Forty-year change in coronary heart disease mortality among working aged men and women in Eastern Finland: the role of primary prevention and risk factor reduction

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Forty-year change in coronary heart disease mortality among working aged men and women in Eastern Finland: the role of primary prevention and risk factor reduction

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Abstract

Objectives: To estimate the extent to which changes in the main cardiovascular risk factors (smoking prevalence, and serum cholesterol and systolic blood pressure levels) in the population explain the 40 year decline in coronary heart disease (CHD) mortality among working aged men and women.

Design: Predicted change in CHD mortality was estimated with a logistic regression model using risk factor data collected in nine consecutive population-based risk factor surveys conducted every five years since 1972. Data on observed CHD mortality were obtained from the National Causes of Death Register.

Setting: Eastern Finland

Participants: 34,525 men and women aged 30-59 years who participated in the National FINRISK Studies between 1972 and 2012

Interventions: Change in main cardiovascular risk factors through population-based primary prevention

Main outcome measures: Predicted and observed age standardized CHD mortality

Results: During the forty year time period, the levels of major cardiovascular risk factors, smoking prevalence, serum total cholesterol and systolic blood pressure declined, except a small increase in serum cholesterol levels between 2007 and 2012. From the early seventies (1969-1972) to 2012 CHD mortality decreased by 82% (from 643 to 118 per 100,000) among men aged 35 to 64 years. Among women the decline was 84% (from 114 to 17 per 100,000). During the first ten years, changes in these three target risk factors explained nearly all of the observed mortality reduction. Since the mid-1980s, the observed reduction in mortality has been larger than predicted. In the last ten years about two thirds (69% in men and 66% in women) of the CHD mortality reduction was explained by changes in risk factors and one third by other factors.

Conclusion: Large declines in disease burden and mortality due to CHD can be achieved through population-based primary prevention programs. Secondary prevention among high risk individuals and treatment of acute CHD events confer additional benefit.

What this paper adds

Section 1: What is already known on this subject

Tobacco smoking, high serum cholesterol and high blood pressure are the main classical risk factors for CHD. Age-standardised CHD mortality has been declining in many western countries but is increasing in many developing countries and countries in transition. Relatively little is known, as to how much primary prevention and changes in risk factors can explain the changes in CHD mortality.

Section 2: What this study adds

Risk factor reduction at a population level is a very effective way of reducing CHD mortality. Secondary prevention among high risk individuals and treatment of acute CHD events confer additional benefit.

Introduction

Even though mortality from coronary heart disease (CHD) and other cardiovascular diseases (CVD) has been decreasing in many countries, particularly in western industrialized countries, in the last decades, they are still the most common causes of death in the world. Furthermore, CVD mortality is increasing in many developing countries and countries in transition. Of the total of 54.9 million deaths in the world in 2013, 17.3 million (31%) were due to CVDs. Globally CVD is the most common cause of death in all World Health Organization (WHO) regions except in the African region. CHD is the most common CVD in Europe, Americas and Australia, whereas cerebrovascular diseases are more important in many Asian countries [1].

CHD epidemic started in the United States in the 1930s and spread into the Western European countries after the Second World War [2]. Data on the causes of CHD started to accumulate in the 1940s and 1950s. Large epidemiological studies, such as the British Medical Doctors Study, the Framingham Study and the Seven Countries Study, could identify a few behavioral and biological factors associated with the CHD risk, particularly tobacco smoking, high serum cholesterol and high blood pressure [3-5]. Since then their importance and causal relationship with the CHD risk have been confirmed in a large number of observational epidemiological studies and clinical trials [6-8]. Also dietary factors contributing to high cholesterol and high blood pressure levels, high intake of saturated fat and salt (sodium chloride), have been known already for decades [9,10].

CHD mortality started to increase in Finland in the 1950s associated with increasing standard of living and changes in in diet and other lifestyles. In the late 1960s, CHD mortality in Finland was the highest in the world, and mortality was particularly high among the working aged men in the eastern part of the country. The North Karelia Project, first community-based CVD prevention project in the world, was launched in 1972. The main aim of the project was to reduce the extremely high CHD mortality among the working aged men by reducing the levels of the three main cardiovascular risk factors [11]. Main emphasis was put on behavioral change through community action and participation, supported by screening of high risk individuals and medical treatment [12]. Systematic population-based risk factor monitoring was developed as part of the project and since 1972 risk factor surveys have been conducted every five years [13].

