatively low value of the Finnish β coefficients compared with those from international studies (23). The gap may also reflect improvements in other risk factors such as diet, particularly the consumption of fruit and vegetables (46). It was not possible to quantify the effect of other risk factors such as dietary antioxidants, alcohol, obesity, diabetes, physical inactivity, and increasing affluence. However, these risk factors are generally considered minor (47). Furthermore, no important treatments were excluded from this model (15, 16).

The model included data on only those persons aged 35–64 years, since risk factor data were not available for older age groups. We considered CHD deaths only and ignored the possibility of some increases in deaths from “competing causes.” However, reductions in smoking have actually reduced deaths from lung and other cancers (48). A decrease in stroke mortality can also be expected with decreasing levels of cardiovascular risk factors. In fact, in Finland, total mortality in the respective age groups has also declined about 30 percent among men and 17 percent among women following the trend observed in CHD mortality. A significant increase in mortality is evident in only alcohol-related causes of death (49). Our analysis focused on only mortality rather than incidence, symptomatic relief, or “life-years gained.” These important areas merit attention in future work.

The model assumed that estimates of efficacy from randomized controlled trials could be generalized to effectiveness in clinical practice. This may be acceptable (50). It also assumed that the mortality reductions from concomitant risk factor reductions and treatments within the same patient were independent. This assumption appears reasonable on the basis of the limited data available (12, 16, 51). However, although the β coefficients were all independent, being adjusted for major confounders, “residual confounding” may have remained, and potential risk factor interactions were not considered (52, 53). Further development work is clearly needed.

Lag times were not explicitly considered but are likely to be modest when a 15-year time period is considered. Randomized clinical trials and meta-analyses generally suggest that substantial risk reduction occurs relatively quickly,

within 2–5 years of quitting smoking or reducing cholesterol levels (27, 28, 51).

In conclusion, much of the decline in CHD mortality can be explained by changes in the major risk factors. These Finnish results provide a valuable opportunity to quantify the role of risk factors and improved treatment in the decline in cardiovascular diseases. This information can be used in health care policy formulation and strategic planning. Our findings emphasize the importance of a comprehensive strategy that actively promotes primary and secondary prevention programs for diet and smoking and that maximizes population coverage of evidence-based treatments.

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APPENDIX TABLE 1. Contribution of treatments in 1982 to the decrease in coronary heart disease mortality in Finland in 1982

Treatments and risk factors in 1982	Patients eligible (no.)	Uptake of intervention (%)	Absolute risk reduction	Deaths prevented, postponed, or observed	
				No.	Minimum, maximum
Myocardial infarction	412			4	3, 5
In-hospital CPR*		2	0.300	3	
β blocker		68	0.004	1	
Secondary prevention	1,893			8	6, 10
β blocker alone		65†	0.010	6	
Acetylsalicylic acid and β blocker		2†	0.016		0, 5
Warfarin		7†	0.006		0, 5
Rehabilitation		15†	0.011	1	
Angina				1	1, 1
CABG* surgery	92‡	100	0.011	1	
Total treatment effect in 1982				13	10, 16

* CPR, cardiopulmonary resuscitation; CABG, coronary artery bypass grafting.

† Prescription rate. In addition, patient compliance, assumed to be 50% in the community, was taken into account.

‡ No. of patients who had had CABG surgery during the last 5 years.