Twenty years ago, in 1994, we reported the role of the risk factor change in CHD mortality reduction from 1972 to 1992 among working age men and women in Eastern Finland [14]. In the last twenty years, cardiovascular risk factor patterns, secondary prevention practices and treatment of acute events have markedly changed [13,15]. The aim of the present study is to analyse the role of primary prevention and risk factor changes in forty-year (1972-2012) CHD mortality trends in the same population, and whether the role of the three main risk factors in explaining CHD mortality trend has changed in the last twenty years.

Material and methods

Participants

The study population consists of participants of nine independent population-based surveys conducted in the provinces of North Karelia and Kuopio in Eastern Finland first time in 1972. Since then, the levels of behavioral and biological risk factors have been continuously monitored every five years and the last risk factor survey was conducted in 2012 [13].

For each survey year a random sample was drawn from the national population register. In 1972 and 1977 the sample was 6.6% of the population born during 1913-1947. Since 1982 an age, sex and study area stratified random sample was taken from the population aged 25 to 64 years according to the WHO MONICA Project protocol [16]. A total of 34,525 men and women aged 30 to 59 years, which is the common age range of all nine surveys, participated in the risk factor surveys. In the first surveys, participation rate was high, over 90% but declined in the later surveys being 64% in the last survey.

Ethical approval has been obtained according to the commonly required research procedures and Finnish legislation during each survey. The three last surveys were approved by the Coordinating Ethics Committee of the Helsinki and Uusimaa Hospital District. From 1997 onwards a written informed consent has been obtained from each participant. The study has been conducted according to the World Medical Association Declaration of Helsinki on ethical principles for medical research.

Risk factor measurements

In each survey year, data collection included (1) a self-administered questionnaire filled in at home, checked and, if needed, completed at the study site, (2) physical measurements at the study site done by trained study nurses and (3) blood samples for laboratory analyses. During the whole forty year period, collection of risk factor data was done following the same standardized core protocol [13].

Smoking was assessed with a standard set of questions in the study questionnaire. Non-smokers were those who had never smoked regularly, and those smokers who had stopped smoking at least six months before the survey. At the study site, blood pressure was measured using mercury sphygmomanometers. Before the survey, all nurses who did blood pressure measurements received a four-day training to ensure a standardized measuring technique. Blood pressure was measured from the right arm of the subject after a five minute rest. Serum cholesterol analyses were done in the same central laboratory in the National Institute for Health and Welfare (formerly National Public Health institute). Due to changes in laboratory technology during the 40 year period, several methods were used for determining serum cholesterol levels.

Methods, instruments and reagents for cholesterol measurement and the quality analysis data have been described elsewhere [17]. The laboratory has taken part in international quality assurance systems, first with the WHO laboratory reference center in Prague and the last three surveys with the Center for Disease Control and Prevention in Atlanta. Based on the quality analysis, systematic measurement errors due to changes in laboratory methods and reagents in different study years have been corrected [13].

Mortality prediction

CHD mortality was predicted using a logistic regression model based on a 15-year follow-up of 14,536 men and 15,278 women who participated in the risk factor surveys between 1972 and 1997. Age, serum total cholesterol, and systolic blood pressure were included into the model as continuous variables and smoking status as a dichotomous variable. Data on CHD mortality were obtained from the National Causes of Death Register. During the follow-up, 1014 (830 in men and 173 in women) CHD deaths were observed. The probability of death in the logistic regression model was 1/(1+exp (13.0-0.102 x age-0.818 x smoking-0.016 x systolic blood pressure-0.368 x cholesterol) for men and 1/(1+exp (16.22-0.119 x age-1.06 x smoking-0.022 x systolic blood pressure-0.330 x cholesterol) for women. All terms were significant at 0.001 level. The original model included both systolic and diastolic blood pressure. In stepwise analysis systolic pressure was selected in the final model.

Average probability of CHD death for each five-year period from 1972 to 2012 was calculated by including the mean levels of the measured risk factors in the logistic regression functions. The relative importance of each risk factor was estimated separately by changing the logistic regression function value of only that risk factor and keeping the other risk factors unchanged at the 1972 level. The predicted percentage change in CHD mortality compared with the 1972 level was then calculated for each survey period. Confidence interval for the predicted mortality change was calculated by taking into account the standard errors of parameters' estimates in the logistic regression function.

Observed mortality

Data on the forty-year trend in CHD mortality in the study area were obtained from the National Causes of Death Register for men and women aged 35-64 years. The following ICD codes were classified as CHD deaths: ICD 8 and 9 410-414, and ICD 10 I20-I25. Annual mortality rates were standardized for age in five year age groups using the baseline (1972) population structure as a standard population. The percentage decline in observed CHD mortality was calculated using the mean of 1969-1972 mortality as baseline.

Results

During the forty year time period, from 1972 to 2012, marked changes were observed in the levels of risk factors (table 1). Smoking prevalence decreased from 52.6% to 29.3% among men. Among women smoking was rare in the 1970s, only 11.4% of women were smokers. The proportion increased up to 22.4% in 2002 and declined after that slightly being 19.4% in 2012. In both genders, serum total cholesterol declined remarkably during the first 35 years of the study but a small increase was observed between 2007 and 2012. In 1972, the mean serum cholesterol level was 6.8 mmol/l in men and 6.7 mmol/l in women, and in 2012 the levels were 5.4 mmol/l and 5.3 mmol/l, respectively. Mean systolic blood pressure also declined remarkably from 147.1 mmHg to 135.9 mmHg in men and 149.2 to 129.1 mmHg in women.

From the baseline level at the early seventies (1969-1972) to 2012 CHD mortality decreased from 643 to 118 per 100,000 among working aged men (aged 35 to 64 years) and from 114 to 17 per 100,000 among working aged women (Figure 1). The decrease was 82% in men and 84% in women (Table 2).

During the first ten years, changes in smoking prevalence, and serum cholesterol and systolic blood pressure levels explained nearly all of the observed mortality reduction (Figures 2 and 3). Between 1982 and 2002, the observed CHD mortality decline was faster than predicted. In the last ten years trends in observed and predicted mortality have been quite similar. In the 1990s about three quarters (in 1992 75% in men and 76% in women), and in the last ten years about two thirds (in 2012 69% in men and 66% in women) of the CHD mortality reduction was explained by changes in the analysed three risk factors. In men, reduction in serum cholesterol levels explained most of the mortality decline. In women, reductions in serum cholesterol and systolic blood pressure levels contributed equally to the mortality reduction.

Discussion

In the 1970s, reduction in risk factors explained practically all of the observed reduction in CHD mortality, in the 1980s the observed mortality started to decline faster than the mortality estimates predicted by the risk factor changes. In the 1990s risk factors explained about three quarters of mortality reduction, and in the last ten years still about two thirds. Changes in serum cholesterol levels explained most of the CHD mortality reduction in men, whereas changes in serum cholesterol and systolic blood pressure levels were equally important in women. The remaining one third of mortality reduction may be explained by three major groups of factors: (1) changes in other primary risk factors, which were not included in our analysis, such as diet and physical activity, (2) improvement in secondary prevention and (3) improvement in the treatment of acute cardiac events.

Smoking used to be very common among men in Finland and some two thirds of men were smokers in the 1950s. Smoking prevalence started to decline among men in the 1960s and 1970s and in the 1980s the decreasing trend accelerated due to active anti-smoking campaigns and legislation. Among women, smoking was not part of the culture in Eastern Finland, and the prevalence of smoking used to be low, but it started to increase in the 1980s and 1990s due to urbanization and change in the culture. Among women the increase of smoking prevalence levelled off in the 1990s and in the last ten years the prevalence of smoking has been decreasing also in women. The first comprehensive tobacco law was introduced in Finland in 1976, and since then the law has been revised several times [18]. Currently the smoking prevalence in Finland is one of the lowests in Europe. The official target according to the latest amendment of the tobacco law is smoke-free Finland (defined as smoking prevalence below 5%) by 2040.

In the early 1970s, serum cholesterol levels in eastern Finland were extremely high, the average was nearly seven mmol/l and over 90% of the middle-aged population had serum cholesterol higher than five mmol/l, the recommended upper limit in current international guidelines [19, 20]. Thus, a population-based strategy to move the whole cholesterol distribution downward was the most effective way to advance the prevention. The role of fat content in the diet as the determinant of serum cholesterol levels, the relationship of polyunsaturated and saturated fat intake, was known already in the 1960s [9]. Due to high consumption of fatty milk products and butter, which were core components in traditional diet and also the main agricultural products in the area, saturated fat intake used to be very high. On the other hand, forty years ago vegetable oils were hardly known and vegetable consumption was low in Eastern Finland. Even though the promotion of dietary change was challenging, major changes were observed.

Main dietary changes were the change from fatty milk to low-fat and skimmed milk, a huge reduction in butter consumption, from nearly 20 kg to less than 5 kg per capita per year, and a marked increase in the use of vegetables and vegetable oils [21,22]. In parallel with dietary changes, serum cholesterol levels started to decline. Based on nutritional data collected at the same time with cholesterol measurements, at population level over 80% of the cholesterol lowering was explained by dietary changes and only less than 20% by the pharmaceutical drug (statins) treatment of high cholesterol [23]. Paradoxically, reduction of serum cholesterol levels was fastest in the 1970s and 1980s, at the time when pharmaceutical drug treatment of high serum cholesterol was minimal, and the decrease levelled off in the 1990s when the drug treatment became more common. In the 2012 survey, 18 % of participants (21% of men and 15% of women) were using statins.

In the last five years, both reported intake of saturated fat and dietary cholesterol, and measured cholesterol levels increased slightly. Actually, the measured cholesterol change was nearly the same as

calculated from the dietary data by using the classical Hegsted equation [24]. The reason for the unfavorable dietary change and cholesterol increase might be the boom of low carbohydrate (and high fat) diet in Finland during the last risk factor survey in 2012. How permanent this change is will be seen in the next risk factor survey in 2017.

Also mean blood pressure levels were very high in eastern Finland in the early 1970s. In the lowering of blood pressure, a combined strategy of lifestyle change, mainly reduction of high salt intake, and screening and pharmaceutical drug treatment were applied [25]. The average salt intake declined by one third, from 14 grams in men and 10 grams in women in the 1970s to 8.9 and 6.5 grams in 2007, respectively [26]. A small increase in salt intake was observed in the last five years. Both systolic and diastolic blood pressures declined the first thirty years, and systolic pressure has continued its decline since then, but in diastolic pressure the reduction levelled off in the 1990s and a small increase has been observed in the last ten years. This difference in trends may be due to the difference in systolic and diastolic blood pressure pathophysiology: systolic pressure is mainly determined by the stiffness of large arteries whereas diastolic pressure depends more on the peripheral resistance [27, 28].

In addition to the three classical risk factors, a few other factors, such as physical inactivity, obesity, and elevated blood glucose, and diabetes as their consequence, have been identified as major causes for CHD [29,30]. The role of alcohol drinking in CHD risk is controversial, very modest drinking may reduce the risk but heavy and binge drinking are most probably harmful [31]. Physical inactivity and obesity were not particularly common in Eastern Finland in the 1970s but they become evident health problems later on. Physical activity in work and travel to and from work has decreased whereas leisure time physical activity has continuously increased during the last decades [32]. Objective measurement of total physical activity, however, is complicated in large population-based studies. Mean body mass index (BMI) and prevalence of obesity started to increase in the late 1970s but the increase levelled off during the last five years [13]. Including BMI in the predictive model did not affect the results markedly, most probably because the effect of obesity on CHD risk is largely mediated through its effect on blood pressure. Unfortunately we do not have comparable data on the changes in diabetes prevalence during the survey period. Self-reported data is not reliable because diagnostic activity to detect diabetes in health care, and also international diagnostic criteria for diabetes, have changed during the last decades. Total alcohol consumption has about doubled in Finland in the last four decades, and most probably the prevalence of harmful drinking has increased [33].

In the 1970s and early 1980s, practically the whole observed CHD mortality reduction was explained by reduction of risk factors. The trend lines started to separate in the middle of the 1980s and the observed

mortality reduction was faster than predicted. In the last ten years the trends in predicted and observed mortality reduction have been quite similar and about two thirds of the mortality reduction is explained by changes in risk factors and one third by other factors. These findings fit quite well with the development of secondary prevention and treatment practices in the last decades. In the 1980s new secondary prevention guidelines were introduced including active drug treatment with aspirin, beta-blockers, ACE inhibitors and later on statins [34]. Invasive cardiology also expanded at the end of 1980s. Percutaneous coronary interventions (PCI) were started in the early 1990s. The number of PCI procedures rose fivefold in ten years between 1994 and 2004. Accordingly, case fatality of acute CHD events reduced by one third between 1994 and 2004 and the decline has continued [35,36]. Among CHD patients aged 30 years or more (self-reported previous myocardial infarct or coronary heart disease) age-standardised prevalence of revascularization procedures (bypass operation or PCI) was 54 % in men and 34 % in women in 2011, compared to 33 % and 12 % in 2000 [37]. However, because we do not have individual level data on secondary prevention and treatment, we can estimate their role in the mortality change only indirectly.

Even though a large number of other factors, including sensitive C-reactive protein (CRP) and other markers of low grade inflammation, hemostatic factors, vitamin and flavonoid intake and other dietary factors, amount and quality of sleep, and depression and social deprivation have been shown to be associated with the risk of CHD, final evidence on their causal role in the development of CHDs is still lacking [38]. Family history of CHD and a number of genetic markers are associated with CHD risk but the role of hereditary factors in the prevention of CHD is largely open [39]. Genetic background of the population has not changed during the last forty years and cannot explain the dramatic decrease in CHD mortality.

The role of risk factor changes and treatments in CHD mortality have been analyzed in many countries using the IMPACT-model approach developed in the United Kingdom [40,41]. The model takes into account population level changes in main risk factors, and the most effective treatments including lipid and blood pressure lowering drugs both in primary and secondary prevention, treatments in acute events and rehabilitation. Reduction in CHD mortality attributable to risk factors and treatments vary based on the time period, baseline situation in risk factor levels and treatments, and observed changes in risk factors and treatment practices, as well as the age group of patients. In countries, where the decline in risk factors have been significant during the observation period the majority of the mortality decline has been attributable to risk factor reduction. In IMPACT studies conducted in different countries, such as Sweden, Turkey, Portugal and US, the proportion of mortality decline attributed to treatment and secondary prevention varied between from a quarter to a half [42,43,44, 45].

The main strengths of our study are the long and systematic population-based risk factor monitoring using the same standardized protocol over four decades and a practically complete mortality data. To our knowledge, the systematic risk factor monitoring system in Eastern Finland is the longest continuous risk factor monitoring in the world. Even though individual verification of the cause of death is not possible in large population studies, validation studies have shown that the reliability of diagnosis in the Finnish Causes of Death Register is good [46].

The main limitations are related to the decreasing participation rate and possible measurement error in risk factor surveys. Even though the 60% participation rate in large health examination surveys is still fairly good in international comparison, we know that the risk factor levels among non-participants are somewhat higher than among the participants [47]. Therefore, our model may overestimate the importance of the risk factor change in the last couple of decades. On the other hand, our predictive model is based on single measurements of the risk factors being prone to random measurement error which diminishes the strength of the true association between the measured risk factor and the endpoint, and as a consequence underestimates the importance of risk factor change in CHD mortality reduction [48]. Because we assessed the smoking status only at the baseline, and we were not able to update it during the follow-up, our model most probably underestimated the role of smoking in the CHD mortality decline [49]. Even though saturated fat (and trans-fat) intake, intake of dietary cholesterol and drug treatment are the main determinants of serum cholesterol level, the observed change in serum cholesterol levels most probably reflects also other dietary changes. Similarly, reduction of salt intake may be associated with other healthy lifestyle changes. Therefore, the estimated effect of the change in serum cholesterol and blood pressure may include unmeasured confounding caused by the change of other dietary factors. Finally, even though pharmaceutical treatment reduces the levels of serum cholesterol and blood pressure effectively, most probably it does not completely reverse the atherosclerosis developed before the start of the treatment.

In conclusion, even though secondary prevention and treatment protocols have markedly developed in the last decades, primary prevention and reduction of the levels of main classical cardiovascular risk factors should still be considered as the main strategy to reduce disease burden and mortality due to CHD. This is in accordance with the current WHO non-communicable disease action plan, which stresses the role of population-based approach in prevention and control of cardiovascular and other non-communicable diseases [50]. Secondary prevention among high risk individuals and treatment of acute CHD events confer additional benefit.

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Figure 1. : Age-standardised CHD mortality 1969-2012, men and women 35-64 years (per 100 000, logarithmic scale)

Figure 2. Predicted and observed reduction in CHD mortality 1972-2012, men 35-64 years

Figure 3. Predicted and observed reduction in CHD mortality 1972-2012, women 35-64 years

Table 1. Cardiovascular risk factor levels (95% Confidence Interval) among men and women aged 30 to 59 years in eastern Finland from 1972 to 2012

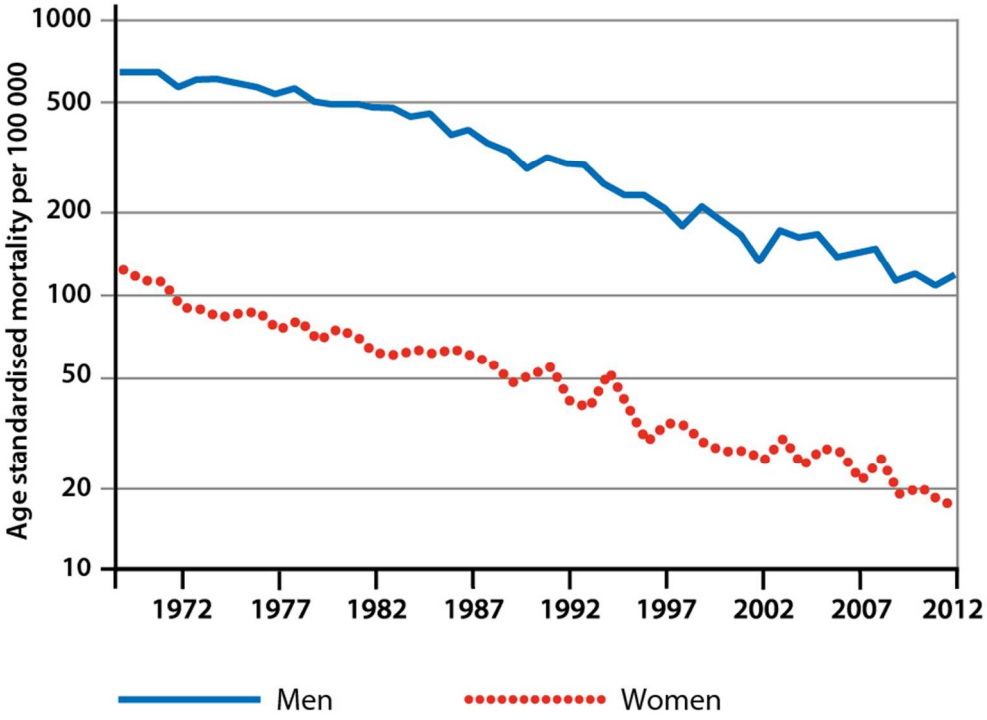
	Men				Women			
Year	Number	Smoking (%)	Serum cholesterol (mmol/L)	Systolic blood pressure (mmHg)	Number	Smoking (%)	Serum cholesterol (mmol/L)	Systolic blood pressure (mmHg)
1972	4462	52.6 (51.2-54.1)	6.77 (6.73-6.81)	147.1 (146.5-147.7)	4804	11.4 (10.5-12.3)	6.69 (6.65-6.72)	149.2 (148.5-149.9)
1979	4436	46.6 (45.2-48.1)	6.52 (6.49-6.56)	144.2 (143.6-144.7)	4659	12.7 (11.8-13.7)	6.34 (6.30-6.38)	141.6 (140.9-142.2)
1982	2144	41.7(39.6-43.8)	6.26 (6.21-6.31)	145.5 (144.7-146.3)	2005	16.3 (14.7-17.9)	6.04 (5.98-6.09)	141.6 (140.7-142.5)
1987	1528	40.5 (38.0-42.9)	6.23 (6.17-6.29)	144.0 (143.1-144.9)	1672	17.3 (15.5-19.2)	5.92 (5.86-5.98)	138.1 (137-2-139.1)
1992	962	36.8 (33.7-39.8)	5.91 (5.84-5.98)	140.7 (139.5-141.8)	1031	21.3 (18.8-23.8)	5.55 (5.48-5.61)	134.6 (133.3-135.9)
1997	1004	33.3 (30.3-36.2)	5.70 (5.64-7.77)	138.8 (137.7-139.9)	1107	17.9 (15.6-20.1)	5.54 (5.48-5.60)	132.6 (131.5-133.7)
2002	895	36.9 (33.7-40.0)	5.60 (5.53-5.68)	137.2 (136.0-138.4)	1036	22.4 (19.8-24.9)	5.33 (5.28-5.39)	131.8 (130.5-133.0)
2007	699	32.2 (28.7-35.7)	5.35 (5.27-5.42)	138.0 (136.7-139.3)	770	21.9 (19.0-24.9)	5.16 (5.10-5.23)	132.2 (130.8-133.6)
2012	605	29.3 (25.6-32.9)	5.44 (5.35-5.52)	135.9 (134.5-137.2)	706	19.4 (16.5-22.3)	5.30 (5.23-5.37)	129.1 (127.9-130.4)

Table 2. Observed and by the risk factor change predicted CHD mortality decline among men and women aged 35 to 64 years in Eastern Finland

Year	Observed CHD mortality decline (%) *	Predicted CHD mortality decline (%) ** and 95% Confidence Interval				Proportion of the predicted CHD mortality decline from the observed mortality decline (%)
		Smoking alone	Systolic blood pressure alone	Serum cholesterol level alone	All three risk factors together	
Men						
1969-72 (baseline)	0	0	0	0	0	
1977	17	4.6 (0.5-8.8)	4.4 (0.3-8.6)	8.4 (4.2-12.5)	16.5 (12.3-20.7)	97
1982	25	8.3 (3.1-13.4)	2.5 (-2.7-7.6)	16.6 (11.5-21.8)	25.4 (20.3-30.6)	101
1987	38	9.1 (3.3-14.9)	4.7 (-1.1-10.5)	17.4 (11.6-23.2)	28.5 (22.7-34.3)	75
1992	55	11.7 (4.8-18.7)	9.4 (2.4-16.4)	26.4 (19.4-33.3)	41.3 (34.3-48.2)	75
1997	67	14.2 (7.3-21.0)	12.0 (5.2-18.9)	31.6 (24.7-38.4)	48.5 (41.7-55.4)	72
2002	75	11.7 (4.5-18.9)	14.2 (7.0-21.3)	34.0 (26.8-41.2)	50.2 (43.0-57.4)	67
2007	78	14.9 (6.9-22.9)	13.1 (5.2-21.1)	39.8 (31.8-47.8)	55.7 (47.7-63.7)	71
2012	82	16.9 (8.4-25.3)	15.9 (7.4-24.4)	37.8 (29.3-46.3)	56.8 (48.3-65.3)	69

Women						
1969-72 (baseline)	0	0	0	0	0	
1977	28	-1.4 (-5.4-2.6)	15.5 (11.5-19.5)	10.9 (6.9-14.9)	23.7 (19.6-27.7)	85
1982	41	-5.3 (-10.5- -0.1)	15.4 (10.2-20.6)	19.3 (14.1-24.5)	28.1 (22.9-33.3)	68
1987	45	-6.4 (-12.0 - -09)	21.6 (16.0-27.2)	22.4 (16.-27.9)	35.5 (29.7-40.8)	79
1992	59	-11.0 (-17.7- -4.3)	27.5 (20.7-34.2)	31.3 (24.5-38.0)	44.7 (37.9-51.4)	76
1997	72	-7.0 (-13.6- -0.5)	30.6 (24.1-37.1)	31.4 (24.8-37.9)	49.0 (42.5-55.6)	68
2002	77	-12.2 (-19.0- -5.5)	31.6 (25.2-38.6)	35.9 (29.2-42.6)	51.5 (44.3-57.7)	67
2007	79	-11.7 (-19.3- -4.1)	31.3 (23.6-38.9)	39.4 (31.8-47.0)	53.5 (45.9-61.1)	68
2012	84	-8.8 (-16.7- -0.9)	35.7 (27.8-43.6)	36.6 (28.7-44.5)	55.7 (47.8-63.6)	66

*Five year means, **Predicted decline in CHD mortality based on risk factor changes during each five year period



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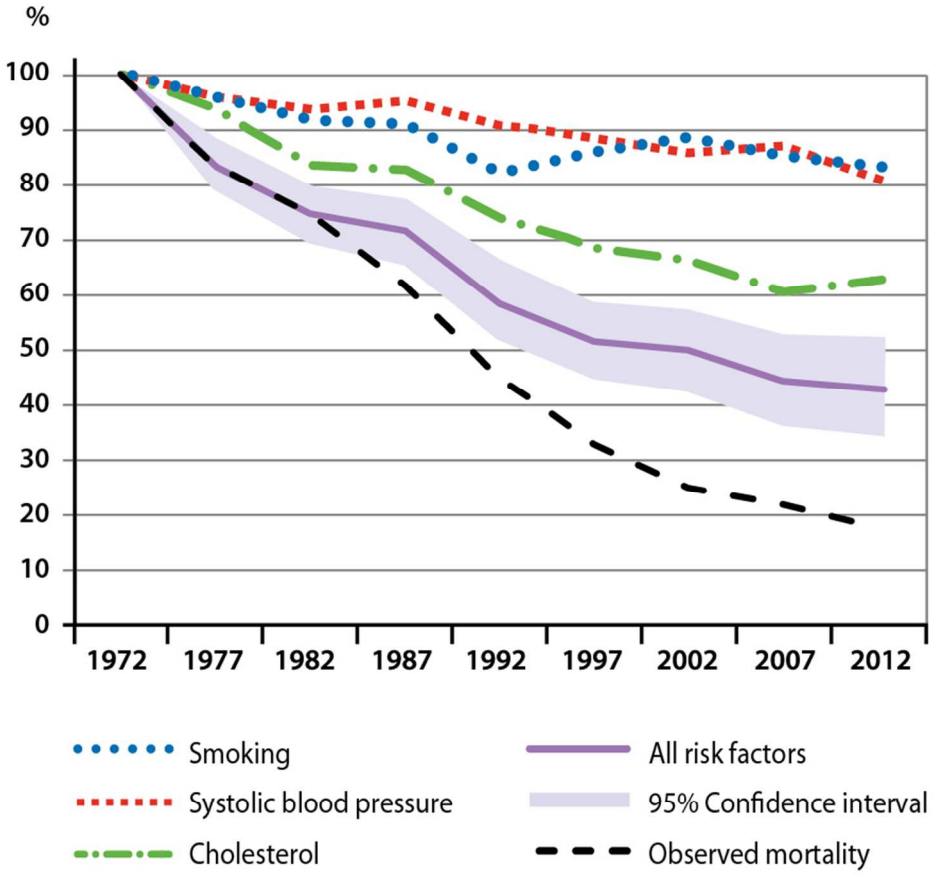

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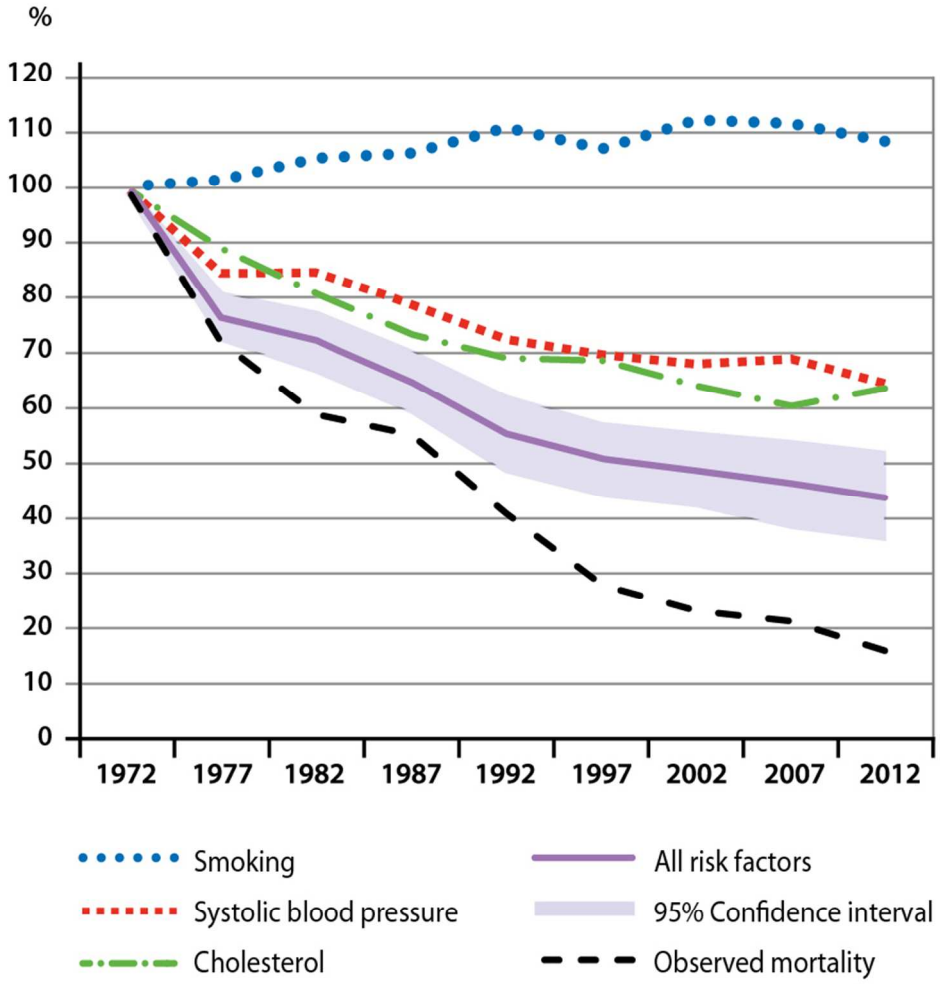

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